ENVIRONMENTAL LEAD EXPOSURE IN ST. JOHN'S NEWFOUNDLAND

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ENVIRONMENTAL LEAD EXPOSURE IN ST. JOHN'S, NEWFOUNDLAND

by

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Abstract

Residential soil and house dust samples were collected in St. John's to quantify the extent of lead contamination. Fifty-one percent of soil samples collected exceeded the CCME guideline of 140 ppm for lead (n=1231). Only 12% of house dust samples were above the US EPA standards (n=95). Historical use of leaded paint and combustion of coal and leaded gasoline have contributed to lead concentrations, especially downtown.

Based on soil lead concentrations there may be an increased lifetime health risk for all residents living on properties with soil lead concentrations greater than 900 ppm. Infants and toddlers are at particular risk, and when backyard produce is included, increased risk may occur at soil lead concentrations as low as 38 ppm for these particular receptors. There is a lot of uncertainty in the estimation of lead in backyard produce, but it maybe advisable to avoid consuming it until further research is conducted.

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List of Abbreviations and Symbols

CCME	Canadian Council of Ministers of the Environment
IEUBK	Integrated Exposure Uptake and Biokinetic Model for Lead in Children
HHRA	Human Health Risk Assessment
ppm	parts per million
PQRA	Preliminary Quantitative Risk Assessment
US EPA	United States Environmental Protection Agency

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Chapter 1 Introduction and Overview

Prior Research

The first indications that environmental lead levels may be elevated in the St. John's area arose from research conducted by Christopher *et al.* (1993) comparing the sediment chemistry of four urban ponds in St. John's and a background pond near Bay Bulls, a community just south of the city with similar underlying geology. Sediment cores were taken to collect information on how trace elements, pollen, diatoms, soot, charcoal, oil droplets, and stable lead isotopic ratios have changed through time in an effort to see how the urbanization of St. John's has affected environmental change.

The study found that lead concentrations at the top of the core samples were elevated for all sites including the background pond, although the increase for this pond was much less than its urban counterparts: only 17 ppm compared to 34 ppm for the other sites. The authors hypothesized that the slight increase observed in the background pond was long range transport and deposition of lead that may be impacting the entire region. This was supported by similar evidence of elevated lead concentrations in lake sediments in south-central Newfoundland (Davenport *et al.*, 1992). In contrast, recent urban lake sediments had lead concentrations 250 to 600 ppm higher than corresponding older sediments which were presumably unaffected by urbanization. The fact that all urban ponds exhibited this pattern prompted the authors to suggest that the widespread combustion of leaded gasoline during the middle of the 20th century was the most likely factor contributing to

elevated lead concentrations. Coal combustion was hypothesized as an additional source of lead for ponds located closer to the old downtown district based on the corresponding increase in sediments of coal-associated heavy metals. Additionally, the presence of soot particles correlated well with lead concentrations in one of the ponds located close to downtown (Quidi Vidi Lake), which provided added support for the link between coal combustion and increased lead levels. Christopher (1999) hypothesized that the historical combustion of both leaded gasoline and coal had most likely created a persistent source of lead in catchment soils, which may provide an important source of lead intake for the general population through the ingestion of soil and dust.

To test the hypothesis that lead concentrations were elevated in urban soils in St. John's a pilot study was conducted in 2003 (Bell, 2003). Soil was tested for a variety of heavy metals in locations where children might be exposed, including open spaces, parks, and school and residential properties. Sample locations were strategically chosen in both the older downtown as well as more recently developed areas of the city. The results from 341 samples ranged between 17 and 7048 ppm, with a median value of 266 ppm (Bell, 2003). Sixty-nine percent of the samples had lead contents which exceeded the Canadian Council of Ministers of the Environment (CCME) residential soil lead guideline of 140 ppm. Nine percent of the samples were ten times the CCME guideline, the majority coming from residential properties in downtown St. John's. Specifically, the soil lead median concentration for only those samples collected on residential properties was 744 ppm (Bell, 2003).

Objectives

There are two major research questions addressed in this project and each are discussed separately in Chapters 2 and 3. The first question deals with environmental lead concentrations and explores spatial and temporal relationships between concentrations and residential characteristics. The second paper uses the environmental lead data to undertake a human health risk assessment for lead exposure.

Because the pilot soil lead study indicated that soil lead concentrations were higher than federal guidelines for a number of property types and ages, it was first necessary to confirm these patterns across the entire city. The sampling program was also expanded to include indoor dust lead levels because of the potential impact that elevated soil lead may have on indoor dust lead. This relationship between outdoor soil lead and indoor dust lead was a particular focus of the investigation. Finally, the property and dwelling age of the sample sites were evaluated in terms of their capacity to explain measured soil and dust lead concentrations. It was hypothesized that older homes would have elevated environmental lead levels due to the historic use of lead based paint and coal burning heating systems, and a longer exposure to leaded gasoline emissions. If property age was determined to be a reliable predictor of elevated soil and/or dust levels then it may prove useful in focusing city-wide community interventions.

Once environmental lead levels are established for St. John's, an important next step is to conduct a human health risk assessment (HHRA). In a HHRA, exposure to the

contaminant of concern is measured for all possible pathways (food, air, water, soil, dust, consumer products etc.) and for all possible intake routes (ingestion, inhalation, and dermal contact). Soil lead levels for St. John's were entered into both worst-case and average-case scenarios along with other exposure and intake parameters which were adopted from the literature. The primary objectives were to establish: (i) whether daily lead intake averaged over either an entire lifetime or a specific age category were higher than recommended by Health Canada; (ii) what age of receptor was most at risk; and (iii) at what soil lead level did risks become elevated. In addition, the US EPA Integrated Exposure, Uptake and Biokinetic Model for Lead in Children (IEUBK) was used to predict blood lead concentrations, and therefore health risk, from site-specific soil data for children in St. John's. The relationship between housing age and soil lead concentrations was used in combination with the HHRA and IEUBK output to extrapolate the predicted health risks to the community level in order to identify the neighbourhoods most at risk. It was hypothesized that elevated health risk may be associated with properties which have very high soil lead levels. The dominant exposure pathway was expected be from soil, mainly from ingestion, and to a lesser extent inhalation and dermal contact. The receptor most at risk was hypothesized to be toddlers because of their characteristic hand-to-mouth behaviour and therefore increased soil ingestion.

Study Significance

Through investigation of the spatial patterns of lead contaminated soil and indoor dust and estimation of potential negative health effects, important information is generated for the residents of St. John's. The spatial analysis of environmental lead concentrations will help identify which areas of the city are most at risk and where on individual properties elevated soil lead levels are most likely to be found, thus focusing any remedial efforts to mitigate exposure. The risk assessment informs residents how exposure to the measured environmental lead levels may prove deleterious to their health. In order for negative health effects to occur from environmental contamination people in the community must be exposed to the contaminant during their daily activities. Conducting a risk assessment puts the environmental lead levels found in the first portion of the research study into perspective. Risk assessment methods can help identify the specific age groups of the population that are most at risk which can help target any future educational efforts or the need for blood lead monitoring programs. It can also identify the most influential exposure pathways contributing to lead intake and thus can help focus remedial action. If the risk assessment finds unacceptable health risks then further medical research and a blood lead screening program may be warranted.

This research may also benefit the scientific community. Most of the research on exposure risks to heavy metals in Canada has been conducted on industrially contaminated sites. This project provides a new set of Canadian soil and dust data for residential sites that are not influenced by a direct source of industrial pollution. It also

presents a modification of traditional risk assessment methods for residential properties. Results investigate the predictive relationship between outdoor soil lead concentrations and indoor dust lead loadings and examine the capacity of housing stock age to effectively predict environmental lead levels and risk. Study findings may also be applicable to other communities with a similar development history, a legacy of painted clapboard houses, and a dependency on coal for both industrial and residential energy supply.

Outline of Thesis

Chapter 1 outlines the background information needed to fully understand the research problem. The health effects of lead are addressed along with potential sources of lead in the environment. The components of, and approaches to, HHRA are then described. Specific comments are made on the treatment of variability and uncertainty in the dataset for risk assessment for lead in St. John's. Finally, the contributions of project participants and partners are acknowledged at the end of this chapter.

The lead levels in outdoor soil and indoor dust samples collected in St. John's are presented and analyzed in Chapter 2. Soil data are divided into three distinct sample categories: those collected near roads (road), adjacent to the foundation of buildings (dripline), and away from both roads and buildings (ambient). These three categories are analyzed and mapped at both city-wide and neighbourhood levels and are also related to

the age of the property from which they were taken. From this analysis, the areas most at risk of exceeding CCME soil guidelines are determined, including where on properties the highest concentrations are found (road, dripline, or ambient), and at what property age are soil lead levels elevated.

Chapter 3 presents two different types of risk assessment for residential homes in St. John's. The first is a Preliminary Quantitative Risk Assessment (PQRA) based on federal risk assessment guidelines. This risk assessment looks at an average exposure scenario to determine a probable range of risk for individuals living in houses with elevated soil lead concentrations. The IEUBK model generated by the United States Environmental Protection Agency (US EPA) is also used to predict blood lead levels for children using modified average exposure parameters. The output from the two models are used to identify the age of dwelling and associated soil lead level which correspond to the highest health risk for individuals in certain age classes. Maps are presented that identify neighbourhoods of St. John's with older housing stock that may have associated health risks from soil and dust lead exposure.

Chapter 4 summarizes the combined outcomes from environmental lead sampling and risk analyses in St. John's and presents some recommendations as well as suggestions for future research.

Lead and Human Health

There are three pathways that chemicals can enter the body: inhalation, ingestion, or dermal absorption. Absorption through the skin, however, can be considered minimal for inorganic lead compounds (OMEE, 1994). The amount of lead deposited in the lungs through inhalation ranges from 30 to 50% of the total concentration in the air for adults, and 25 to 45% for children (US EPA, 1998). The amount of lead absorbed is dependent on particle size and the location of deposition within the lungs. Large particles are generally inhaled in occupational settings and they tend to accumulate in the upper respiratory tract, whereas smaller particles typical of urban air deposit in the lower respiratory tract, where absorption into the blood stream is assumed to be 100% (US EPA, 1998). The concentration of fine particulate lead from urban sources therefore can have a large effect on blood lead concentration.

Absorption across the intestinal lining after the ingestion of lead differs greatly between adults and children, and is also dependent on nutritional status. In adults, 10 to 15% of ingested lead is absorbed and up to 50% is absorbed in children and pregnant women (USEPA, 1998). Iron, calcium, and phosphorus deficiencies also increase lead absorption (OMEE, 1994). Once lead is absorbed into the bloodstream it is distributed into various tissues. Some enters the soft tissues of the kidney, bone marrow, liver and brain, whereas some is stored more permanently in the mineralizing tissue of bone and teeth (US EPA, 1998). Lead circulates between tissues and is slowly excreted from the system. The half life of lead - meaning the time taken for half the lead volume to leave the system - from a

single large exposure is roughly 25 days in blood, 40 days in soft tissue, and 25 years in bone (US EPA, 1998). This illustrates how lead can have effects long after the initial exposure.

Children and neonates (via the mother) are the most sensitive to the neurological impacts of lead. There are a number of physiological and behavioral reasons why this is the case. First children have a higher gastrointestinal absorption rate (Ziegler *et al.*, 1978 in ATSDR, 2005) and because their nervous system is not fully developed it is easier for lead to cross the blood-brain barrier (Adinolfi, 1985; Johanson, 1980; both in ATSDR, 2005). Children also display a high rate of hand-to-mouth behaviour which increases the amount of potentially contaminated soil and dust consumed (Moya *et al.*, 2004).

The health effects of lead exposure on children depend on the amount of lead taken into the body (Table 1.1). Typically the amount of lead in the blood stream is used as an indicator of health effects. In general lead affects the developing nervous system causing mental and behavioural problems, the hematological system by inhibiting heme synthesis and decreasing erythrocyte (red blood cell) lifespan, thus eventually causing anemia, the cardiovascular system by increasing blood pressure, and the kidneys by decreasing the glomerular filtration rate (ATSDR, 2005).

Table 1.1. Children's blood lead concentrations and the corresponding adverse health effects (modified from ATSDR, 2005).

Blood Lead (µg/dL)	Adverse Health Effects
~5	Depressed ALAD, an enzyme in the heme biosynthesis pathway
<10	Neuro-developmental effects
~10	Delayed sexual maturation
~15	Depressed vitamin D
>15	Elevated erythrocyte protoporphyrin, a result of enzyme activity change in the heme biosynthesis pathway
~30	Depressed nerve conduction velocity
~40	Depressed hemoglobin
~60	Colic
70-100	Encephalopathy
150	Death

The neuro-developmental effects of lead including decreased IQ scores and other intelligence and developmental deficits have been heavily studied because of their occurrence at low blood lead concentrations. During brain development, lead interferes with the trimming and pruning of synapses, migration of neurons, and neuron/glia interactions so that appropriate connections are not made which may lead to permanently altered function (ATSDR, 2005). Meta-analysis of comparable studies have revealed a highly significant relationship between IQ score and blood lead concentration ($P<0.0001$, Needleman and Gastonis 1990), and Schwartz (1994) found in his meta-analysis that doubling blood lead concentrations from 10 to 20 µg/dL caused a loss of 2.6 IQ points. In general a blood lead concentration increase of 10 µg/dL has been shown to cause a decrease in IQ of between one and five points (ATSDR, 2005). IQ deficits may be related to other behavioural changes that go along with lead exposure including increased distractibility, impulsivity, short attention span, and inability to follow simple and

complex sequences of directions (ATSDR, 2005).

The data surrounding these findings are not unanimous. Three out of four major longitudinal studies flowing children through time found a significant correlation between high prenatal and/ or postnatal blood lead concentration and poor performance on mental development test, both during the toddler years and into older childhood; Boston (Bellinger *et al.*, 1985), Cincinnati (Dietrich *et al.*, 1987a) and Port Pirie, Australia (Vimpani *et al.*, 1985 (all discussed in US EPA, 1998). The Cleveland study was less clear in its conclusions (Ernhart *et al.*, 1985). Numerous cross-sectional studies comparing exposed and unexposed children at a single point in time have also been conducted in countries around the world (US EPA, 1998). Research in Scotland (Fulton *et al.*, 1987), China (Wang *et al.*, 1989), and Greece (Hatzakis *et al.*, 1987) showed significantly decreased development test scores at blood lead concentrations ranging from 3 to 64 µg/dL. Other studies, however, found no association between blood lead and performance on development tests within a blood lead range of 4 to 32 µg/dL (Lansdown *et al.*, 1986; Harvey *et al.*,1988). One of the difficulties in interpreting the results of these studies is that it is hard to define and control confounding factors for mental development as a variety of other factors may influence the scores. Birth weight, gestational age, socio-economic status, parental intelligence scores, and quality of home environment can all play a role in mental development (OMEE, 1994; US EPA, 1998).

The United States Centers for Disease Control and Prevention (US CDC) has set the screening guideline for blood lead at 10 µg/dL, but this level is just a tool for risk management and most studies conclude that there is no threshold in blood level concentration at which health effects first occur (US CDC, 1991; Lanphear *et al.*, 2000; Bellinger, 2004). In 1994 the Federal Provincial Committee on Environmental and Occupational Health Lead Working Group recommended that individual intervention occur when a child's blood lead concentration exceeded 10 µg/dL, and that community level intervention be undertaken when blood lead concentrations from a sample of children exceed the mean from the general population plus three standard deviations, or when the percentage of children with values above 10 µg/dL is double that seen in the general population (Federal Provincial Committee on Environmental and Occupational Health Lead Working Group, 1994). Recent studies now show that negative health effects may occur at blood lead concentrations below 10 µg/dL (Federal Provincial Committee on Environmental and Occupational Health Lead Working Group, 1994; Canfield *et al* 2003; Bellinger and Needleman 2003).

Blood lead concentrations in children have been studied in Canada, particularly for those areas impacted by an industrial source of lead pollution (Table 1.2). In general blood lead concentrations have been steadily declining since the late 1970s as sources of lead intake were phased out. Recent studies indicate that Canadian children appear to have relatively low blood lead concentrations, between 1.9 and 6.7 µg/dL (Table 1.2).

Table 1.2. Results for blood lead studies conducted in Canada.

Year	Location	Point Source of Pollution	Age Range	Blood Lead Level (µg/dL)	Percent Over 10 µg/dL	Reference
1978	Canada Health Survey	N			25%	Health and Welfare and Statistics Canada, 1981
1979	Rouyn-Noranda, QC	Y		Mean 21.4		Goulet *et al*., 1996
1984	Ontario Blood Lead Study	N		Mean 12.0		Duncan *et al*., 1985
1984	South Riverdale, TO, ON	Y	<6 yrs	GM* 12		Langlois *et al*., 1996
1987	Alberta	N	0-16 yrs	Mean 5.8-6	5 samples > 10	Audette, 1990
1989	Quebec City, QC	N	1-6 yrs	Mean 5.6	10.80%	Levallois *et al*., 1990
1989	Vancouver, BC	N	2-3 yrs	GM 5.9	5%	Vancouver, 1990
1989	Rouyn-Noranda, QC	Y		Mean 11.1	2 children > 25	Letourneau *et al*., 1989
1989	St-Jean-sur-Richelieu, QC	Y	6 m-10 yrs	GM 9.2		Goulet *et al*., 1996
1990	Murdochville,QC	Y	6 m-5 yrs	Mean 5.9		Chagnon and Bernier, 1990
1990	Murdochville,QC	Y	5-12 yrs	Mean 6.7		Chagnon and Bernier, 1990
1991	Trail, BC	Y	< 6 yrs	13.5	83%	Hilts *et al*, 2001
1991	St-Jean-sur-Richelieu, QC	Y	6 m-10 yrs	GM 5.0		Goulet *et al*., 1996
1992	South Riverdale, TO, ON	Y	<6 yrs	GM 3		Langlois *et al*, 1996
2000	Trail, BC	Y	< 6 yrs	6.7	27%	Hilts *et al*., 2001
2001	Sydney, NS	Y	1-5 yrs	GM 1.9	0%	NSDH and CBDHA, 2001
2001	Port Colborne, ON	Y	<7 yrs	GM 2.3	0%	Decou *et al*., 2001

- Geometric Mean

In residential areas near the coke oven site in Sydney, Nova Scotia, soil lead levels ranged from 52 to 1700 ppm with a median of 340 ppm; however, none of the children screened had blood lead levels above 10 µg/L (Lambert and Lane, 2004). The geometric mean for blood lead was 1.86 µg/dL for children ages 1 to 5 years, and the maximum observation was just under 9 µg/dL (Nova Scotia Department of Health and the Cape

Breton District Health Authority, 2001). For Port Colborne, Ontario, the geometric mean for blood lead in children under seven years of age was 2.3 µg/dL and no children exceeded the 10 µg/dL guideline (Decou *et al.*, 2001). The average soil lead concentration was 217 ppm in the Port Colborne study, but no correlation was found between blood lead and soil lead levels (Decou *et al.*, 2001).

In studies that included lead abatement measures, blood lead concentrations have also declined dramatically in recent years. The ten-year long soil and dust abatement study in South Riverdale, Toronto, showed a decrease from 12 to 3 µg/dL in the geometric mean blood lead of children under six years of age between the years 1984 and 1992 (Langlois *et al.* 1996). In Trail, B.C., the geometric mean blood lead concentrations for the same age range of children declined from 13.5 to 6.7 µg/dL between 1991 and 2000 (Hilts *et al.*, 2001).

Based on these results it would seem that currently in Canada the majority of children are at a relatively low risk of experiencing health effects from lead exposure. The lead concentrations presented are only averages and there remains a small portion of the population at risk as observed in the Trail study where 27% of the children continued to experience elevated blood lead concentrations in 2000 (Hilts *et al.*, 2001).

Sources of Lead in the Environment

Historically, inhaled emissions from leaded gasoline combustion and industrial activities were the primary sources of lead exposure for Canadians (Health Canada, 2004b). Since the phasing out of leaded gasoline and increasing restrictions on industrial emissions, airborne lead has become less of a concern while soil lead and household dust have drawn increased attention as exposure pathways in urban environments (US EPA, 1989; Mielke *et al.*, 2003). Historical inputs have created a lead sink in soil because lead does not biodegrade and is highly immobile in soil (Davies, 1995). Other pathways of exposure to lead may occur through produce grown in back yard gardens, drinking and bathing water, store-bought food, inhalation of urban air, the direct ingestion of leaded paint, contact with consumer products containing lead, and the import of occupational or hobby lead contamination into the home.

Outdoor Soil

Naturally-occurring background concentrations of lead have an average concentration of 16 mg/kg in crustal rock, whereas surface soils range from 30 to 100 mg/kg due to pervasive low-level contamination at all but the most remote sites (Davies, 1995).
As previously mentioned soil can be contaminated with lead from several sources, including the deposition of airborne combustion materials from point-source emitters and automobiles as well as from the addition of weathered lead-based paint. Lead in soil is a concern because it remains on site long after the initial deposition. Lead is immobilized

in soil under alkaline and high cation-exchange conditions (Zimdahl and Skogerboe, 1977). This is done primarily through reactions with insoluble organic matter, but lead may also be precipitated by carbonate or sorbed onto hydrous metal oxides (Zimdahl and Skogerboe, 1977).

There is a large body of research on urban geochemistry for lead because of the associated health concerns (Table 1.3). Median soil lead concentrations for all samples taken in a pilot study of St. John's were higher than most other community surveys in Canada with the exception of Sydney, Nova Scotia, and Trail, British Columbia, which have direct sources of lead pollution (Table 1.3). Compared to international studies the median soil lead value for St. John's is comparable to New Orleans, U.S.A., and Birmingham, England. As described in Table 1.3, not all studies used the same sampling technique. In general all values are for surface soil which is comparable to the St. John's data, but not all studies sampled the same variety of locations. Those that only sampled garden soil or open areas were generally lower than those that focused on soil around buildings.

Table 1.3 Soil lead concentrations in Canadian, American, and international cities.

City	Number of Soil Samples	Metric	Lead (ppm)	Sampling Method	Reference
Canada					
St. John's, NL	208	Median	266	Surface soil was collected from open spaces, parks, schools, residences	Bell, 2003
	27	Median	744	Surface soil was collected from around residences, mainly in historic downtown.	
Belledune, NB	17-21, depending on the sub-area	Range of median values for 5 sub-areas	43 136	One composite soil sample per garden (10 sub-samples) was taken at a depth of 5-20 cm.	Government of New Brunswick, 2006
Sydney, NS	55	Median	340	Samples were taken from the top 5 cm of soil from the middle of the yard on residences near the Coke Oven site	Lambert and Lane, 2004
Victoria, BC	245	Median	90	A census tract stratified sampling strategy was used to sample surface soil from boulevards, parks, school yards	Bowman and Bobrowsky, 2003
Trail, BC		Geometric Mean	756	The top 2-3 cm of residential soil was sampled after the construction of a new lead smelter	Hilts, 2003
Port Colborne, ON	~2000	Median	167	One composite containing a minimum of 9 cores was collected from residential yards. Driveways, walkways, buildings, fences, and debris were avoided. This median refers to the top 5-10 cm.	Ontario Ministry of the Environment, 2002
Ottawa, ON	50	Geometric Mean	42	One garden soil sample was taken within 15 m of the house.	Rasmussen *et al.*, 2001
Iqualuit, NU	101	Median	13	Samples were collected on commercial and residential sites at the corners of a 200 by 200 m grid as well as on targeted areas such as playgrounds, roads, and culverts.	Peramaki and Decker, 2000

City	Number of Soil Samples	Metric	Lead (ppm)	Sampling Method	Reference
U.S.A					
Syracuse	194	Geometric Mean	80	The top 10 cm were sampled on street sides (44%), parks (28%), residences (28%). Dripline areas around buildings were avoided.	Johnson and Bretsch, 2002
Chicago	62	Median	1773	One composite sample formed from 3-10 sub-samples was collected residential on properties with at least 0.84 m^2 bare soil. Samples were taken from around the foundation of the house and in play areas.	Shinn *et al.*, 2000
Washington	30 for each ward	Range of medians for 8 wards	54 - 471	Residential topsoil was collected at a depth of 15 cm, 1 m from dwellings.	Elhelu *et al.*, 1995
New Orleans	74	Median	212	Inner-city open spaces were sampled, two per census tract. The top 2.5 cm were collected away from busy streets and intersections	Mielke, 1994
	80	Median	40	Mid-city open spaces	
	114	Median	28	Suburban open spaces	
World					
Jacobstad, Finland	32	Median	59	Composite samples composed of 3-5 sub-samples were collected from the top 10-15 cm of soil from a variety of sites: schoolyards, parks, roadsides, fields, abandoned building yards, industrial sites etc.	Peltola and Astrom, 2003
Belize City	25	Mean	638	The top 3-5 cm of residential topsoil was sampled, often near dilapidated structures.	Reeder and Shapiro, 2003
Olso, Sweden	300	Median	34	Urban surface soil was sampled using a grid technique of one sample per km^2. The top 3 cm of soil was collected	Tijhuis *et al.*, 2002

City	Number of Soil Samples	Metric	Lead (ppm)	Sampling Method	Reference
Birmingham, UK	84	Median	244	One composite sample was made from 25 sub-samples taken from the top 5 cm of exposed soil in the front and back garden of residential properties.	Wang *et al.*, 1997
Aberdeen, Scotland	30	Mean	94	Ten parkland soil cores were taken to a depth of 10 cm within a 2 by 5 m rectangle at each site	Paterson *et al.*, 1996
	50	Mean	173	Same as above, but sample was taken at least 1 m from road edge	
United Kingdom	4126	Geometric Mean	266	Top 5 cm of residential soil was collected as a composite of 25 sub-samples. Hotspots of heavy metal were excluded.	Thornton *et al.*, 1990
	578	Geometric Mean	654	Same as above. Samples collected from London Boroughs.	

Soil lead may contribute to children's blood lead levels through direct inhalation or ingestion through hand-to-mouth behaviour. Soil lead concentrations were directly related to hand lead concentrations in inner city New Orleans (Viverette *et al.*, 1996). An average of approximately 30 mg of lead was measured on children's hands after outdoor play, six times more lead than measured while playing indoors, and five times more than the tolerable daily intake for children less than six years old in the United States (Viverette *et al.*, 1996).

In general, studies have shown that soil lead levels of 1000 ppm may contribute 2 to 7 μg/dL to overall blood lead findings (Lanphear *et al.*, 2000). The relative impact of soil

lead exposure on blood lead levels, however, is controversial and elevated soil lead levels do not necessarily correspond with elevated blood lead levels. It has been suggested that the relationship is only present when large scale geographic blood lead data sets are integrated with soil lead data (Johnson and Bretsch, 2002). In Syracuse, NY, for example, blood lead levels only corresponded to soil lead levels when the geographic units were increased from 600 m^2 to 3 km^2, roughly the same size as census tracts, the spatial unit of analysis in another study conducted in New Orleans, which found a nearly identical relationship (Johnson and Bretsch, 2002; Mielke *et al.*, 1999).

Lowering of elevated soil lead levels does not necessarily correspond with reductions in blood lead levels. For the Boston Lead-in-Soil Demonstration Project, a large decline in soil lead (from 2075 to 105 ppm) was required to achieve only a 12% decline in children's mean blood lead (Weitzman *et al.*, 1993). Similarly, an abatement study in Baltimore also found reductions in soil lead concentration ineffective, as blood lead levels dropped for both control and treatment groups (Farrell *et al.*, 1998). It was hypothesized that soil abatement was not effective because paint was the primary lead source for the neighbourhoods tested and the reduction in soil lead on study properties was not sustained because of re-contamination from neighbouring properties that were not part of the study. Additionally, the authors suggested that indoor dust may play a more important role in blood lead concentrations than originally considered during the study design.

Geometric mean blood lead levels for children in three historically contaminated Canadian communities did not exceed 10 µg/dL. In the Sydney, Nova Scotia study of neighbourhoods surrounding the tar ponds and historical coke ovens, and the Port Colborne, Ontario study around the nickel smelter, median soil lead concentrations were 340 ppm and 167 ppm, respectively, and no children tested were over 10 µg/dL (NSDH and CBDHA, 2001; Decou *et al.*, 2001). Around the lead and zinc smelter in Trail, British Columbia, the median soil lead concentration was much higher at 756 ppm and 27% of children tested exceeded 10 µg/dL (Hilts *et al.*, 2001). On the bases of these studies and the median soil concentrations observed in the St. John's pilot study, it appears unlikely that the majority of residents are at risk; however, for those residential properties sampled in downtown St. John's the median soil lead level was comparable to Trail and thus a portion of the children living in this area may be at risk.

Indoor House Dust

Sources of lead in interior dust include the tracking in of contaminated soil and the weathering of leaded interior paint. Dust results from Sydney, Nova Scotia, indicated that lead loading was highest in doorways, in some places an order of magnitude higher than dust located farther within the house (Lambert and Lane 2004). These observations led the investigators to believe that lead contamination was mainly due to exterior sources of lead, possibly contaminated soil (Lambert and Lane 2004). Contaminated soil was estimated to supply 20 to 30% of lead measured in indoor dust in a pilot study specifically designed to address the relative contribution of contaminated soil to indoor

dust (Rutz *et al.*, 1997). This study was conducted at the Fernald Environmental Management Project facility of the U.S Department of Energy outside of Cincinnati, Ohio. Conversely, Lanphear and Roghmann (1997) found that concentrations of lead in interior paint contributed more to indoor dust lead levels than the concentration in soil in Rochester, New York. Rasmussen *et al.* (2001) also suggested that the source of the lead in house dust in Ottawa residences is inside the house, with leaded interior paint being the likely source. This was based on their findings that house dust lead concentrations were higher than soil levels concentrations (232 ppm and 42 ppm, respectively) and were also associated with a distinct multi-element signature.

Lead in house dust has not been as extensively studied as in soil; however, the research which has been conducted indicates that the ingestion of house dust may be a major contributor to blood lead levels in children both directly and through loading on hands (Thornton *et al.*, 1994; Lanphear *et al.*, 1996; Lanphear and Roghmann., 1997; Lanphear *et al.*, 1998; Yiin *et al.*, 2000). In a recent Ottawa study, house dust intake accounted for 30% of the total lead exposure for children, second only to food intake (56%), when geometric mean concentrations were considered (Rasmussen *et al.*, 2001). Dust was the dominant source of lead (69% of total daily lead intake) when the 95th percentile concentration was used. This range corresponds closely with other studies which have found that indoor dust lead contributes around 50% to total lead intake in young children (Thornton *et al.*, 1994).

Those studies which have identified indoor dust as a major source of lead exposure in children have also demonstrated a strong relationship between dust lead loading (amount of lead per area wiped for dust) and blood lead concentrations (Lanphear and Roghmann, 1997; Lanphear *et al.*, 1998; Yiin *et al.*, 2000). Lanphear et al. (1998) pooled twelve epidemiological studies to estimate the contributions of house dust and soil to children's blood lead. Floor dust loading was the most significant environmental predictor of blood lead, with soil contributing to a lesser extent in the multivariate regression model. Elevated blood lead levels increased dramatically at floor dust loadings of 5 to 10 $\mu g/ft^2$. This research was used to lobby the US EPA to lower its house dust lead limits. Currently, the US EPA dust lead loading standards are 40 $\mu g/ft^2$ for floors, 250 $\mu g/ft^2$ for window sills, and 400 $\mu g/ft^2$ for window troughs (US EPA, 2001). Neither Health Canada nor Environment Canada has any protective guidelines for lead in house dust. Health Canada states that there are too many sources of lead exposure to accurately set standards for each potential exposure pathway; instead they encourage homeowners to reduce lead levels in their homes as much as practically possible (Health Canada, 2004b).

Indoor dust lead may present more of a health concern than previously thought for several reasons. First, it may accumulate higher concentrations of lead because of its relatively high organic component, in particular mold and fungi, which are highly effective at accumulating high metal concentrations (Rasmussen *et al.*, 2001). Second, the metal concentration in dust may be more bioavailable than soil. For example, in the Ottawa study by Rasmussen (2004), suburban house dust had a relative bioavailability of

60% compared to roughly 14% for rural topsoil8. Third, preschoolers spend the majority of their time indoors, making the impact of lead in house dust potentially greater than outdoor soil (Yiin *et al.*, 2000).

Backyard Produce

The ingestion of lead from fruits and vegetables grown in the home environment is a potential lead exposure route that has received little attention, but may prove to be a recurring source of lead for both children and adults. Fruits and vegetables grown in contaminated soil may become contaminated as a result of plant uptake of lead from soils or direct deposition of leaded dust onto plant surfaces. There are numerous factors affecting the level of lead present in garden vegetables, including the soil lead concentration, the type of plant and its corresponding lead uptake rate, lead speciation, and soil interactions (Peryea 2001; Samsoe-Petersen *et al.*, 2002). Characteristics of the soil itself, including pH and organic matter, may directly affect the lead uptake rate (Peryea, 2001).

A pilot study in Chicago examined the relationship between soil lead and lead in garden vegetables (Finster *et al.*, 2004), and found that all plants grown in contaminated soil accumulate lead to some degree The geometric mean soil lead levels for the gardens studied was 639 ppm., with a range of 27 to 4580 ppm. These values are comparable to the preliminary soil lead concentrations observed in the pilot study conducted in St. John's making the Chicago data of particular interest. Finster *et al.* (2004) found that lead

is primarily localized in plant roots, with declining amounts present in the shoot and fruit. From the edible portions sampled, herbs, leafy vegetables and root vegetables had the highest concentrations of lead. Elevated plant lead concentrations were not solely derived from root uptake; surface adhesion of contaminated dust was also a factor, indicating that washing vegetables in a mild detergent would help reduce lead exposure.

The consumption of produce grown in lead contaminated soil may not contribute a substantial amount of lead to the total body burden; however, during the harvest season when these vegetables may comprise a large portion of the diet the contribution may be significant, especially for children. For example, Finster *et al.* (2004) estimated that the consumption of 1 tablespoon of dried cilantro with a lead concentration of 49 µg/g (dry weight) adds 85.75 µg of lead to the diet, an amount above the US government's recommended total tolerable intake level for all age groups.

Store-Bought Food

According to the Canadian Total Diet Program conducted in 1985 (Dabeka *et al.*, 1987) the daily intake of lead based on body weight was highest for infants (1.7µg/kg/day) and lowest for adults over twenty (0.57 µg /kg/day). This has decreased to 0.48 µg/kg/day and 0.19 µg/kg/day, respectively, in the most recent assessment (Health Canada, 2005). It is therefore likely that lead intake in store-bought food has decreased since 1985 because the deposition of airborne lead on crops and the use of lead in cans has virtually been eliminated (US EPA, 1998).

Drinking and Cooking Water

Although water from municipal supplies must be below the maximum acceptable concentration (MAC) for lead in Canadian drinking water, (0.010 mg/L; Health Canada, 1992), the concentration of lead can change as it travels through the distribution system. This may result from contamination from lead pipes, connectors, or solder in the municipal water system, or through contact with lead or brass components of coolers, faucets, or other fixtures in the home (US EPA, 1998). Lead service connecters were common in well-built homes before 1920, and solder was comprised of 50% lead until 1990, when the National Plumbing Code of Canada significantly reduced the lead content (Canada Mortgage and Housing Corporation, 2007; Health Canada, 2004b). It is still possible for homes built before 1950 to contain leaded distribution lines and service connections if they have not undergone renovations (Health Canada, 2004b).

Water samples collected in the Canadian Duplicate Diet Study had an average lead concentration of 0.0088 mg/L and a median of 0.002 mg/L (Debeka *et al.*, 1987). Health Canada (1992) suggests that the most realistic estimate of lead intake through drinking water is 0.0048 mg/L based on a survey of 40 homes in Ontario (Graham, 1988). Water lead concentrations obtained from the Atlantic Region Federal-Provincial Toxic Chemical Survey of Municipal Drinking Water Sources (Inland Waters Directorate, 1990) for St. John's indicate that almost all samples were below the detection limit of 0.002 mg/L and well below the MAC.

Given the relatively low concentration of lead in treated municipal water, lead from drinking water likely contributes only a small amount to the total lead in the residential environment. On the other hand, the high absorption rate of lead in water means that drinking water must be considered an important exposure source when lead is present. This may be particularly relevant for residents who are on well water or who have lead pipes or connectors.

To a much more limited extent contaminants in the water supply may impact residents through dermal absorption during bathing. This is more common for organic compounds which are fat soluble and can easily pass through the skin, and less common for inorganic compounds such as lead whose rate of transport through the skin is very slow (0.0001 cm/hr; US EPA, 2004).

Urban Air

The inhalation of lead polluted air was a major source of lead intake for children, but now that leaded gasoline is no longer available in Canada and industrial emissions are controlled, airborne lead concentrations have dramatically declined. According to data from National Air Pollution Surveillance the annual geometric mean for lead in Canadian air has decreased from 0.74 $\mu g/m^3$ in 1973 to less then the detection limit of 0.1 $\mu g\ m^3$ in 1991 (Health Canada, 1992).

Since the 1970s, blood lead concentrations in Canada have also decreased. In South Riverdale, a community in Toronto affected by an industrial lead source, the mean blood lead concentration declined from 14 µg/dL in 1984 to 3.9 µg/dL in 1992 (Langlois, *et al.*, 1996). During this time interval soil and house dust abatement measures were undertaken. Because the final blood lead levels were almost on par with the study control group (4.2 µg/dL) who did not receive abatement, and background blood lead levels (3.5 µg/dL) for Ontario,, the researchers concluded that it was not the decreased soil and dust concentrations that were responsible for lower blood lead concentrations, but rather the general reduction in emissions from leaded gasoline and the local smelter. Similar conclusions were reached in a study of blood lead levels in Trail, B.C., following the adoption of new flash-smelting technology at the local smelter in the 1990s, which reduced emissions (Hilts, 2003).

Lead Based Paint

Ingestion of lead based paint is considered one of the most significant high dose sources of lead (US EPA, 1998). Lead was the main ingredient in oil-based interior and exterior house paints from the 1900s to the 1940s. Its use declined during the 1950s and 1960s as latex paints emerged and lower lead content paints started to be used (Health Canada, 2004a; Health Canada, 2004b; US EPA, 1998). The deterioration of leaded paint can contribute to the lead burden of a child by adding large quantities of lead to soil or dust (Lanphear and Roghmann, 1997).

Commercial Products, Occupational and Hobby Exposures

In addition to the major sources of lead described above, children may also be exposed through consumer products such as leaded pottery, jewellery, or folk remedies, and in more recent news through leaded paint on toys made in China. Furthermore, lead may be introduced into the home through the occupation or hobbies of parents.

Risk Assessment

Risk assessment systematically determines the probability and magnitude of harm to public health, welfare, or the environment due to the release of hazardous agents into the environment (Santos, 1987). It is based on information on the hazardous effects of an agent(s) collected from epidemiological, clinical, toxicological, and environmental studies (National Academy of Sciences, 1983). The information is then extrapolated to estimate the health outcomes for a population exposed to the agent at a determined intensity and duration (NAS, 1983). This comprises the first three steps of the risk assessment process: hazard identification, dose-response relationship, and exposure assessment. In the fourth step - risk characterization - the data are synthesized and summary judgments are made on the existence and magnitude of the overall public health problem (NAS, 1983). Additionally, the uncertainties involved in the risk assessment are addressed to determine the overall confidence in the results.

The outcome of the risk assessment is used by risk managers to decide the best action to take in order to minimize the risk. Risk management is a separate step that involves a consideration of political, social, economic, and engineering information together with the risk assessment data to develop various options to address the situation (NAS, 1983). Risk management places value judgments on the acceptability of risks and the rationality of control costs, whereas risk assessment focuses solely on scientific data (NAS, 1983).

Hazard Identification

Commonly, the first step in a risk assessment is a complete site evaluation to determine which chemicals are present, where they are located, and in what quantities and concentrations. It is also important to note the natural characteristics of the site (geology and hydrogeology, topography, wind patterns etc.) as well as what the land is used for and who may come into contact with the contaminants (Health Canada, 2004a; Santos, 1987). From this information a list of contaminants of concern may be developed. Generally, this list consists of compounds that exceed set guidelines or if no guidelines are available those compounds that exceed background concentrations (Health Canada, 2004a).

It is important in risk assessment to evaluate the chemical and physical properties of the contaminants of concern to help discern the fate of the agent once it is released into the environment. These descriptors would include boiling point, density, particle size, pH, and dissociation constant among others (Paustenbach, 2002). Factors that influence

transport through different environmental media would also be considered, for instance adsorption, water solubility, and vapour pressure, as well as those that influence environmental persistence such as biological and chemical degradation (Paustenbach, 2002).

These first few steps are included in the hazard identification portion of the risk assessment to determine whether the physical or chemical agents present in the environment could increase the incidence of adverse health effects in an exposed population (NAS, 1983). It is based on toxicological data from a combination of both laboratory studies on animals and epidemiologic studies on humans. The studies are evaluated to determine the type of toxic effect, the underlying biological mechanism, as well as the nature and strength of causation (NAS, 1983).

Dose-Response Relationship

Once a chemical or physical agent is deemed hazardous the dose-response relationship is determined. This is often presented as a dose-response curve in which the amount of toxin administered to a test subject (or observed in human populations in epidemiological studies) and the resultant health outcome is plotted against one another. Often the dose is first related to a change in a biological indicator, such a blood concentration, and then the biological indicator is related to a change in health effect. The intensity of exposure, age of subject, pattern of exposure, and other variables such as sex and lifestyle are also considered when determining the dose-response relationship (NAS, 1983).

The dose-response relationship for non-carcinogenic substances, such as lead, generally has a dose threshold below which there are deemed no harmful effects. For lead this is generally observed to be 10 µg/dL, although as discussed above there is considerable debate whether there exists a threshold at all (Lanphear *et al.*, 2000).

Both laboratory and epidemiological studies are used to determine the dose-response relationship. In general well conducted epidemiological studies are preferred because they are done on humans and the exposure level is closer to what is likely to be experienced by the population in question (Paustenbach, 2002). However, these studies have less precision than laboratory studies because subjects are often exposed to multiple risk agents and have lifestyle factors that may also influence disease (Paustenbach, 2002). Still laboratory studies have more uncertainty as the data must be extrapolated from animals to humans and also from high doses administered over a short time period to chronic low dose exposures in human populations (NAS, 1983). In the dose-response section of a risk assessment it is important to be clear on the data source and what extrapolation techniques were used (NAS, 1983).

Exposure Assessment

The exposure assessment combines the environmental side of exposure with the human side (Santos, 1987). It quantifies the intake of the risk agent into the body based on contact with different contaminated environmental media such as air, water, soil, and food (Paustenbach, 2002).

First, the potential exposure pathways are determined. They may include dust and vapour inhalation, dermal contact with soil, dust or water, or the ingestion of contaminated food, water, soil, or dust (Paustenbach, 2002). Second, for each pathway the chemical concentration in the associated media must be determined, either by direct sampling or by estimating concentrations from models (Paustenbach, 2002). Third, the risk assessor must establish the contact rate between the media and the human receptor. For example, for ingestion the contact rate might be measured in grams per day, for inhalation cubic metres per hour, and for dermal contact grams per contact event. Intake rates are dependent on receptor characteristics, especially age. The concentration and contact rate give the magnitude of exposure, but it is also necessary to consider the duration of exposure; how many contact hours or events per day, week, and year are experienced by the receptor.

These factors help determine the actual amount of contaminant that reaches the skin or lining of the respiratory or gastrointestinal tracts, but then the compound must be absorbed across the barrier. This absorption is influenced by the bioavailability of the compound, whether or not it is bound to particulate matter, and its ability to diffuse across the membrane.

Once absorbed, the contaminant can be metabolized, stored, excreted, or transported within the body (Paustenbach, 2002). Thus there is a complex set of biological

mechanisms that determines the amount of contaminant that finally reaches the target organ or tissue and causes harm. Currently, these mechanisms can be reproduced using physiologically-based pharmacokinetic models, such as the US EPA's Integrated Exposure Uptake and Biokinetic (IEUBK) model for lead exposure in children. Otherwise, the application of a bioavailability factor provides a coarse substitution.

The end result for each pathway is an estimation of the daily quantity of contaminant to which a set of receptors is exposed. It is common for the doses from all the exposure pathways to be combined to give an overall estimate of total daily intake; the effects from ingestion, inhalation, or dermal contact are not always the same, however, and this must be considered before adding the doses.

Risk Characterization

Risk characterization combines the estimated exposure with the dose-response relationship to assess the likelihood and severity of the health risk (Paustenbach, 2002). In addition to providing quantitative and qualitative descriptions of risk it presents key information on which risk managers can base their decisions. For example, it should include a summary of the compound's hazard capacity, an overview of the toxicity data and dose-response model used, as well as a description of the key parameters used in the exposure assessment. It is also pertinent at this stage to discuss any variability or uncertainty in the data and modeling to establish the level of confidence that can be placed in the toxicity and exposure estimates (Paustenbach, 2002).

Variability

The US EPA (1997) identifies three types of variability: spatial, temporal, and inter-individual. Both environmental and receptor data vary across all three variability types in the St. John's study. For example, environmental concentrations vary spatially across properties and across the city, exposure changes seasonally in response to weather, ground surface conditions and receptor activities, and receptors may have varying responses to exposure due to individual characteristics such as nutritional status and age. It is difficult for a deterministic risk assessment to incorporate the full range of environmental and receptor characteristics and it becomes necessary to choose representative values for both sets of parameters. For instance, standard body weights are commonly used because the range of possible values is so large.

Variability within a dataset can affect the precision of the health outcome estimates and the degree to which they can be generalized (US EPA, 1997). Variability can be dealt with in several ways. First, it can be ignored if it is small, second, it can be disaggregated by using mathematical models, the average value (if reliable), or by creating subgroups, and third, the maximum and minimum values can be used to explore the range of values present in a population (US EPA, 1997). The following sections describe how variability in environmental and receptor data was treated in the risk assessment for lead in St. John's.

Environmental Data

Spatial differences in soil lead concentrations on individual properties were dealt with by averaging the values for all locations (roadside, dripline, and ambient). Because older properties were exposed to larger quantities of lead for a longer period of time, the temporal variability was accounted for by creating six property age categories. Lead concentrations in soil, dust, garden produce and water varied according to these age categories. The ambient concentration of atmospheric lead tends to be less spatially variable across the city and hence values were not partitioned by property age; instead, it was divided into indoor and outdoor components because of their respective sources. An inter-individual variability present in the environmental data relates to the plant specific nature of lead uptake in garden produce. This was accounted for by averaging plant specific values for those plant varieties grown in St. John's.

Receptor Data

Natural variability in receptors was acknowledged by creating five age categories, as intake rates and body weights vary with age. This division was also important because young children are more susceptible to lead exposure due to higher soil/dust ingestion rates and loading factors. Lead exposure is also highly seasonal with higher exposure to outdoor sources occurring in the warmer months when outside activities are concentrated. Seasonality was indirectly addressed in this risk assessment. High summer exposure values (body surface area exposed, time spent outdoors, amount of garden produce

ingested) were averaged with low winter values to produce a daily value that was representative of the year as a whole and not specific seasons. This may mask seasonal increases in exposure and associated increases in blood lead levels and health effects. Inter-individual differences in receptors, besides age-related differences, are hard to incorporate into the risk assessment and for the most part only age-specific averages were used. Differences in lead absorption were explored using a range of percentages, but that was done mainly because of the uncertainty surrounding lead absorption in different media and not because of individual variations in absorptive capacity.

Uncertainties and Limitations

Uncertainty in risk assessment refers to the lack of knowledge of factors that affect risk, which may result in inaccurate or biased estimates (US EPA, 1997). Uncertainty can be reduced by limiting or eliminating knowledge gaps. Like variability, uncertainty can be organized into several different categories, including scenario, parameter, and model uncertainty (Table 1.4).

There are two ways to address uncertainty in risk assessment. The first is an uncertainty characterization which qualitatively discusses the thought process that lead to the selection or rejection of data, estimates, and scenarios (US EPA, 1992). Additionally, a qualitative exploration of the effect of assumptions on the predicted outcome may also be conducted. Alternatively, uncertainty may be quantitatively assessed using sensitivity

analysis, analytical uncertainty propagation, probabilistic uncertainty analysis, or classical statistical methods (US EPA, 1992).

Table 1.4. Three types of uncertainty in risk assessment (US EPA, 1997; US EPA, 1992).

Type of Uncertainty	Sources	Examples
Scenario	Descriptive Errors	Incorrect or insufficient information
	Aggregation Errors	Spatial or temporal approximations
	Judgment Errors	Selections of an incorrect model
	Incomplete Analysis	Overlooking an important pathway
Parameter	Measurement Errors	Imprecise or biased measurements
	Sampling Errors	Small or unrepresentative samples
	Variability	In time, space, or activities
	Surrogate Data	Structurally-related chemicals
Model	Relationship Errors	Incorrect inference on the basis for correlations
	Modeling Errors	Excluding relevant variables

Risk Assessment for Residential Lead Exposure in St. John's

This research project does not comprehensively address hazard identification and the dose-response relationship for chronic low-level lead exposure in the residential environment, as they have been discussed extensively in the literature (ATSDR, 1999; US EPA, 1998). Instead, the primary goal is to create both worst-case and average-case exposure scenarios for residents of St. John's and to characterize the associated health risks.

A review of risk assessments conducted by commercial and governmental organizations in Canada shows that methods, and thus risk estimates, vary tremendously (OMEE, 1994;

Hilts, 1995; OME, 2002; Health Canada, 2004a; Government of New Brunswick, 2006). Therefore in order to allow comparisons with other studies this risk assessment was based on the methods outlined in "Guidance on Human Health Preliminary Quantitative Risk Assessment (PQRA)" as set out by the Contaminated Sites Program of Health Canada (Health Canada, 2004a). PQRAs calculate a series of total daily lead intakes for receptors which can then be compared to Health Canada lead intake guidelines to determine if there is a risk of negative health effects.

PQRA was developed as a standardized screening tool to facilitate comparisons between federal contaminated sites and to allow decision makers to prioritize remediation and risk management efforts. It purposely uses highly conservative parameter values as a first run. If a worst-case screening scenario produces minimal risk then no further investigation is needed; however, if a risk to human health is found in PQRA then a more detailed assessment using site and receptor specific data may be necessary before a risk management strategy can be implemented (Health Canada, 2004a). This approach and its recommended parameters form the basis of the worst-case risk assessment for St. John's. The results of this kind of assessment need to be interpreted with caution as they most likely overestimate the risk for the average resident.

Because health risks were found in the worst case scenario, the parameters in the PQRA were adjusted to produce a more realistic average exposure scenario using values from the IEUBK model described below, and data from relevant, mostly Canadian, studies. In

general the average risk assessment used median or geometric mean values for soil, dust, and water lead concentrations, soil loading factors, and the length of time spent bathing, instead of the 95th percentile values that were used in the worst case scenario.

The second portion of the risk assessment involved using the IEUBK model to estimate blood lead concentration probabilities for children under seven years of age. This model has the advantage of being designed specifically for lead exposure, and it includes a detailed modeling section for the uptake and internal distribution of lead within the body. Both worst case and average risk scenarios were run using the IEUBK model, however the model was not designed for extreme values and the blood lead results generated using these parameters are not considered plausible and are therefore only presented in Appendix C.

Within both each of the worst case and average risk assessments three different risk scenarios were explored. Early exploratory runs of the PQRA and IEUBK models indicated that the consumption of garden produce was a significant contributor to total daily lead intake and blood lead concentrations. Because not all residents of St. John's eat fruits and vegetables from a backyard garden the first scenario was run without garden produce as an additional source of lead intake. For those residents who do eat garden produce two additional scenarios were evaluated, each with a different plant uptake factor. This was done because there were conflicting recommendations regarding this parameter in the literature and the impact of changing the uptake factor greatly affected

the final lead concentration estimated for the plant. One set of uptake factors from the Multimedia, Multipathway, and Multireceptor Risk Assessment (3MRA) Modeling System (US EPA, 2003a) was recommended to Health Canada by a consulting firm (Health Canada, 2005a), and another set was taken from Boyd *et al.* (1999) because the pattern reflected plant uptake in other studies (Finster *et al.*, 2004).

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Co-authorship Statement

The preliminary soil lead survey conducted in 2003 was designed and implemented by Drs. Trevor Bell and David Liverman, Geography Department, Memorial University of Newfoundland. Thus the basic soil survey approach, with the focus on spatial analysis at the municipal and individual property level was already established; however, the candidate expanded the study by designing and conducting a preliminary indoor dust lead sampling program which included the collection of indoor dust wipes and also a questionnaire. Additionally, the candidate used the environmental data to conduct a health risk assessment for residents exposed to residential sources of lead in St. John's.

Soil samples collected during the preliminary study were collected mainly by student Melissa Putt as well as by Drs. Bell and Liverman. A select number of homeowners in St. John's were also recruited to collect their own samples for analysis in the preliminary stage. The remainder of the soil samples were collected by the candidate either directly or by another student under the supervision of the candidate. The soil samples were analyzed at the Geochemistry Lab of the Geological Survey of Newfoundland and Labrador, under the supervision of Mr. Christopher Finch. The candidate was responsible for digesting the dust samples for inductively coupled plasma mass spectroscopy (ICP-MS) analysis, which was carried out at the Trace Element Lab of the Earth Sciences Department of Memorial University, under the supervision of Ms. Pam King and Ms. Lakmali Hewa.

All data analysis was carried out by the candidate in consultation with Drs. Bell and Liverman, with additional advice from Drs. David Allison, Health Officer for the Eastern Health Region, and Dr. Veeresh Gadag, faculty member of the Department of Community Health, Faculty of Medicine, Memorial University of Newfoundland. For the risk assessment, Mr. Norm Healey (Health Risk Assessment and Toxicology Specialist, Health Canada – BC Region), Ms. Louise White (Regional Health Risk Assessor and Toxicology Specialist, Healthy Environments and Consumer Safety Branch, Health Canada – Halifax), and Ms.Jacinthe David (Health Canada, St. John's) were consulted for their expertise, but all analysis was completed by the candidate. Mapped data was generated by the candidate with the assistance of Dr. David Liverman, Mr. Larry Nolan and Mr. Andrea Bassan at the Geological Survey of Newfoundland and Labrador. The candidate was responsible for writing the manuscript with editing provided by the supervisory committee.

Chapter 2: Environmental Lead Exposure in St. John's, Newfoundland: Spatial and Temporal Analysis of Residential Soil and Dust

Introduction

The neurological and developmental effects of chronic low-level exposure to environmental lead have been well documented (Bellinger, 2004; Lanphear *et al.*, 2000; Schwartz, 1994), in particular with respect to young children who absorb lead more easily and have more direct contact with lead through hand to mouth behaviour, and playing and crawling on the ground (Bellinger, 2004; Moya *et al.*, 2004; Viverette *et al.*, 1996). The investigation into the level of environmental lead in St. John's, Newfoundland, was initially prompted by results from an earlier study on lake sediment chemistry in local catchments (Christopher, 1999). Upper layers of the sediment cores were found to be elevated in many heavy metals, with lead levels increasing from a baseline of 2 to 23 ppm in lower sediment layers to 250 to 600 ppm in upper layers over the last 200 years. Dating of core sediments suggested that the high lead levels were initially due to combustion of coal, and then later to leaded gasoline emissions. Christopher (1999) further hypothesized that since these are highly dispersed sources of pollution high metal concentrations might also be present in catchment soils.

A pilot study to investigate the lead content of urban soils in St. John's was initiated in 2003 (Bell, 2003). Soil samples were collected to represent a variety of land uses where

children may be exposed, including open spaces, parks, and school and residential properties. Areas were strategically chosen both in the older downtown region and more recently developed areas of the city. The results from 341 samples ranged between 17 and 7048 ppm, with a median value of 266 ppm (Bell, 2003). Sixty-nine percent of the samples had lead contents which exceeded the Canadian Council of Ministers of the Environment (CCME) residential soil lead guideline of 140 ppm. Nine percent of the samples were ten times the CCME guideline, the majority coming from selected residential properties in downtown St. John's.

A major objective of this paper is to establish through an expanded soil sampling program the spatial pattern of contaminated soil that exceeds the CCME residential soil lead guidelines in St. John's. Because elevated soil lead can contribute to increased indoor dust lead levels (Lanphear and Roghmann, 1997; Rutz *et al.*, 1997), another objective is to make a preliminary assessment of lead levels in indoor dust and determine the relationships between soil lead and indoor dust lead levels. Studies elsewhere have demonstrated an association between older houses and higher soil and dust lead concentrations (Rasmussen *et al.*, 2001; CMHC, 1997; Thornton *et al.*, 1994). A third objective therefore addresses the relationship between property or building age and environmental lead concentrations in St. John's. It is important to make a distinction between property age and building age. Property age refers to the length of time that piece of land has experienced human development; while building age refers to the length of time the current structure has been on that piece of land. Property age may be a proxy

indicator of soil lead levels as it reflects the aggregated contribution of any development on the property together with past accumulation from ambient leaded gasoline and coal combustion emissions. In contrast, building age may significantly influence indoor dust levels as older, non-renovated buildings likely contain interior leaded paint.

St. John's and historical sources of soil lead

St. John's, Newfoundland, is a historic port city on the east coast of Canada (Figure 2.1). The first Europeans began using St. John's harbour for a fishing port over 500 years ago, but permanent urban development only began 250 years ago on the northwest side of the harbour and continued outwards around the harbour and along major roadways (Poole, 1994; Figure 2.2). Currently, the over 100 000 residents inhabit almost 500 km^2 of land, which doubles in population and area if the surrounding metropolitan region is considered (Statistics Canada, 2007). Historically small scale commercial and industrial businesses have served the local community, but St. John's has never been a highly industrialized city. It acts as a service and political centre for the island and capital city for the Province of Newfoundland and Labrador.

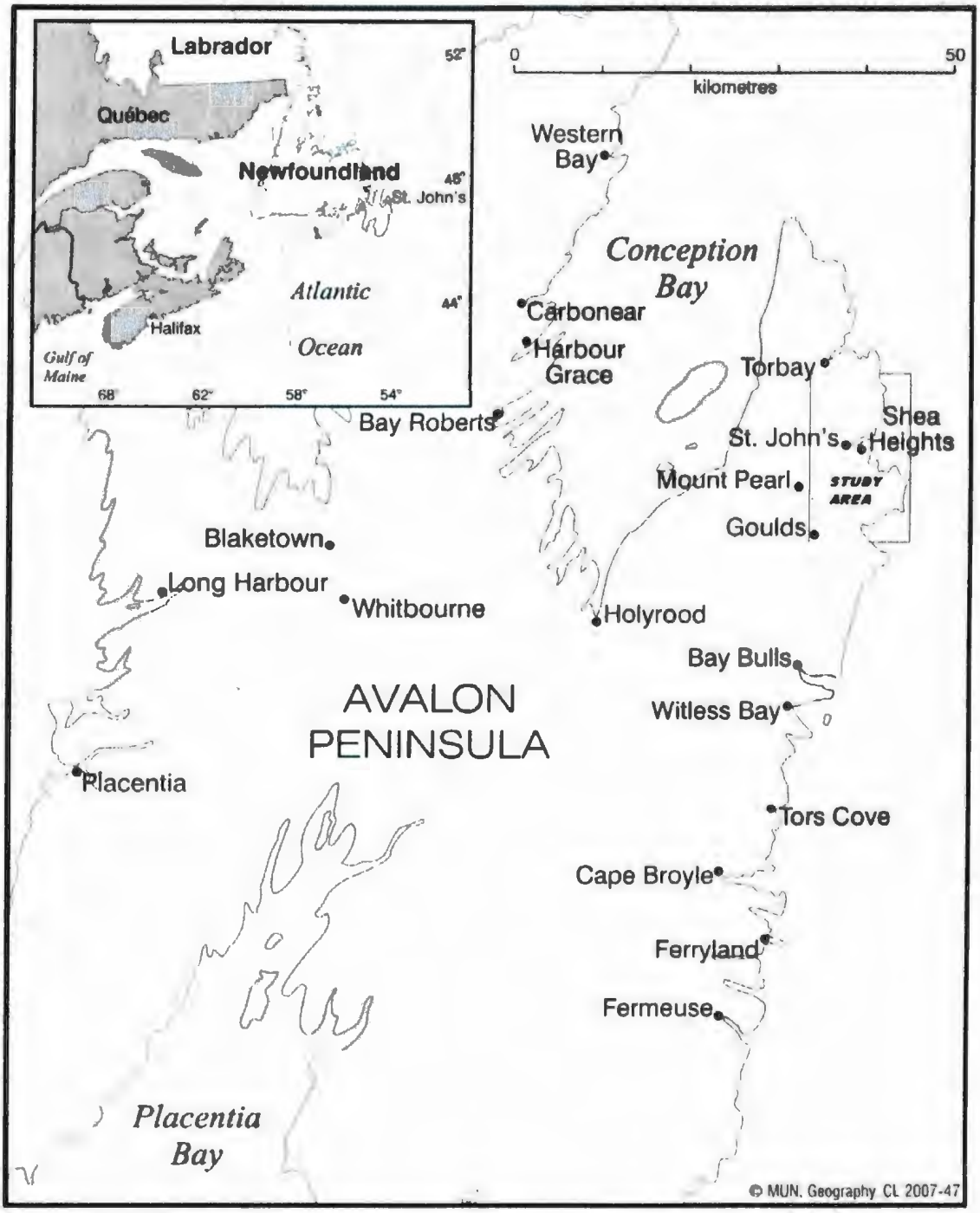


Figure 2.1. Location of study area around St. John's, Newfoundland (Memorial University of Newfoundland, 2007).

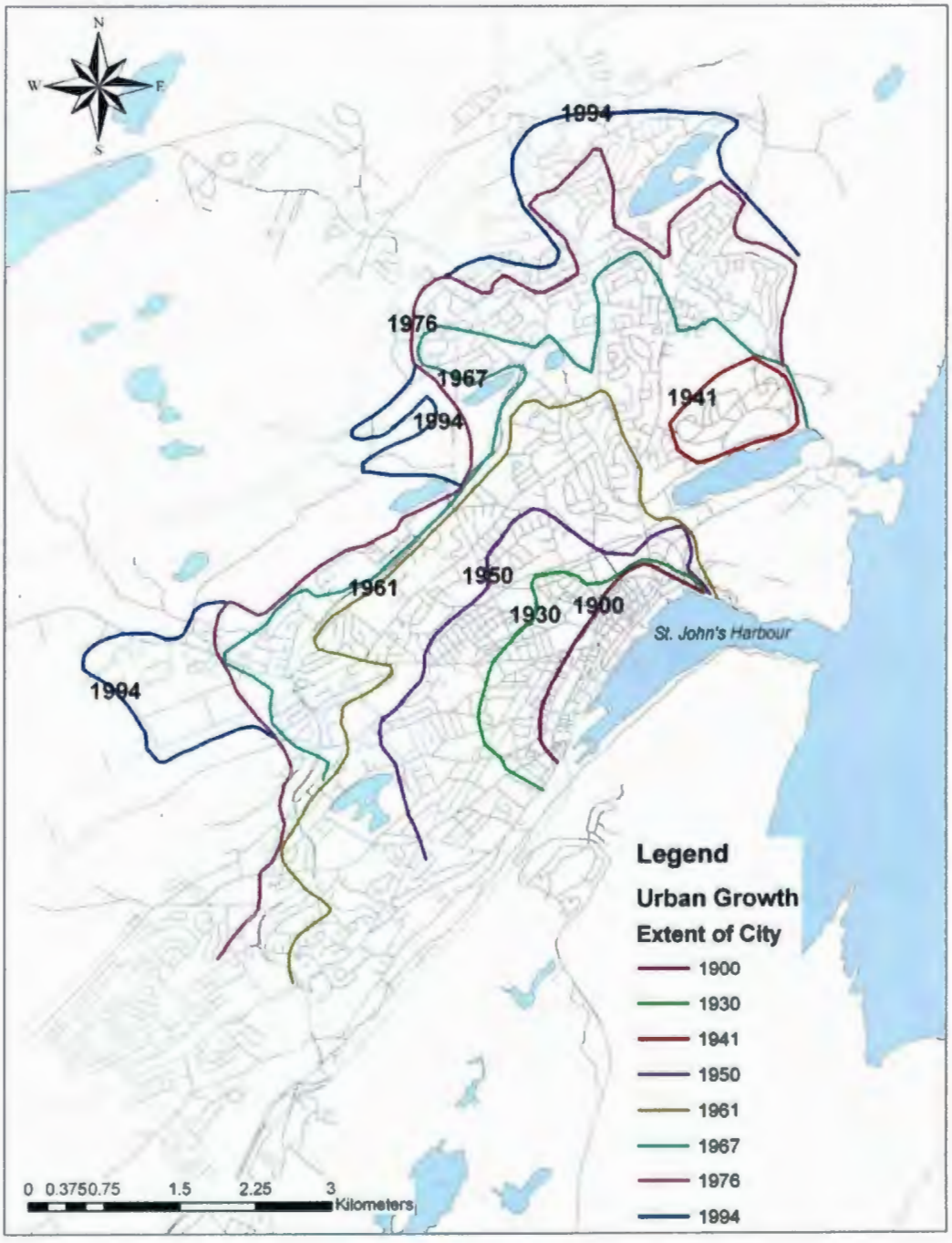


Figure 2.2. Urban growth pattern for St. John's, Newfoundland (modified from Christopher, 1999).

Low concentrations of lead occur naturally in St. John's because of the mineral composition of the underlying bedrock. Three distinct Upper Precambrian age geological groups run north-south roughly parallel to the eastern coast of the Avalon Peninsula (Figure 2.3). Most of the city, including the downtown core, lies on the middle St. John's Group which consists of grey and black shale and sandstone (Christopher *et al.*, 1993). According to lake sediment data lead levels in this group ranges from 15 to 139 ppm (Geological Survey of Newfoundland and Labrador, 2007). To the west lies the older Conception Group that is composed of chert, sandstone, conglomerate, tuffaceous siltstone and sandstone, and to the east is the Signal Hill Group that consists of conglomerate, siltstone, sandstone, and tuff. The Signal Hill Group has lower natural lead concentrations, <9 to 20 ppm in lake sediment (Geological Survey of Newfoundland and Labrador, 2007) (Figure 2.3).

The urban soils of St. John's would have elevated environmental lead levels for several reasons. The continual demolition and construction of buildings built with painted clapboard in the high-density downtown core of the city may have added large amounts of lead to the soil through the deposition of paint chips and weathered paint by-products. Additionally, several devastating fires in the 1800s and early 1900s would have generated ash and other lead-laden combustion products, which were deposited in local soils. The combustion of coal from the early 19th century to the late 20th century for both industrial and residential purposes would have added large amounts of lead into the air and directly into the soil as stove ashes were commonly disposed of in back gardens (Bell, 2003).

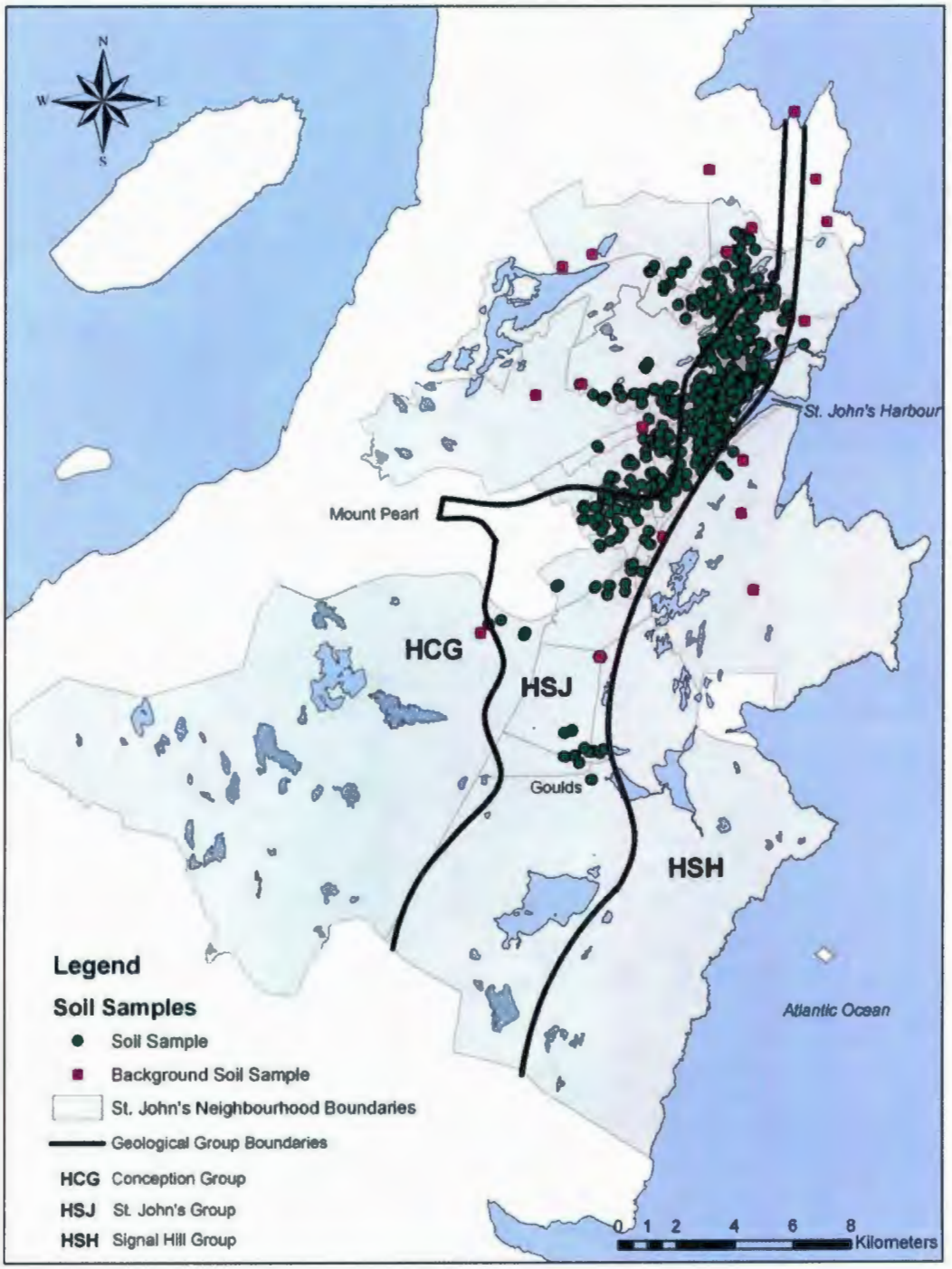


Figure 2.3. The location of background and surface soil samples overlain onto the neighbourhood structure of St. John's and the underlying geological groups (based on King, 1990 in Christopher, 1999).

Last, lead would have been introduced to the environment through emissions from motor vehicles using leaded gasoline, which first appeared in St. John's in 1903 (Poole, 1994).

Methods

Sampling Strategies

An extensive soil sampling survey was conducted between 2003 and 2005 on a variety of property types across the city of St. John's, with a particular focus on residential properties because they represent a major source of soil lead exposure for children. A subset of houses was chosen for preliminary testing of indoor dust lead levels based on the age of the dwelling, location within the city, and soil lead levels.

Data Sources

Soil Samples

While sampling strategies differed slightly between the pilot study conducted in 2003 and the expanded study conducted in 2004/2005, data from all three years are combined and analyzed in this paper. In 2003, the objective was to sample a wide range of land use types, including schools, parks, and residential properties (Bell, 2003). In addition, several transects running perpendicular from long-established roads and the exterior walls of old clapboard houses were sampled. In 2004 and 2005 the program incorporated a systematic sampling of residential properties across the city. Neighbourhood areas as defined by the Community Accounts information system of the Newfoundland and

Labrador Statistics Agency were chosen as the spatial sampling units (Community Accounts, 2007). Each of the 95 neighbourhoods was created by grouping postal code units together to generate areas of equal population, roughly 1000 people. Basic demographic data were available for each neighbourhood, including population age structure and gender, and dwelling type and age.

A minimum of three houses were sampled in each neighbourhood. An opportunistic sampling method was used with some effort made to spatially distribute the sample locations throughout the neighbourhood. Ultimately the choice of property largely depended on who was home at the time of sampling, whether or not they owned the property, and if they agreed to participate in the study. Homeowners were presented with a consent form to sign before sampling proceeded.

Multiple soil samples were taken from each property to address the question regarding lead sources and pathways. If possible for each property one sample was taken within 5 metres of the adjacent road (from now on referred as a road sample), one within 1 metre of the foundation of the house (dripline sample), and one in an open area of the property away from buildings and roads (ambient sample). Soil lead concentrations from these samples provide some indication of the relative contribution from vehicular emissions, leaded exterior paint, and ambient atmospheric deposition, respectively. A 250 ml soil sample was collected at each of the three property locations from either the surface of bare ground or the top 5-10 cm where a vegetation mat was present. A plastic trowel and

paper sampling bags were used to collect the sample, and the trowel was rinsed with water and wiped between each sampling to prevent cross-contamination of samples. Duplicate soil samples were collected at every tenth site or so.

The soil sampling methodology was based on a study conducted in New Orleans (Mielke, 1994). In this study an equal number of surface samples were collected in every census tract; 10 samples were taken within 1 metre of the street, 3 samples were taken within 1 metre of house sides, and 2 samples were collected from open spaces (Mielke, 1994). The US EPA recommends that bare soil is collected from the foundation of the house as well as children's play areas (US EPA, 2000). Other studies have collected samples only in open areas away from buildings and roadways in order to avoid localized contamination (Lambert and Lane, 2004; OME, 2002), whereas others have focused on garden soil only (Government of New Brunswick, 2005; Rasmussen *et al.*, 2001). These other studies have also combined soil samples to create one composite sample for either the entire residence or for a specific sample location (Government of New Brunswick, 2005; Lambert and Lane, 2004; OME, 2002; Rasmussen *et al.*, 2001; Hilts, 1995; US EPA, 1995). In this study only one sample per location was collected and all samples were analyzed separately.

To determine naturally-occurring or background lead levels, samples were collected from subsurface exposures of undisturbed glacial diamicton (till), both within the city limits and along urban-rural transects. In addition, surface samples were collected from many

of these sites to estimate the magnitude of anthropogenic input at the surface and to determine the extent of atmospheric lead precipitation away from the city. This process was necessary to ensure that the elevated lead concentrations measured in surface soil were not from natural sources of lead in the underlying bedrock.

Soil samples were analyzed at the Geological Survey of Newfoundland and Labrador's geochemical laboratory in St. John's and followed standard protocols for sediment analysis (Finch, 1998). Samples were oven-dried, sieved to less than 180 μm, and digested with a mixture of hydrochloric, hydrofluoric, and perchloric acids. Samples were then analyzed for a wide array of elements, including lead, using inductively coupled plasma-emission spectrometry (ICP-ES). This was a complete digestion yielding total lead concentration. Canadian certified reference materials were analyzed with the samples for data comparison. Every 20th sample was split in the laboratory and run as a duplicate to assess analytical precision.

Indoor Dust Samples

Dust lead samples were collected from a small sub-sample of houses originally tested for soil lead levels. Houses in the soil lead database were grouped into 11 age categories: one for each decade in the 20th century and one for houses built before 1900. New houses built since 2000 were included in the 1990-1999 category. In general three houses were sampled for house dust from within each category. All sampling was conducted during a

two week period at the end of September 2005 when indoor dust lead levels from the tracking-in of soil or blowing in of outdoor dust through open windows were likely to be at a maximum.

Interior dust was collected using *Ghost Wipes* purchased from the Maine State Health and Environmental Testing Laboratory as per US EPA protocol (US EPA, 1995). The main limitation of using the wet wipe method is that only the dust lead loading (the amount of lead per area wiped), not actual concentration of lead in the dust can be determined. Nevertheless some studies have indicated that dust lead loadings are a better predictor of childhood blood lead levels than dust lead concentrations, especially on non-carpeted surfaces (Lanphear *et al.*, 1995; Yiin *et al.*, 2000). One deviation from standard protocol was that samples were collected in sealable plastic bags, not centrifuge tubes. Three samples were collected at each house: one from the most frequently used entrance floor, one from the kitchen floor, and one from a window sill of a frequently opened window. A square plastic 0.25 m^2 template was used to guide the floor sample collection, which was wiped from left to right to left again in an "S" pattern with overlapping passes. The wipe was then folded in half and the area was wiped in the same manner in the opposite direction, from top to bottom. The wipe was then sealed in a labeled plastic bag. The template was rinsed with water and fresh latex gloves used at every new location sampled within the home to prevent cross-contamination. For sampling of window sills, the total area of the sill was measured and wiped. Field duplicate and blank wipes were collected

after every tenth sample, with duplicate samples collected directly beside the original sample.

Most houses had linoleum or tile floors, but frequently mats were present in entrance ways. In general these were avoided and sampling was done as much to the front or side of the mat, while remaining within 1 m of the door. In order to have enough floor space for the template in some entrance ways, mats were lifted and samples collected from underneath. In these cases, some material in the mat may have preferentially accumulated on the sampling area, whereas in others where the mat had a rubber or cloth backing there was likely less dust on the floor beneath. These situations may have resulted in over and under representation of lead loading values for the entrance ways, respectively.

Participants were asked to refrain from cleaning the selected surfaces for a few days prior to sampling to ensure there would be sufficient dust to collect. For comparative purposes, the number of days since a sampling surface was last cleaned was recorded, together with a visual inspection of the degree of dustiness. The type of sill surface and its general condition was noted to distinguish between a plastic window sill and one that was painted and peeling. Some homeowners mistakenly neglected the cleaning instructions, especially the kitchen floor, and this may have resulted in under representation of lead loading on these surfaces.

Indoor dust wipes were digested according to the US EPA "Modified SW-846 Method 3050A Acid Digestion Procedure for Single-Wipe Samples" using nitric acid and hydrogen peroxide (US EPA, 1996). There were a few deviations from the standard protocol; samples were digested in 150 ml not 250 ml beakers, and were made up to a final weight of approximately 50 g in centrifuge tubes, not the recommended final volume of 100 ml in volumetric flasks, as outlined in the procedure. Water standards, laboratory reagent blanks, wipe blanks and wipe duplicates were digested by the same procedure. Digested samples were analyzed for the full suite of trace elements by inductively coupled plasma-mass spectrometry (ICP-MS) at the geochemistry laboratory in the Earth Sciences department at Memorial University.

Indoor dust lead was reported by the laboratory in concentrations of parts per billion (ppb). This concentration does not reflect the amount of lead per mass of dust, as it would be extremely difficult to accurately measure the mass of dust collected on a wet wipe, but rather the amount of lead in the digested sample. The laboratory concentrations were transformed into dust lead loadings ($\mu g/ft^2$), the amount of lead per area sampled, using the following equation:

$$LeadLoading = \frac{\left(\frac{C * Mf}{A}\right)}{2.691} \qquad [1]$$

where C represents the concentration reported by the laboratory (ppb or $\mu g/kg$), Mf is the final mass of the diluted sample after digestion (kg), and A is the area wiped (m^2). The constant value of 2.691 changes the area from square metres to square feet, the loading

units used by US EPA to set guidelines for indoor dust levels. Prior to data analysis, the average amount of background lead in clean wipes was subtracted from the measured loadings to reflect the actual amount of lead on the sampled surface.

The data were logarithmically transformed to improve the distribution and normalize the residuals prior to statistical analysis.

Property and Dwelling Age

Dwelling and property ages provided by owners were verified using aerial photographs from the Newfoundland and Labrador Department of Environment and Conservation and historic fire insurance maps from the City of St. John's. While houses were originally grouped into 11 categories based loosely on decade, age data were finally reduced to six age categories based on the dates of available aerial photographs and insurance maps in order to coincide with important dates in the history of lead use in the city (Table 2.1). Aerial photographs were available for much of the sampling area from 1948 to 2001 and insurance maps were available from 1880 to1963, but only for houses within city limits at the time of mapping. The age of houses built before 1948 outside the contemporary city limits could not be independently verified and therefore the housing age given by the homeowner was assumed to be accurate and used for both the age of dwelling and the age of property.

Table 2.1. Final dwelling and property age categories based on important dates in the history of lead use and the dates of available maps and photographs used to verify the age of sampled properties.

Final Age Category	Important Dates	Dates of Maps and Photographs
< 1926	• Lead carbonate, the main ingredient of lead based paint, sold separately as a wood preservative. Mainly for wooden ships, but also potentially used on clapboard houses	1880, 1889, 1893 1902, 1914, 1925
1926 – 1948	Lead added to gasoline in mid 1920s [a]	1947, 1948
	Maximum concentrations of lead in paint in the 1940s, up to 50% by weight [b]	
1949 – 1960	Coal use in home heating slowly replaced by oil in 1950s in St. John's	1951, 1960
	Lead slowly becomes more common in exterior than interior paints [b]	
1961 – 1976	Lead in paint less significant after 1960 [c]	1966, 1973, 1976
1977 - 1992	Unleaded gasoline introduced in 1975 [a]	1978, 1981, 1985, 1992
	Lead concentration in interior paints limited to 0.5% by weight in 1976 [b]	
1993 - present	Leaded gasoline banned in Canada in 1990 [a]	1995, 2001
	Lead voluntarily limited in consumer paints by Canadian Paint and Coating Association [b]	

[a] Health Canada (2004a)
[b] Health Canada (2004b)
[c] Canadian Housing and Mortgage Corporation (2007)

To facilitate the integration of study results with neighbourhood demographic and housing data provided by the provincial statistics agency, there was a further modest adjustment required in the housing age categories (Table 2.2).

Table 2.2. How the confirmed dwelling or property ages were combined according to the age categories used by the Community Accounts information system.

Community Accounts Age Categories	Dates of Maps and Photographs
- 1946	1880, 1889, 1893, 1902, 1914, 1925
1946 - 1960	1947, 1948, 1951, 1960
1961 - 1970	1966, (some 1973)
1971 - 1980	1973, 1976, 1978, (some 1981)
1981 1990	1981, 1985, (some 1992)
1991 2001	1992, 1995, 2001

Results

Analysis of laboratory and field duplicate soil samples indicates that laboratory methods are precise and field samples are representative of local soil and indoor dust conditions (Appendix 1). Because of the skewed nature of the data, logarithmic transformations were used to improve data distribution; however, the Anderson-Darling test indicated that normality was still not achieved ($p<0.05$). Nevertheless geometric means and their associated confidence intervals are used instead of medians because they better represent the data.

There are several limitations to the soil and dust lead study in St. John's. First, samples were only collected from houses where homeowner consent could be obtained from residents, so very few rental properties were included. Rental status has been associated with higher blood lead levels, which may reflect both lower building maintenance levels (i.e., deteriorating leaded paint may be more common) and an increased representation of

low income families who may be more prone to poorer nutrition and consequently additional risk for elevated blood lead (Lanphear *et al.*, 2002).

Second, the number of houses sampled for each housing age category is not representative of the housing distribution in St. John's (Figure 2.4). The soil and dust surveys sampled a higher percentage of older homes built before 1948 (Figure 2.5). As a result summary lead concentrations calculated for the city as a whole over-represent values from older homes, which may produce higher city-wide averages.

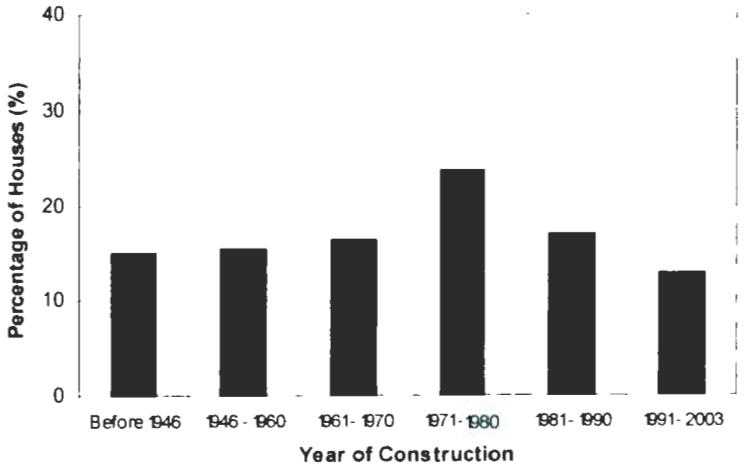


Figure 2.4. The percentage of houses built in St. John's during specific time periods (Community Accounts, 2007).

Figure 2.5. The percentage of houses sampled in the soil and dust surveys built in St. John's during specific time periods.

Soil Lead

Background Soil Lead Concentrations

Twenty-nine subsurface soil samples were collected from 19 different rural and suburban locations around St. John's. Sites included construction areas where soil profiles were exposed during foundation excavation, soil pits dug five to ten kilometres away from the city centre along arterial roadways, and a gravel pit near the town of Whitborne, roughly 90 kilometres west of St. John's (Figure 2.1). Sampling depths ranged between 16 and 130 cm with an average of 45 cm. The geometric mean lead concentration of the 29 subsurface soil samples is 17 ppm (95% CI; 15-20 ppm) (Table 2.3).

Table 2.3. Descriptive statistics for the concentrations of background soil lead samples.

Background Sample Type	n	Average Depth (cm)	Mean (ppm)	SE of Mean (ppm)	GMean* (ppm)	Min (ppm)	25th (ppm)	50th (ppm)	75th (ppm)	Max (ppm)
Rural and Suburban Surface Soil	18	6	51	11	37	9	19	36	70	171
Rural and Suburban Sub-Surface Soil	29	45	19	2	17	8	13	17	22	45
Urban Sub-Surface Soil	21	174	255	71	111	18	37	73	487	1107

*Geometric Mean

In contrast, the geometric mean lead concentration for the 18 surface soil samples from rural and suburban settings is 37 ppm (95% CI; 25-55 ppm), more than double the subsurface concentrations (Table 2.3). This indicates that surface lead concentrations are truly elevated compared to natural underlying concentrations of lead in the bedrock. Three surface samples have concentrations greater than 100 ppm and one sample (171 ppm) exceeds the CCME guidelines for lead in residential soil. Most of the sites with elevated surface lead concentrations are located within walking distance of long-established arterial roads, which suggests a leaded gasoline source for the soil lead in these rural/suburban settings.

The variability in surface soil lead concentrations appears to reflect distance from downtown St. John's and distance of the sampling site from the road. For example, along rural roads surface concentrations are similar to subsurface concentrations (Southland Road in Figure 2.6); however along busier and older roads surface concentrations are

higher than subsurface levels (in Figure 2.6). The Southland road samples were collected one metre from a minor road and were farthest from downtown St. John's (approximately 10 km), whereas Portugal Cove Road and Thorburn Road samples were closer to the city centre by 2 and 5 km, respectively. The substantial difference between concentrations for samples from the Thorburn and Portugal Cove road sites likely reflects distance of sampling site from the road, 125 m for the former and only 4 m for the latter.

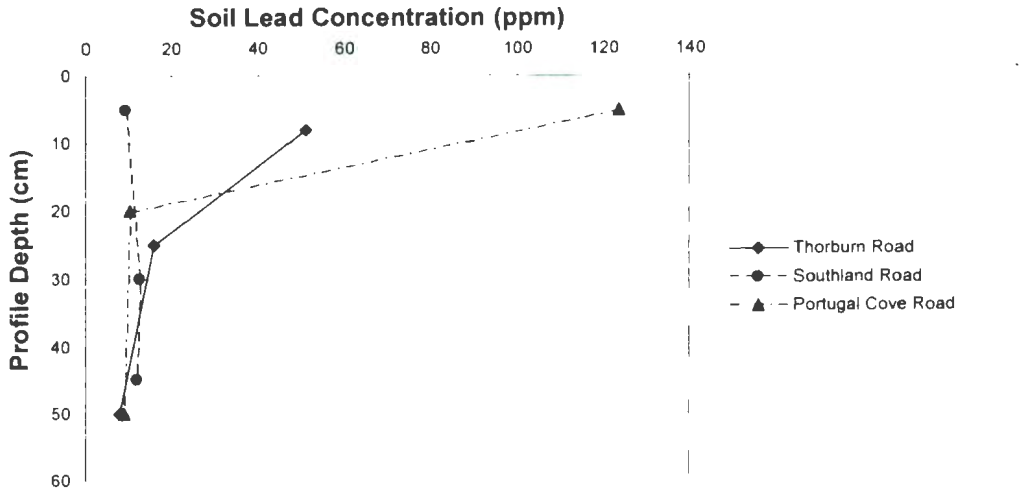


Figure 2.6. The effect of sample depth on soil lead concentrations for three rural soil profiles.

Twenty-one subsurface soil samples from a depth of 0.25 to 5.20 m at six different urban sites have a geometric mean of 111 ppm (95% CI; 62-199 ppm); however, there is high variability in the data, likely indicating deeper penetration of anthropogenic disturbance than originally considered (Table 2.3). Not all profiles were deep enough to sample undisturbed till because soil lead concentrations remain relatively high compared to rural areas, even at a depth of 0.5 m (York Street in Figure 2.7). The lowest concentration in downtown St. John's is 37 ppm and was recorded at a depth of roughly 3.5 m, whereas

farther away from the downtown, in residential areas developed in the 1950s, lead concentrations of 18 and 27 ppm were found at 2 m depth (Argyle Street is one example in Figure 2.7). The significant difference between the lowest subsurface (18 to 73 ppm) and surface (494 to 1748 ppm) lead concentrations in each urban soil profile strongly suggests that lead levels are elevated in St. John's.

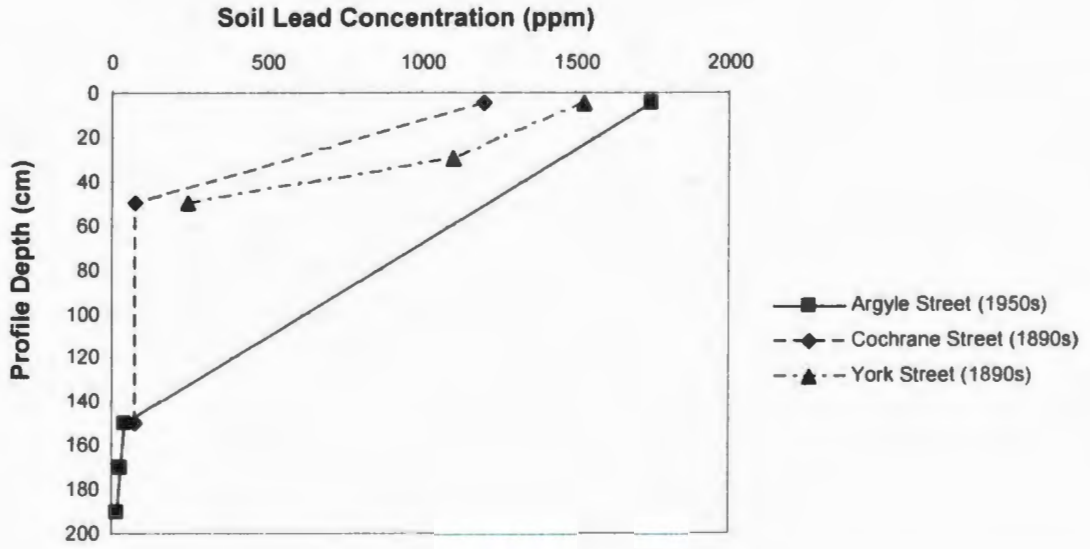


Figure 2.7. The effect of sample depth on soil lead concentrations for three urban soil profiles from urban communities of varying age.

Subsurface soil lead values were also analyzed by underlying rock type to explore the influence of bedrock geology on soil lead concentrations (Table 2.4). The geometric mean soil lead concentration for urban subsurface samples overlying the St. John's Group is 37 ppm, almost twice that of all other groups, including those samples overlying the St. John's Group in rural/suburban settings. Samples overlying the Conception Group have the lowest associated soil lead values of the three bedrock groups.

Table 2.4. Descriptive statistics for subsurface soil lead concentrations measured above the three geological groups underlying the city of St. John's.

Geological Group	n	Mean (ppm)	SE of Mean (ppm)	GMean* (ppm)	Min (ppm)	25th (ppm)	50th (ppm)	75th (ppm)	Max (ppm)
St. John's - Urban	5	41	9	37	18	23	37	61	73
St. John's - Rural/Suburban	5	23	2	23	16	19	22	29	30
Conception	17	16	1	15	8	11	16	19	32
Signal Hill	7	24	4	22	12	15	21	29	45

*Geometric Mean

Descriptive Statistics for the City

In total 1231 surface soil samples were collected and analyzed between 2003 and 2005, 514 from open spaces away from buildings and roads (ambient), 328 from along the foundation of buildings (dripline), and 389 from along roadways (Table 2.5). Because the total included samples collected in the pilot study the distribution between sample categories is uneven. The pilot study focused on open spaces, parks, school properties, residential properties, and roadways and therefore a greater number of samples were collected from these settings. The number of road side samples is lower than anticipated because row houses in downtown St. John's are located adjacent to the road and commonly there is no exposed soil to sample.

Table 2.5. Descriptive statistics for lead concentrations of the three soil sample categories in St. John's.

Sample Type	n	Mean (ppm)	SE Mean (ppm)	GMean* (ppm)	Min (ppm)	25th** (ppm)	50th** (ppm)	75th** (ppm)	Max (ppm)
Ambient	514	411	40	154	9	50	138	424	12738
Dripline	328	766	112	219	15	57	194	831	24 477
Road	389	222	12	136	16	57	136	306	1765
All	**1231**	**446**	**35**	**162**	**9**	**55**	**148**	**415**	**24 477**

* Geometric Mean
** Percentile

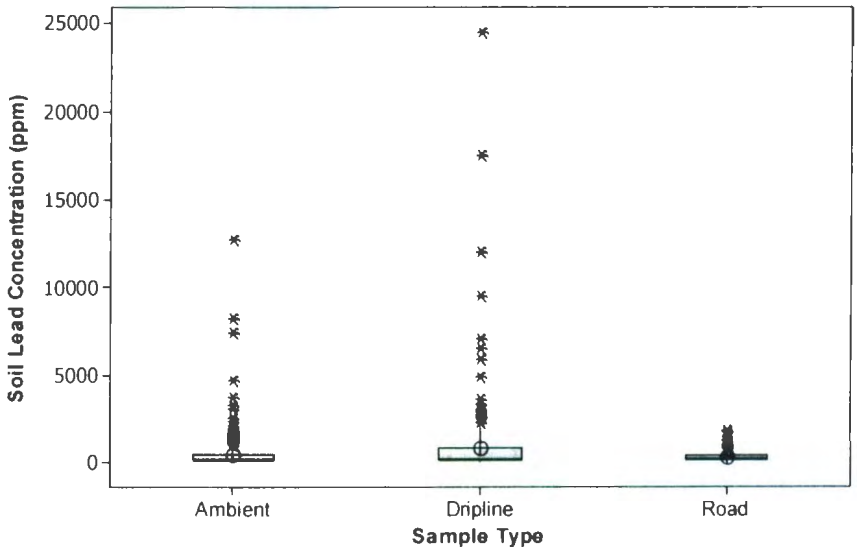


Figure 2.8. Box plots of the descriptive statistics for the three different soil sample types taken in St. John's. The bottom line of the grey box represents the first quartile of the data, the middle line represents the median, and the top line of the box represents the third quartile of the data. Two whiskers protrude from the box. The upper whisker extends to the maximum data point within 1.5 box heights from the top of the box, while the lower whisker extends to the minimum data point within 1.5 box heights from the bottom of the box. The asterisks represent outlier data that are beyond the upper or lower whisker. The circle represents the mean value. Not all symbols are visible in this figure because the large scale.

The lead concentrations are highly skewed with 50% of the samples below 147 ppm and 10% above 1000 ppm (Table 2.5). Dripline samples have the highest geometric mean soil lead concentration followed by ambient then roadside samples. Both dripline and ambient samples have a wide range of soil lead concentrations whereas road samples have concentrations more or less below 1200 ppm (Figure 2.8 and Figure 2.9). The majority of highly elevated soil samples come from dripline and to a lesser extent ambient locations. Fifty one percent of all soil samples exceed the CCME residential soil lead guideline of 140 ppm, 26% exceed the 400 ppm US EPA guideline for soil in children's play areas, and 9% exceed the 1200 ppm US EPA guideline for soil outside play areas (Figure 2.9).

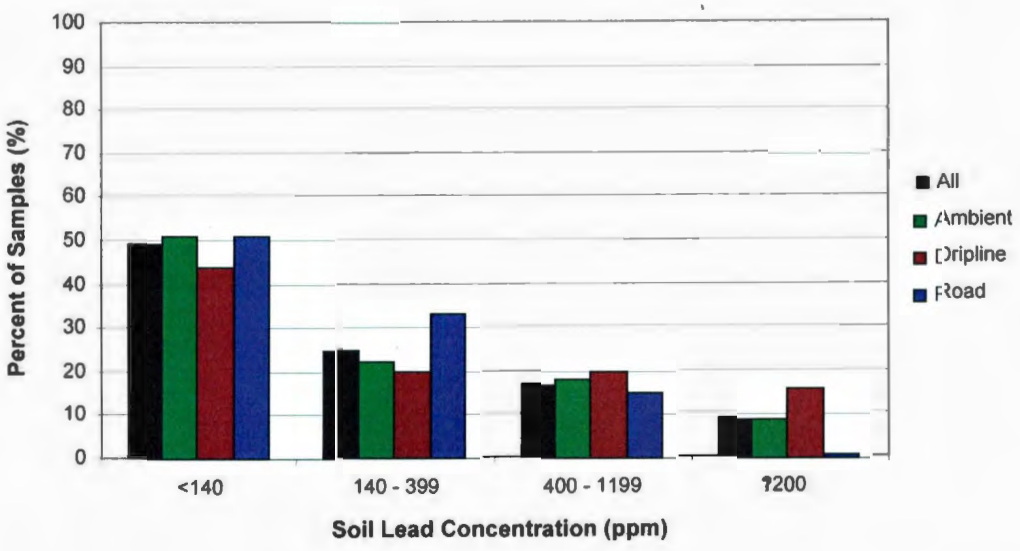


Figure 2.9. The percentage of soil samples with lead concentrations below and above the CCME residential soil lead guideline of 140 ppm, and the US EPA guidelines for bare soil in play areas (400 ppm), and bare soil not in play areas (1200 ppm).

Spatial Distribution

The spatial distribution of soil lead concentrations show a clustering of high lead samples in the downtown core (Figure 2.10). This is especially true for lead concentrations above 1200 ppm. In contrast, samples with soil lead concentrations below 140 ppm are found throughout the city. The map pattern of ambient sample concentrations is similar to the one for the full dataset except that there are fewer samples with low lead concentrations in the downtown core and fewer samples with high lead concentrations in suburban areas (Figure 2.11). Almost every dripline sample taken downtown is above the CCME soil lead guideline of 140 ppm, and high lead concentrations outside the downtown core are typically from dripline locations (Figure 2.12). Only four roadside sample sites exceed 1200 ppm, and they are found in both the downtown and the suburbs (Figure 2.13). Unlike dripline and ambient samples, road samples below the CCME guideline are common in the downtown area.

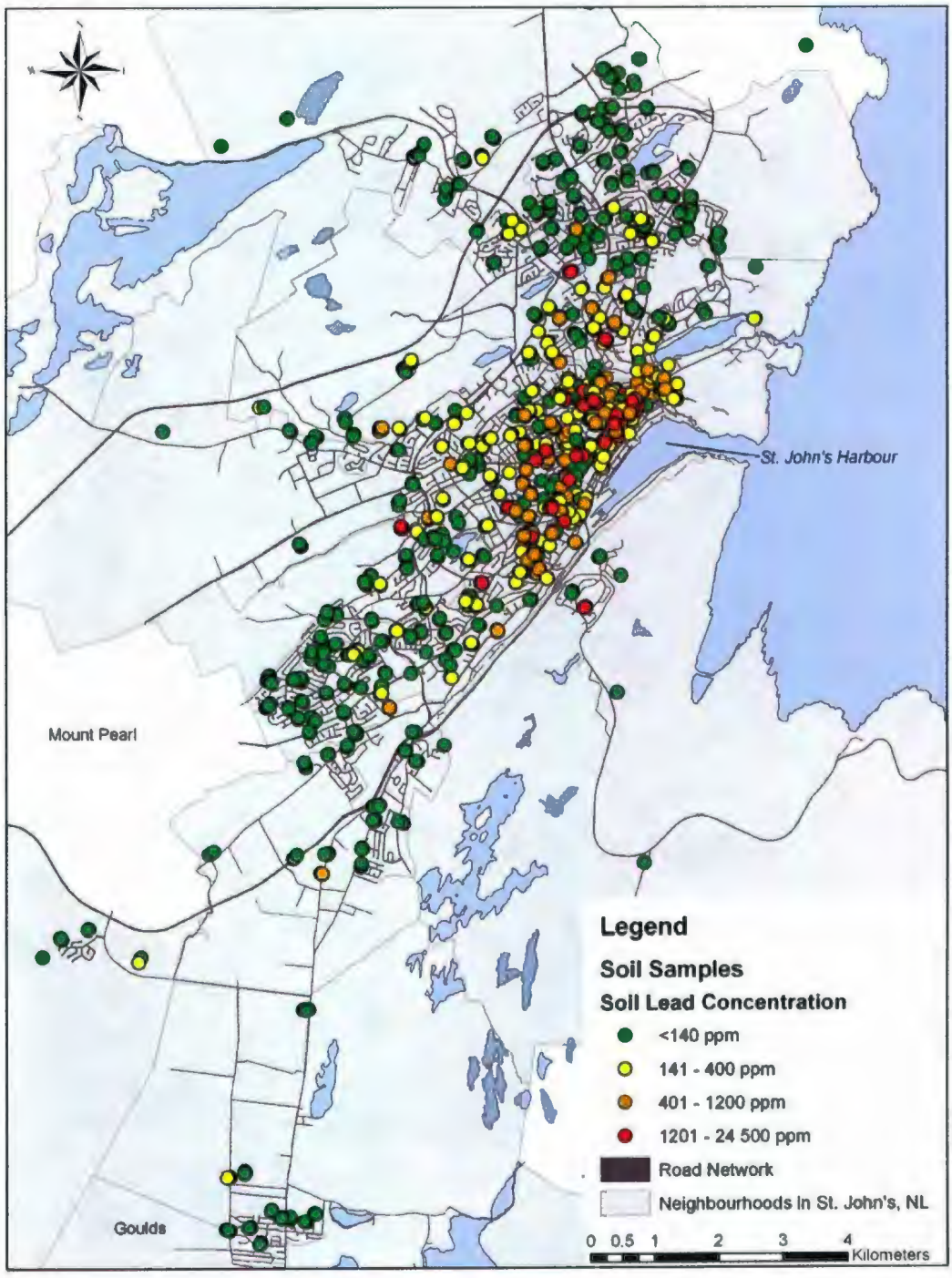


Figure 2.10. The spatial distribution of all soil samples according to soil lead concentration.

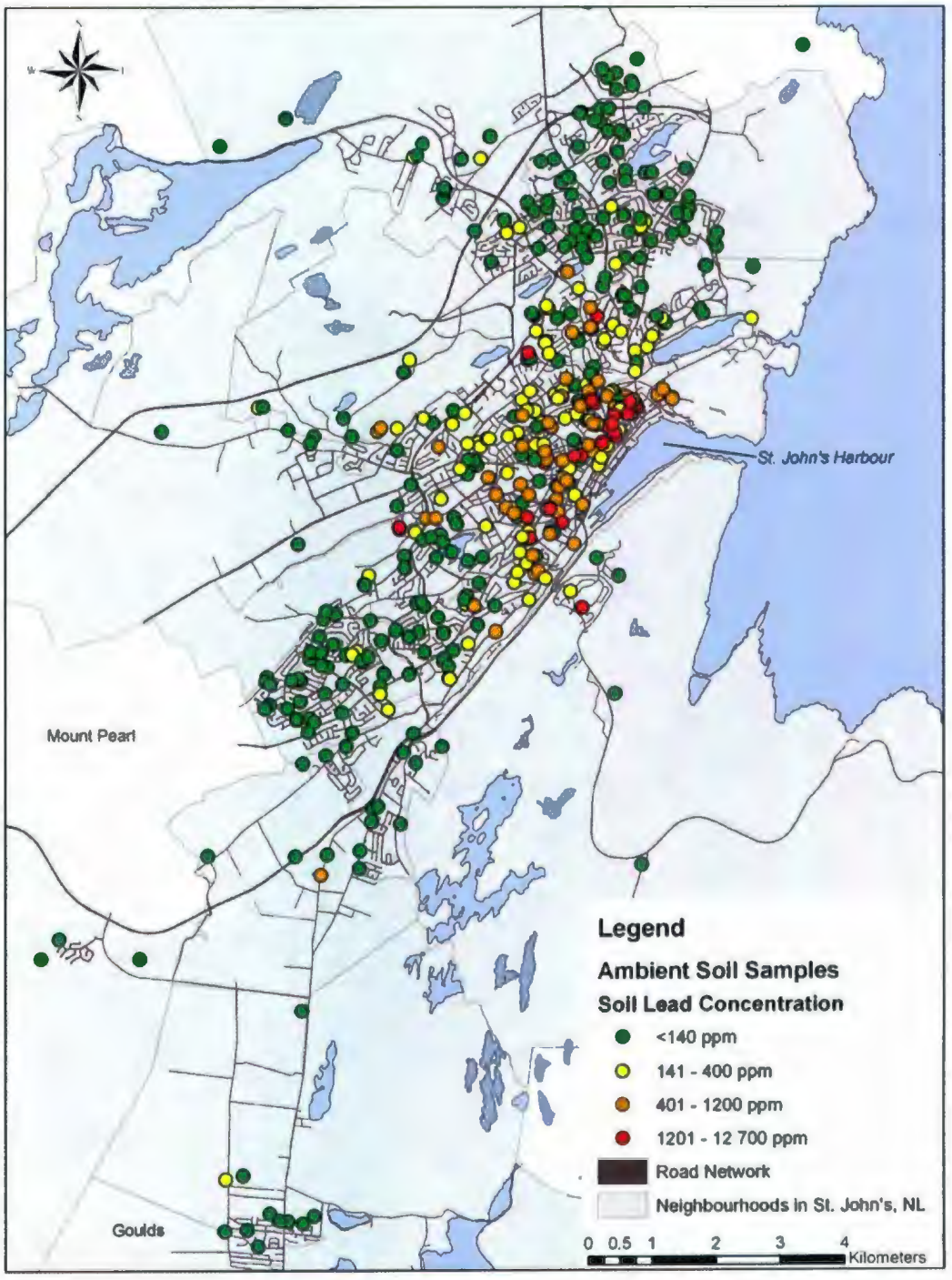


Figure 2.11. The spatial distribution of ambient soil samples according to soil lead concentration.

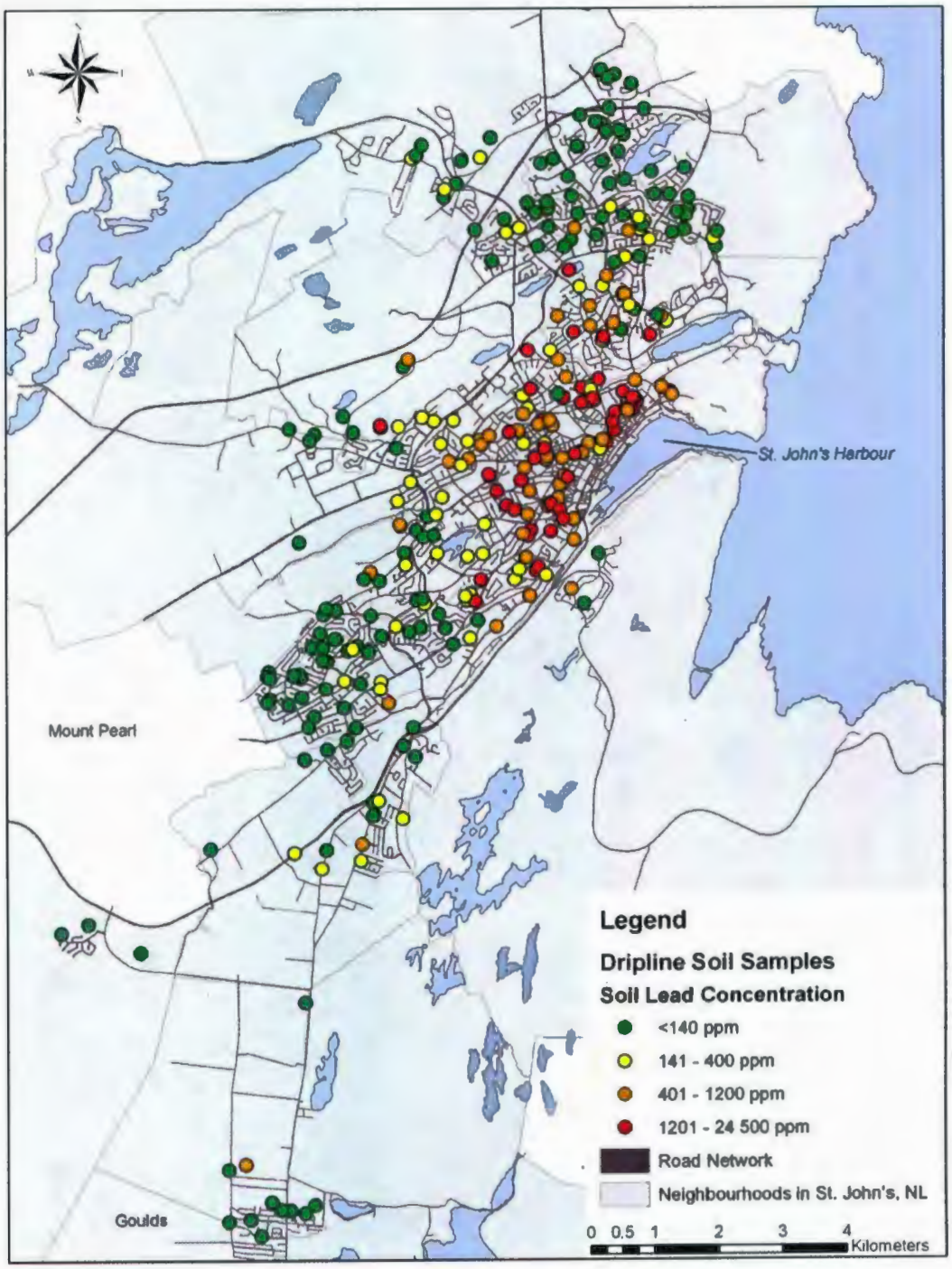
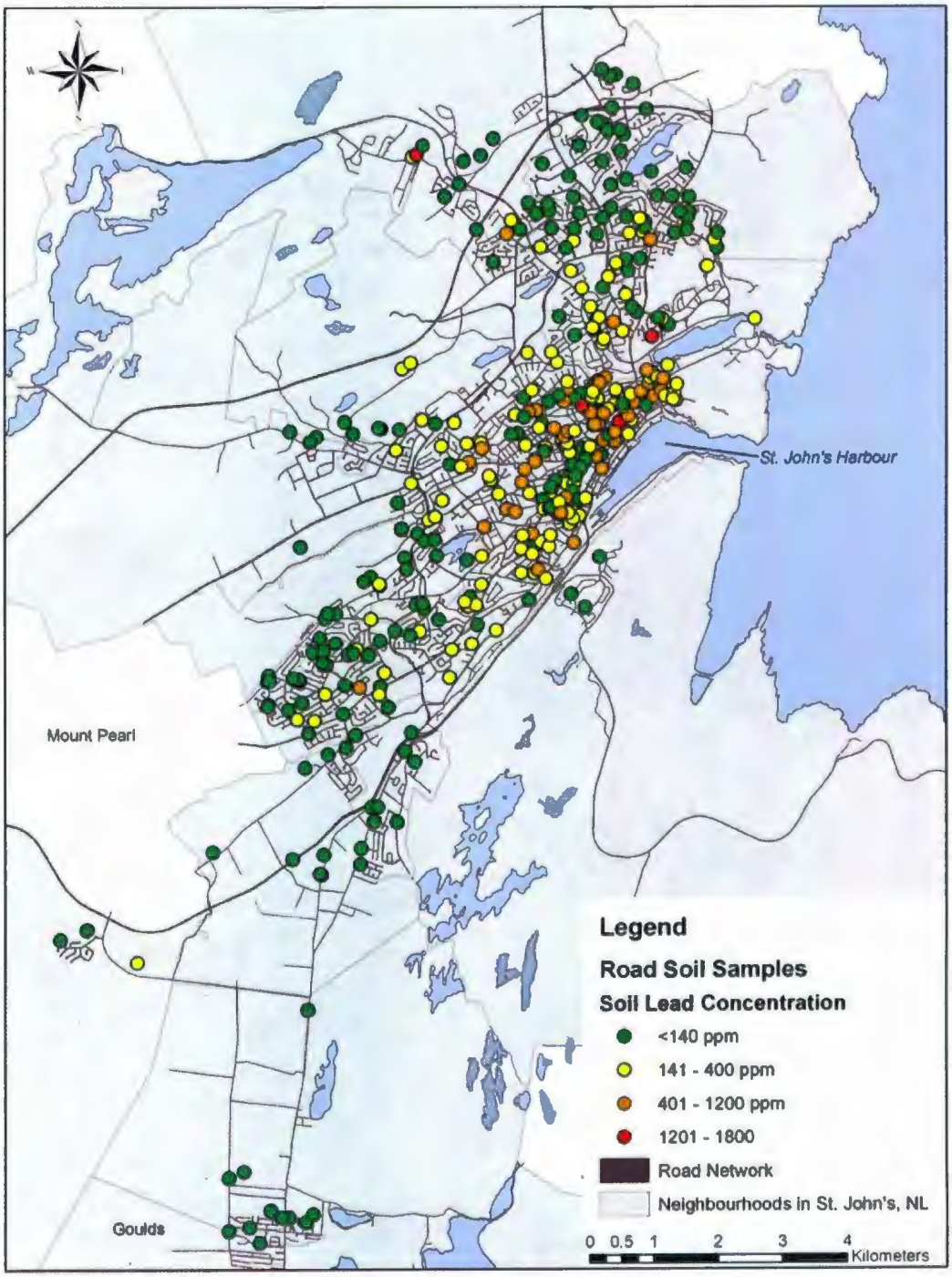


Figure 2.12. The spatial distribution of dripline soil samples according to soil lead concentration.

Figure 2.13. The spatia



distribution of road samples according to soil lead concentration.

Neighbourhood Soil Lead Levels

Neighbourhoods with the highest geometric mean soil lead concentrations for all sample types were concentrated in the downtown core (Figure 2.14). The lead concentrations in these neighbourhoods in general exceeded the US EPA's guideline for soil in children's play areas (>400 ppm). Those neighbourhoods with concentrations below this level, but above the CCME guideline of 140 ppm were distributed around the downtown core and scattered in suburban areas. Neighbourhoods with concentrations below the CCME guideline were largely restricted to suburbs and outlying areas.

On the basis of geometric mean soil lead concentrations for all sample types, 43% of neighbourhoods in St. John's exceed the CCME guideline of 140 ppm, 15% exceed the US EPA guideline of 400 ppm, whereas no neighbourhoods exceed the 1200 ppm US EPA guideline for bare soil outside play areas. There were noticeably fewer neighbourhoods with geometric mean soil lead concentrations above 400 ppm based on road samples alone (Figure 2.17) compared with those based solely on ambient or dripline samples (Figures 2.15 and 2.16). When dripline samples are considered separately, over 10% of St. John's neighbourhoods exceed the 1200 ppm guideline (Figure 2.18), representing more than half the downtown neighbourhoods (Figure 2.16).

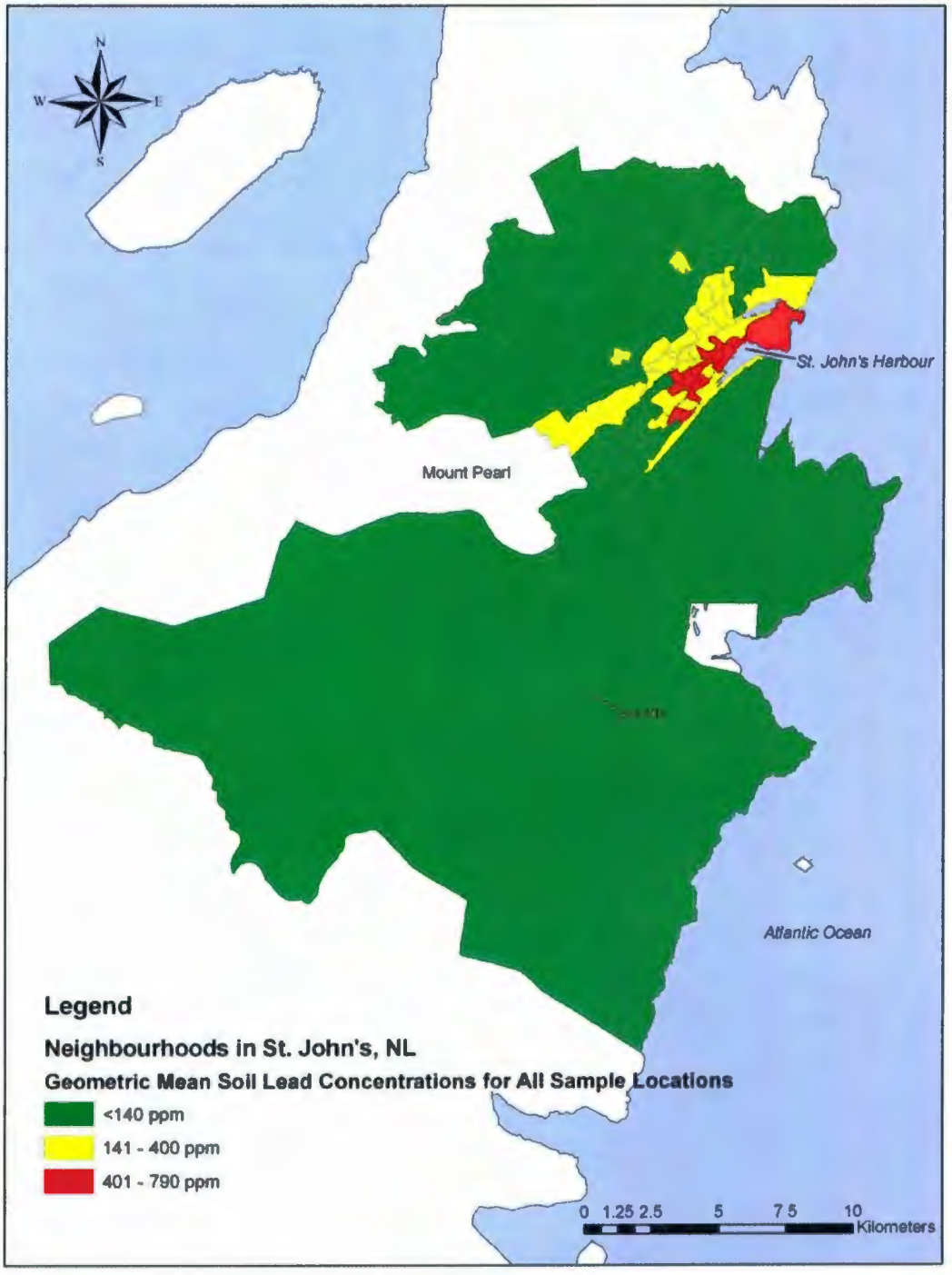


Figure 2.14. Neighbourhood soil lead concentrations based on the geometric mean of all samples

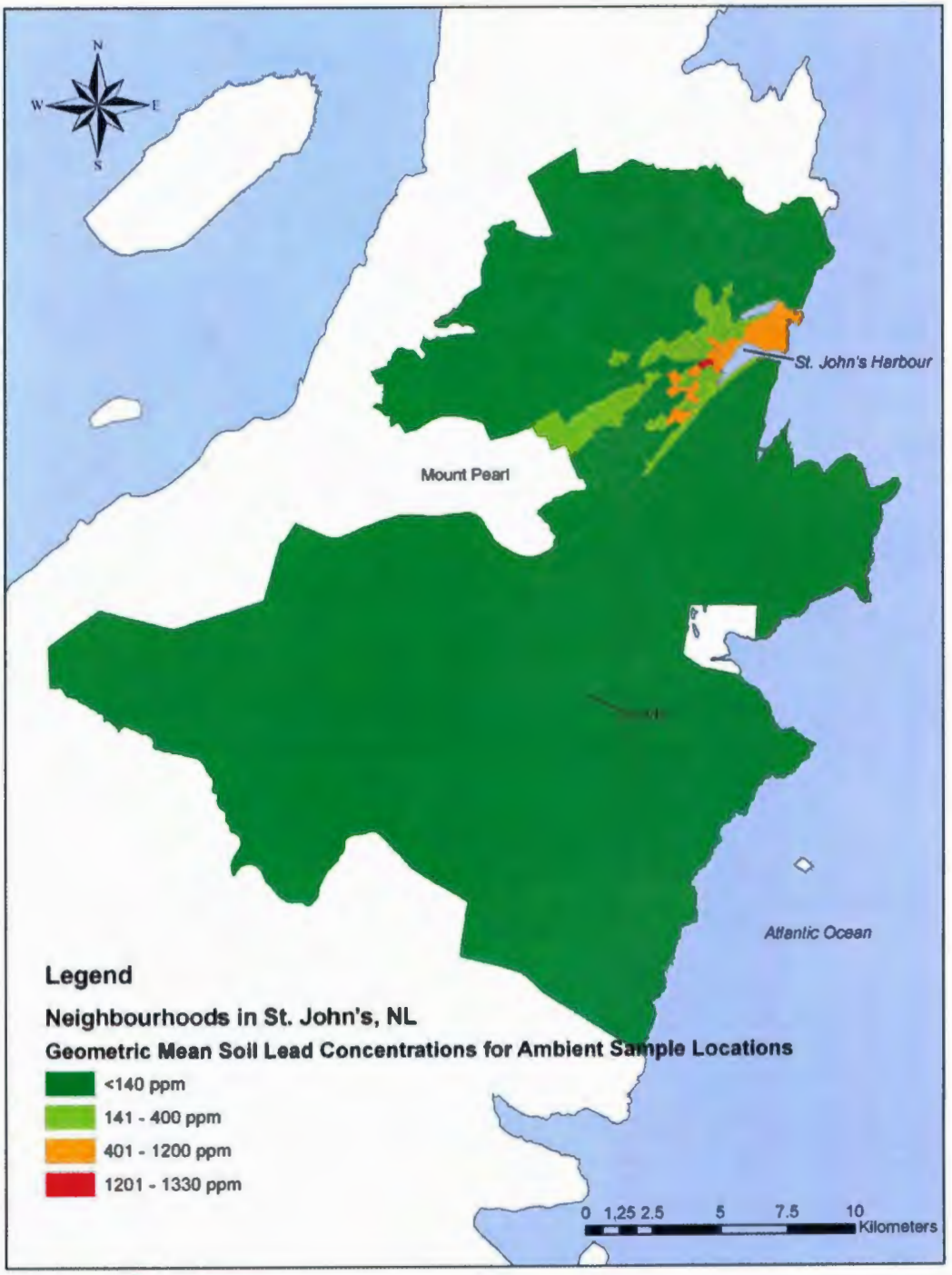


Figure 2.15. Neighbourhood soil lead concentrations based on the geometric mean of ambient samples.

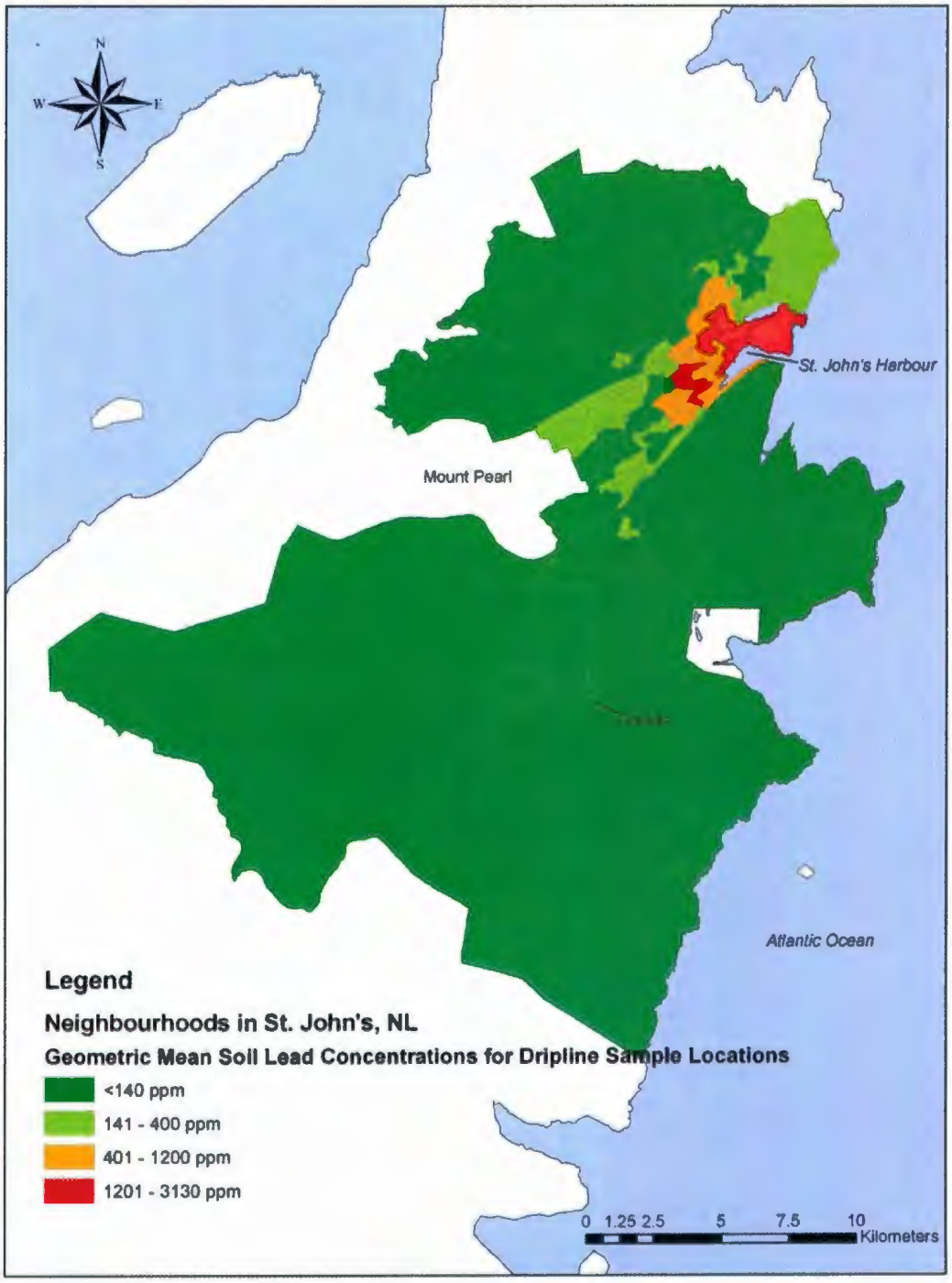


Figure 2.16. Neighbourhood soil lead concentrations based on the geometric mean of dripline samples.

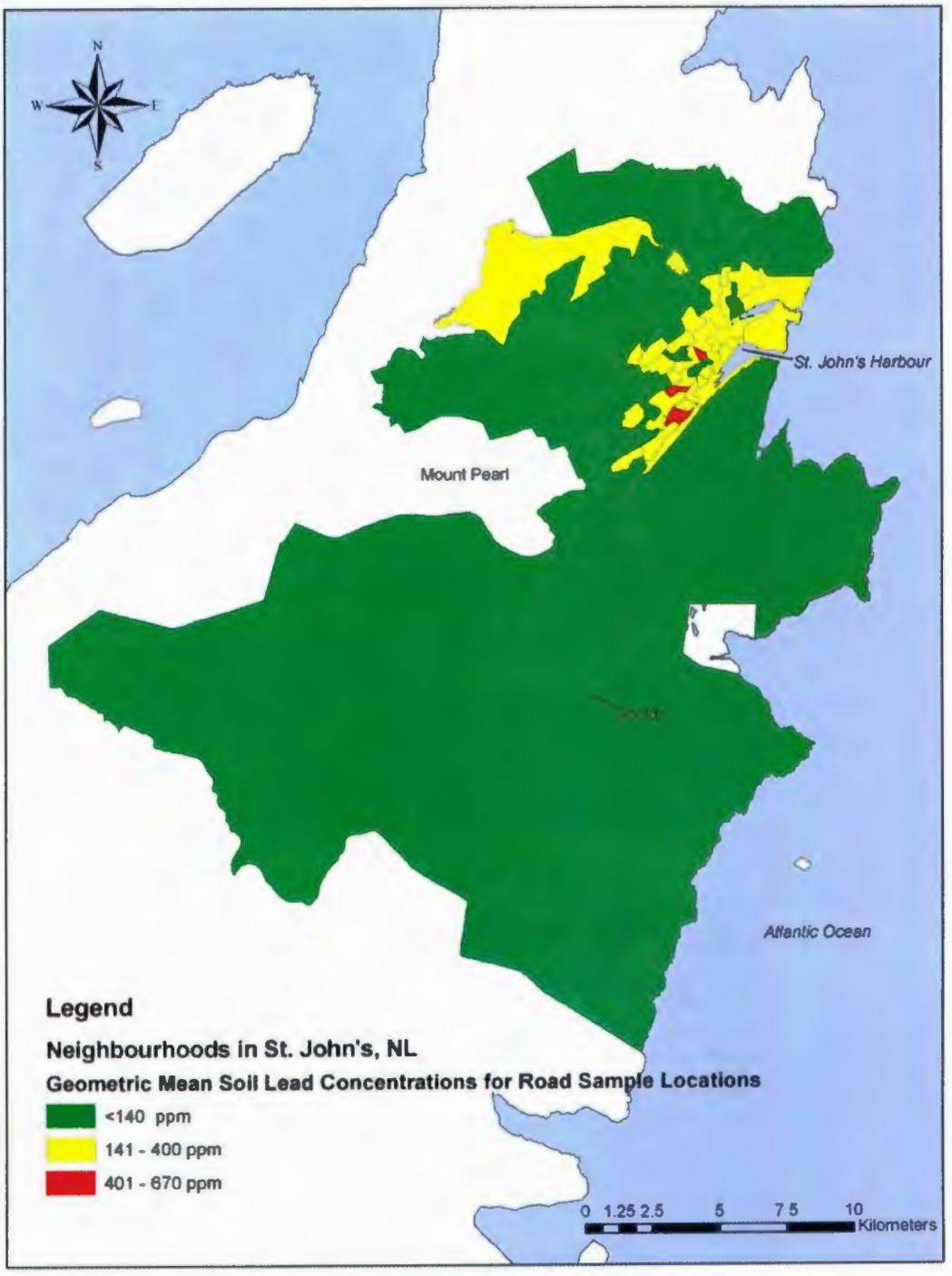


Figure 2.17. Neighbourhood soil lead concentrations based on the geometric mean of road samples.

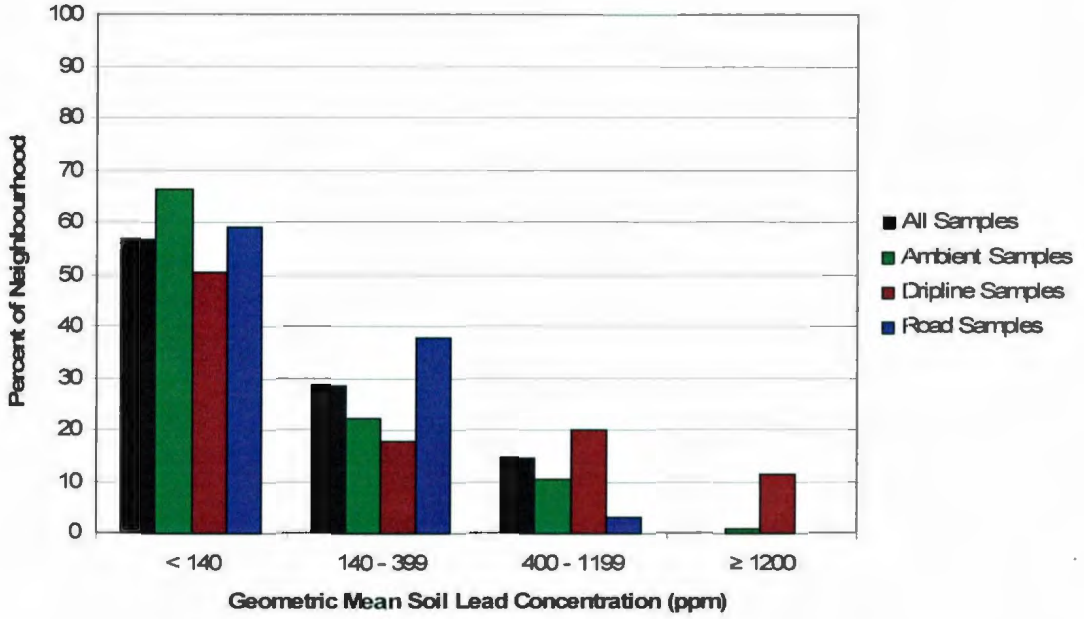


Figure 2.18. The percentage of St. John's neighbourhoods with geometric mean soil lead concentrations below and above the CCME residential soil lead guideline of 140 ppm, and the US EPA guidelines for bare soil in play areas (400 ppm), and bare soil not in play areas (1200 ppm).

Relationship to Property Age

In total 939 soil samples were collected on 311 properties whose age could be independently verified. Ambient, dripline, and road samples were collected on each property, with the exception of houses built prior to 1926 for which there were less opportunities to collect road samples because of paved frontages (Table 2.6). Box plots of the data illustrate the high number of outliers and the large variability (Figure 2.20).

The data show a decrease in geometric mean soil lead concentration for all three sample types as property age declines (Figure 2.19). Geometric mean soil lead concentrations

for roadside samples are consistently lower than the other sample types until the late 1970s when all sample types display relatively low mean concentrations. Ambient and dripline samples have relatively higher geometric mean lead concentrations in houses built prior to 1926 and 1960, respectively.

Almost all samples collected from properties built before 1926 exceed the CCME residential soil guideline of 140 ppm. The proportion of elevated soil samples remains high until the 1960s and by the late 1970s the percentage decreases to less than 10% (Figure 2.21). The percentage of samples exceeding the 400 ppm US EPA guideline is over 80% for ambient and dripline samples in houses built before 1926, and less than 10% in houses built after 1961. The only appreciable proportion of samples above the 1200 ppm US EPA soil guideline occurs on properties built before 1926 and consists of both dripline (~50%) and ambient (45%) samples.

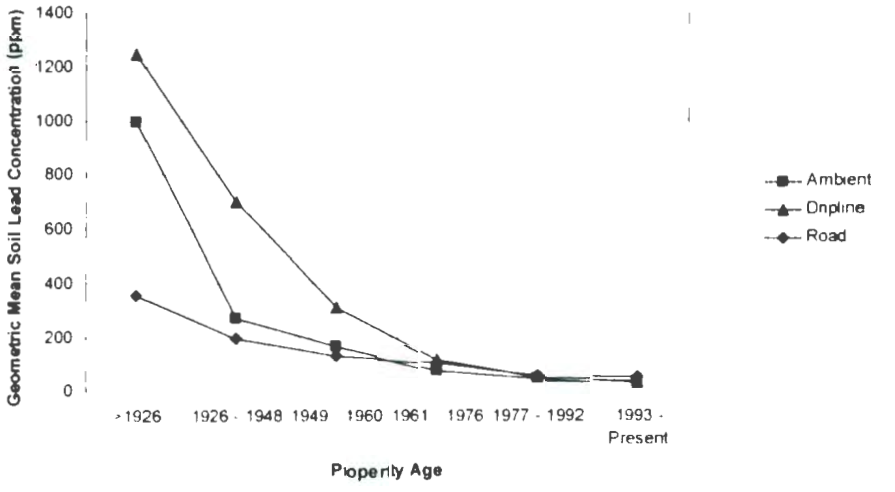
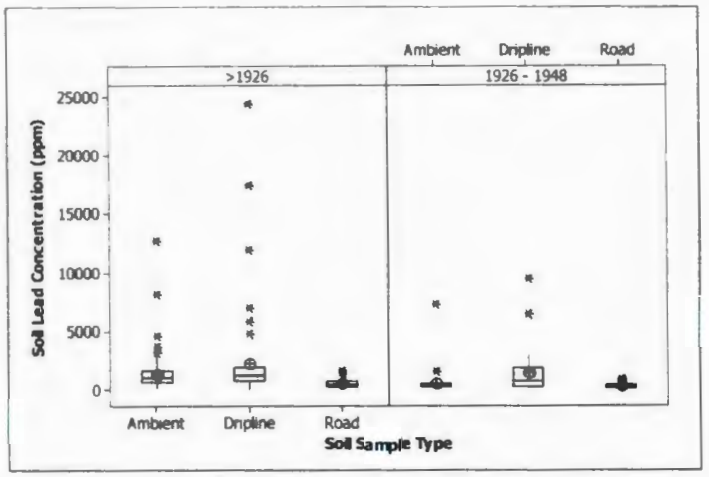
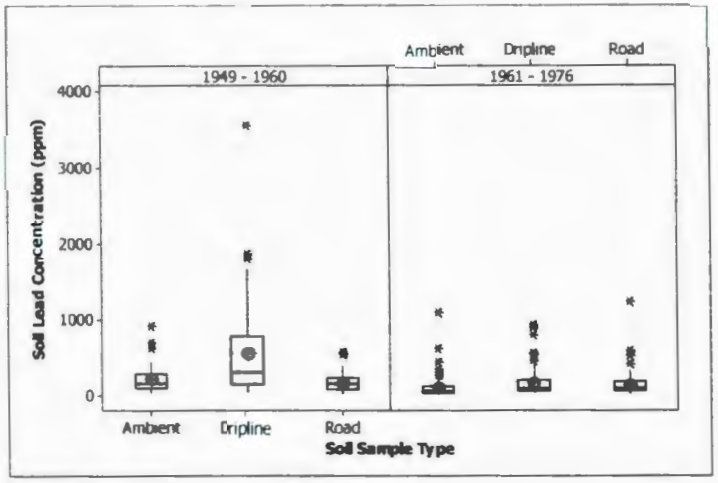


Figure 2.19. The relationship between geometric mean soil lead concentrations and property age for three different soil sample types.

Table 2.6. Descriptive statistics for soil lead concentrations organized by property age and sample location.

Property Age	Sample Type	n	Mean (ppm)	SE Mean (ppm)	Min (ppm)	25th (ppm)	50th (ppm)	75th (ppm)	Max (ppm)
Pre 1926	**All**	**178**	**1555**	**200**	**30**	**531**	**981**	**1609**	**24477**
	Ambient	85	1398	180	148	595	1077	1621	12738
	Dripline	62	2296	501	34	765	1253	1960	24477
	Road	31	500	70	30	213	376	708	1664
1926 - 1948	**All**	**103**	**750**	**138**	**21**	**159**	**335**	**617**	**9509**
	Ambient	36	550	201	22	162	318	503	7370
	Dripline	34	1413	331	67	223	742	1920	9509
	Road	33	284	42	21	128	208	371	989
1949 - 1960	**All**	**164**	**326**	**34**	**21**	**88**	**187**	**356**	**3567**
	Ambient	53	221	25	26	102	155	282	920
	Dripline	58	558	85	26	149	311	786	3567
	Road	53	178	19	21	74	154	237	587
1961 - 1976	**All**	**267**	**141**	**10**	**18**	**50**	**85**	**178**	**1245**
	Ambient	92	108	15	18	39	65	127	1092
	Dripline	89	173	19	26	60	98	212	940
	Road	86	145	17	19	56	95	187	1245
1977 - 1992	**All**	**154**	**63**	**6**	**13**	**32**	**45**	**64**	**768**
	Ambient	53	57	8	13	32	38	59	343
	Dripline	51	64	9	21	31	43	76	396
	Road	50	68	15	24	36	49	65	768
1993 - Present	**All**	**73**	**47**	**5**	**10**	**24**	**36**	**52**	**235**
	Ambient	24	44	6	10	20	37	55	137
	Dripline	24	34	3	15	23	30	42	69
	Road	25	64	11	16	31	41	77	235





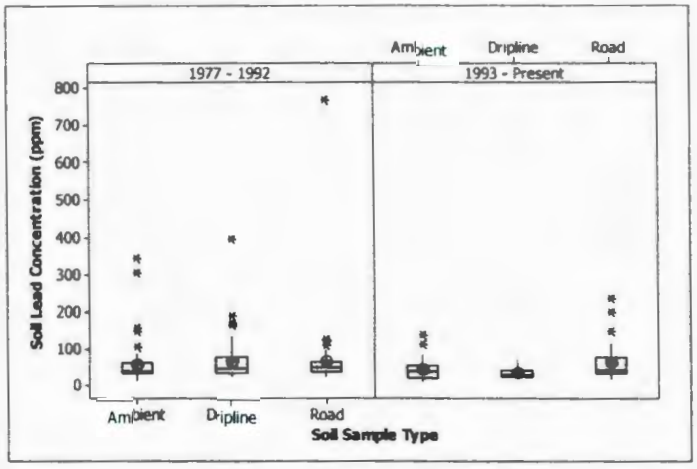
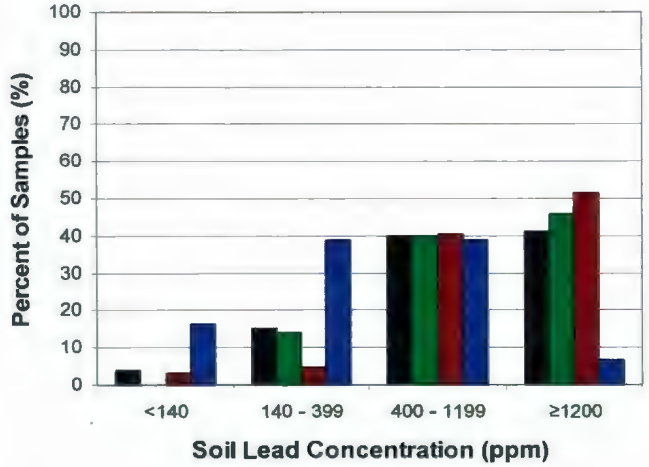
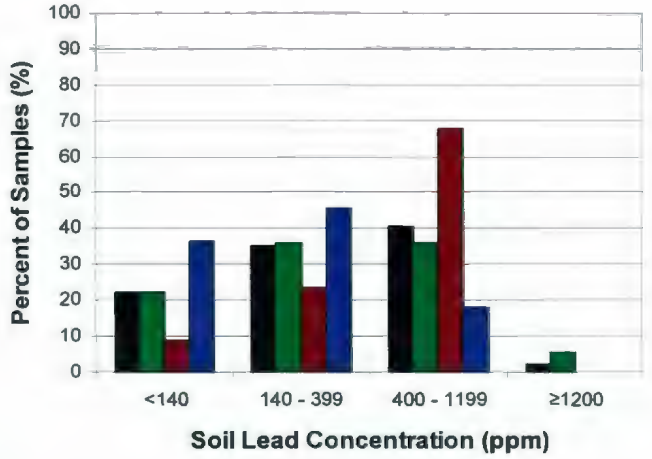


Figure 2.20. Box plots of the descriptive statistics for soil samples categorized by age of property and soil sample location.


Percent of Samples (%)
100
90
80
70
60
50
40
30
20
10
0
<140
140 - 399
400 - 1199
≥1200
Soil Lead Concentration (ppm)

a)


Percent of Samples (%)
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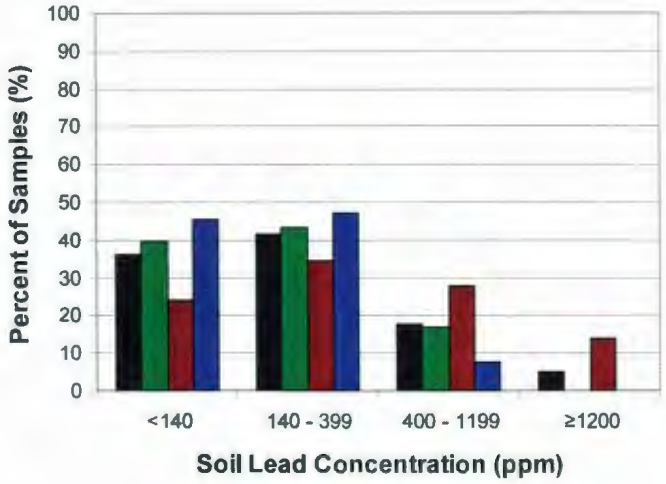
140 - 399
400 - 1199
≥1200
Soil Lead Concentration (ppm)

b)


Percent of Samples (%)
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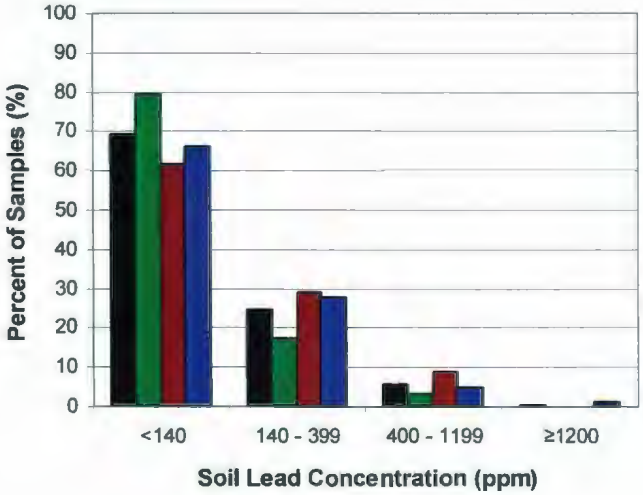
<140
140 - 399
400 - 1199
≥1200
Soil Lead Concentration (ppm)

c)


Percent of Samples (%)
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80
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50
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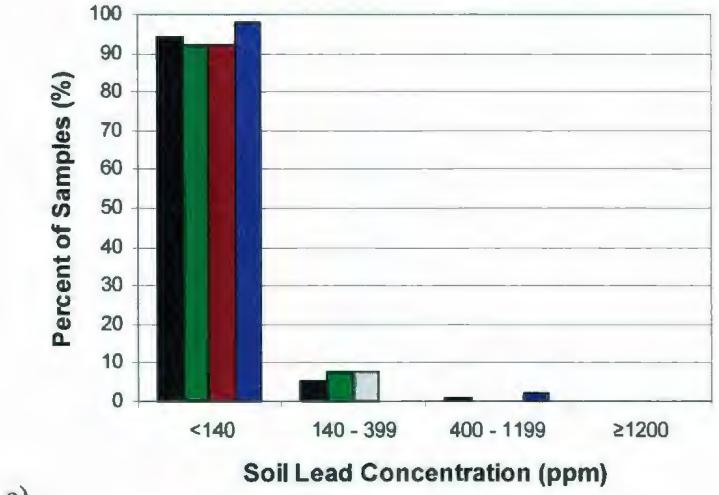
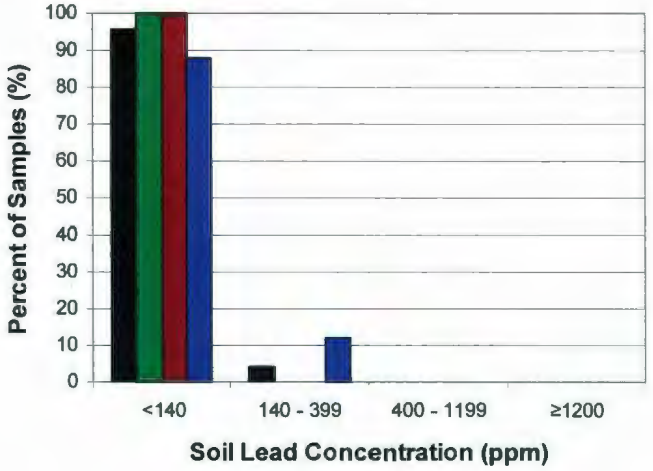
30
20
10
0
<140
140 - 399
400 - 1199
≥1200
Soil Lead Concentration (ppm)

d)



e)



f)

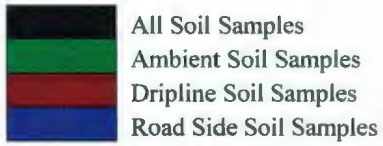


Figure 2.21. The percentage of soil samples with lead concentrations below and above the CCME residential soil lead guideline of 140 ppm, and the US EPA guidelines for bare soil in play areas (400 ppm), and bare soil not in play areas (1200 ppm) on properties developed a) before 1926, b) between 1926 and 1948, c) between 1949 and 1960, d) between 1961 and 1976, d) between 1977 and 1993, and f) after 1993.

Indoor Dust Lead

Descriptive Statistics

Ninety-six dust samples were collected from 32 homes in St. John's. Seventy-five percent of the samples have dust loading values below 23 $\mu g/ft^2$, with individual loadings as high as 3169 $\mu g/ft^2$ (Table 2.7). The geometric mean dust lead loading is 8 $\mu g/ft^2$ (95% CI: 3-10 $\mu g/ft^2$), but there is a broad range of values depending on the sample location within the home. Window sills have a geometric mean dust lead loading three times higher than entrance floors and six times higher than kitchen floors. Window sills, and to a lesser degree entrance floors, have several very high lead loadings, whereas kitchen floors are consistently low (Figure 2.22).

Table 2.7. Descriptive statistics for corrected indoor dust lead loadings.

Sample Type	n	Mean ($\mu g/ft^2$)	SE Mean ($\mu g/ft^2$)	GMean* ($\mu g/ft^2$)	Min ($\mu g/ft^2$)	25th** ($\mu g/ft^2$)	50th** ($\mu g/ft^2$)	75th** ($\mu g/ft^2$)	Max ($\mu g/ft^2$)
All	96	129.6	51.5	8.1	0.1	2.3	7.0	23.1	3169.4
Entrance	32	116.9	98.7	6.7	0.1	2.9	4.7	13.5	3169.4
Kitchen	32	10.1	3.2	3.3	0.2	0.9	2.7	9.4	79.5
Window	32	261.9	116.7	24.3	0.3	6.7	15.5	82.5	2938.2

* Geometric Mean
** Percentile

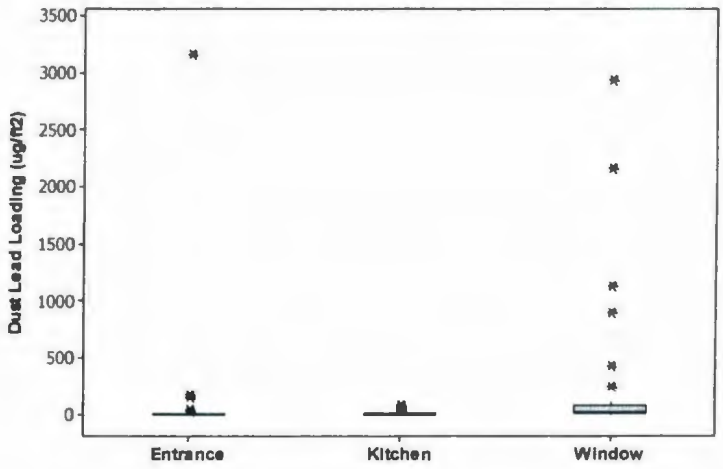


Figure 2.22. Box plots of the descriptive statistics for the three different dust lead loading sample locations in St. John's homes.

The US EPA has set standards for dust lead loadings on floors (40 $\mu g/ft^2$) and window sills (250 $\mu g/ft^2$) in residential homes. Eleven samples from seven different houses exceed the guidelines. In total, 12% of all samples, 16% of window sill samples, 13% of entrance floor samples, and 6% of kitchen floor samples exceed the guidelines (Figure 2.23).

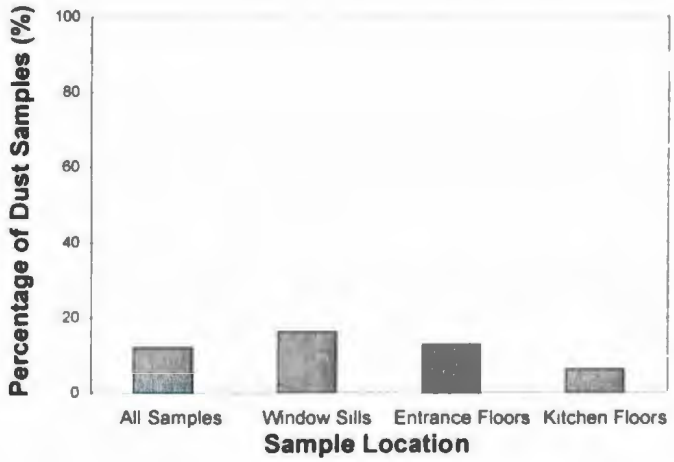


Figure 2.23. Percentage of dust lead samples exceeding US EPA guidelines for entrance floors, kitchen floors, and window sills.

Table 2.8. Site characteristics of the eleven dust samples exceeding US EPA dust lead loading guidelines.

Dust Lead Loading ($\mu g/ft^2$)	Class	Surface	Description	Days Since Last Cleaned	Age of Property	GMean Soil Lead (ppm)	Dripline Soil Lead (ppm)
50.3	e	wooden subfloor (tiles taken up for renovations)	very dirty, bits of plaster	60	1926-1948	404	927
57.8	k	wood	didn't look dirty until wiped	1	Pre 1926	2163	24477
79.5	k	concrete		2	Pre 1926	1175	1816
149.0	e	tile	pretty dirty and sandy (from last winter),	14	Pre 1926	1796	2656
177.3	e	heavier than linoleum (cushion flooring?)	pretty dirty moderately dirty (yarn, straw)		1926-1948	343	2553
433.2	w	painted wood	dirty, paint chipping	30	Pre 1926	1796	2656
902.1	w	painted wood	old and peeling white paint on window itself (not sill)	7	Pre 1926	1175	1816
1136.5	w	painted wood	very dirty, paint in moderate condition	14	Pre 1926	821	1655
2165.4	w	stained wood	very clean	7-14	Pre 1926	1152	1097
2938.2	w	painted wood	dusty, paint in fair condition	60+	Pre 1926	2163	24477
3169.4	e	tile		14	Pre 1926	2163	24477

Additional information on wiped surfaces reveal that those that appeared cleaner and were reportedly cleaned most recently prior to sampling produced lower dust lead loadings. For instance, kitchen floors were cleaned on average 3 days before sampling, whereas entrance floors were cleaned on average 12 days before, and window sills 34 days before. Nevertheless, for the 11 dust lead samples that the exceeded EPA guideline, the property age and soil lead concentration may be as important as the type and condition of the surface sampled and the elapsed time since it was last cleaned (see below and Table 2.8). For the most part the surfaces with elevated dust lead results were dirty or in poor condition, though in some cases they were recently cleaned.

Relationship with Soil Lead

In general for all samples there is a positive linear relationship between dust lead loading and dripline soil lead concentration (r^2=0.677, p=0.000) (Table 2.9, Figure 2.24). This relationship is strongest for entrance floor samples, less so for window sill samples, and least for kitchen floor samples. Kitchen floor samples correlate most strongly with the geometric mean for all samples taken on the property (Table 2.9).

Table 2.9. The Pearson correlation values for dust lead loadings at three different locations and corresponding soil lead concentrations.

Sample Location	Gmean Soil Lead		Ambient Soil Lead		Dripline Soil Lead		Road Soil Lead	
	r^2	p	r^2	p	r^2	p	r^2	p
All Samples	0.511	0.000	0.020	0.847	0.677	0.000	0.417	0.000
Entrance Floors	0.495	0.000	-0.010	0.939	0.955	0.000	0.511	0.000
Kitchen Floors	0.656	0.000	0.007	0.959	0.597	0.000	0.561	0.000
Window Sills	0.631	0.000	0.053	0.681	0.747	0.000	0.509	0.000

All eleven samples that exceeded the EPA guideline for dust lead loading were associated with dripline soil lead concentrations above 900 ppm; however, not all properties with high dripline soil lead concentrations produced high dust lead loadings.

Eight of the eleven dust samples are associated with geometric mean soil lead concentrations above 900 ppm, the other three are from properties with high dripline soil concentrations but very low ambient and/or road soil concentrations

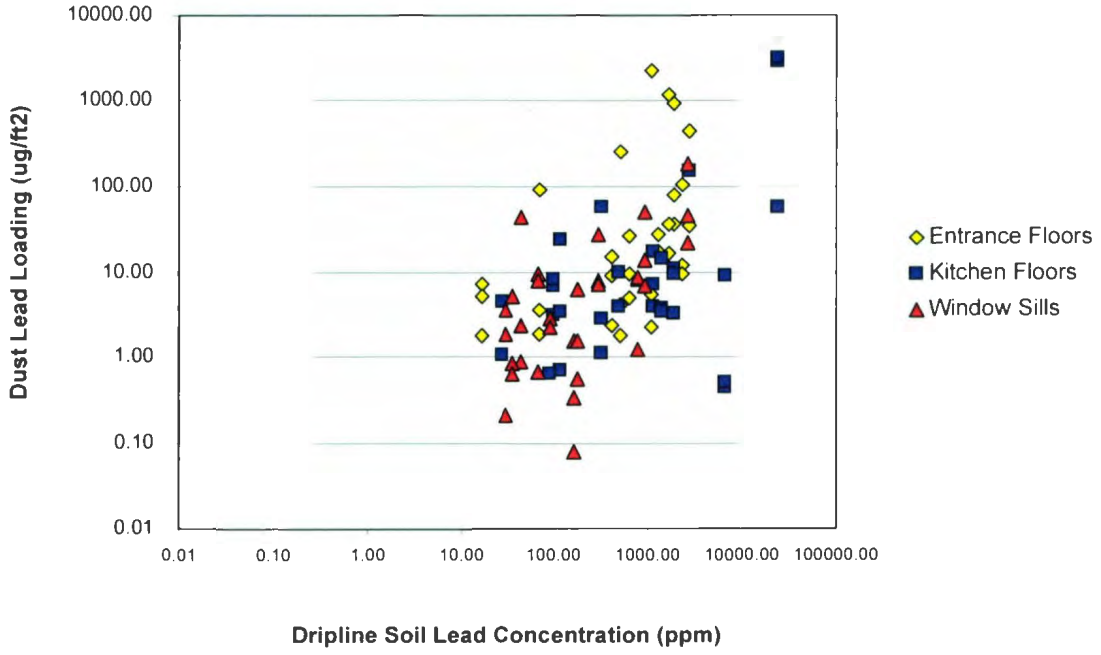


Figure 2.24. The correlation between dust lead loadings and corresponding dripline soil lead concentrations.

Relationship with Housing Age

Geometric mean dust lead loadings for all samples decrease with declining dwelling age until the late 1940s, after which there is little change with age (Table 2.10, Figure 2.25).

The highest dust lead loadings are recorded predominantly in houses built before 1926, especially for window sill loadings (Table 2.10). Even though window sill dust lead loadings decline dramatically in houses built after 1926, window sill loadings are always slightly higher than floors until the 1990s when all three sample types produce equally low loading values (Table 2.10, Figure 2.25). There are fewer outliers in the dust data compared to the soil data, but there is still a wide range of dust lead loading values (Figure 2.26). Of the 11 dust samples that exceed the US EPA dust lead loading guidelines, nine are from properties built before 1926 and two are from properties built between 1926 and 1948.

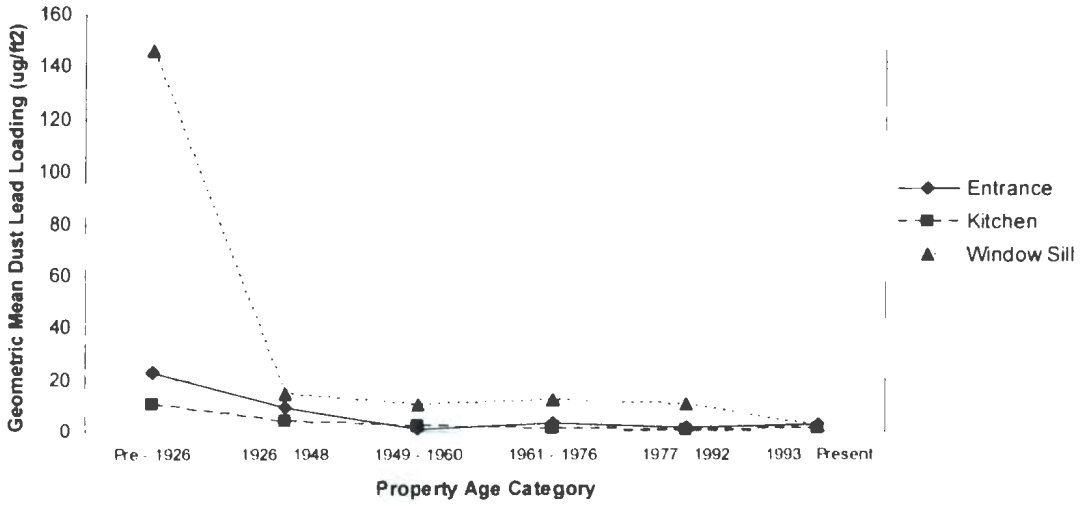
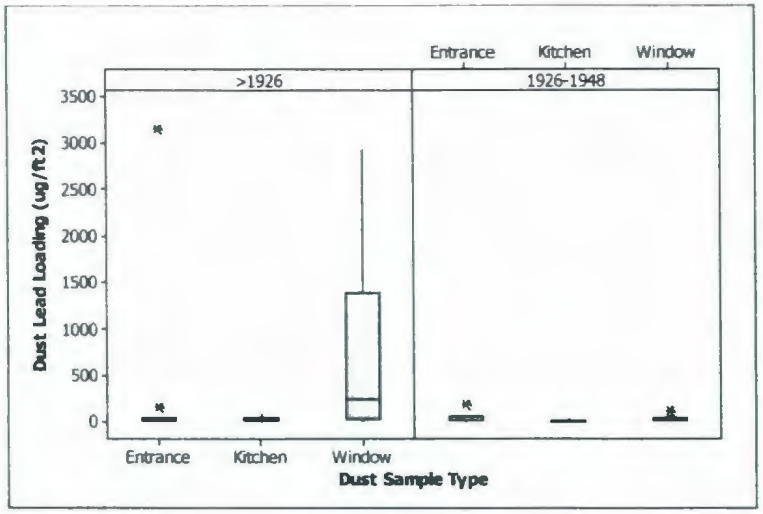
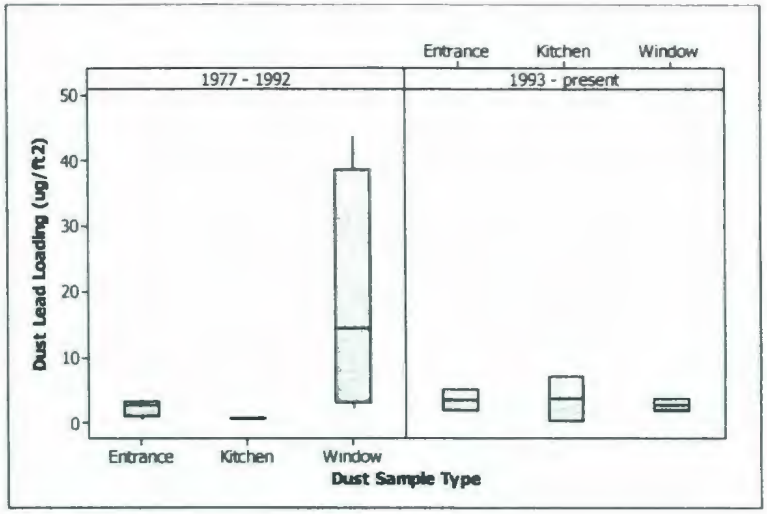


Figure 2.25. How geometric mean dust lead loadings for different sample locations vary with increasing property age.

Table 2.10. Descriptive statistics for dust lead loadings by sample location and age of dwelling.

Age Category	Sample Location	n	Mean (µg/ft2)	SE Mean (µg/ft2)	GMean (µg/ft2)	Min (µg/ft2)	25th (µg/ft^2)	50th (µg/ft^2)	75th (µg/ft^2)	Max (µg/ft^2)
Pre - 1926	Entrance	11	312.8	285.9	22.7	3.9	7.1	9.5	35.3	3169.4
	Kitchen	11	22.4	7.9	10.6	1.2	3.7	7.9	36.4	79.5
	Window Sill	10	767.0	328.9	146.1	8.0	17.2	229.9	1393.7	2938.2
1926 - 1948	Entrance	7	36.8	24.3	9.4	0.5	3.2	7.9	50.3	177.3
	Kitchen	7	7.5	2.8	3.8	0.4	0.7	6.8	10.5	21.5
	Window Sill	8	25.7	12.3	14.3	3.8	9.0	9.2	37.9	105.3
1949 - 1960	Entrance	3	2.5	1.2	1.0	0.1	0.1	3.4	4.0	4.0
	Kitchen	3	2.3	0.7	2.2	1.6	1.6	1.8	3.7	3.7
	Window Sill	3	87.0	79.8	10.6	0.3	0.3	14.3	246.4	246.4
1961 - 1976	Entrance	5	3.8	0.9	3.4	1.5	2.2	3.5	5.7	6.9
	Kitchen	5	1.5	0.4	1.3	0.6	0.8	1.1	2.4	3.1
	Window Sill	5	32.7	17.7	12.4	1.1	3.7	8.2	74.0	91.1
1977 - 1992	Entrance	4	2.3	0.6	1.9	0.6	1.1	2.6	3.2	3.4
	Kitchen	4	0.8	0.1	0.8	0.7	0.7	0.8	0.9	0.9
	Window Sill	4	18.7	9.6	10.5	2.3	3.0	14.4	38.8	43.8
1993 - Present	Entrance	2	3.4	1.6	3.0	1.8		3.4		5.0
	Kitchen	2	3.6	3.4	1.2	0.2		3.6		7.0
	Window Sill	2	2.7	0.9	2.5	1.8		2.7		3.6





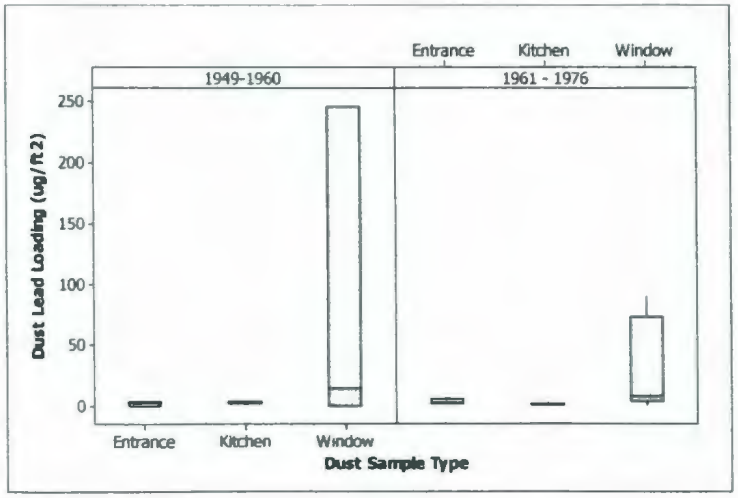


Figure 2.26. Box plots of the descriptive statistics for dust samples categorized by age of property and dust sample location.

Discussion and Conclusions

Soil

Background Soil Lead Concentrations

The Eastern Avalon has not been sampled for till geochemistry, instead the only background lead concentration data available are from lake sediment cores (Christopher *et al.*, 1993; Davenport *et al.*, 1992). While background lead concentrations in Quidi Vidi Lake were found to have a similar range as rural subsurface soil (13 ppm to 23 ppm) it is not possible to directly compare the two types of samples. Lake sediment is derived from soil, undisturbed till, and eroded bedrock from the entire catchment basin, and concentrations can be both diluted and enhanced through the processes of transportation and deposition on the lake bed (Bell, 2007).

The only data that can be compared to background soil lead samples is the geochemical analysis of till from similar geological groups in the Western Avalon on the Bay de Verde Peninsula (Batterson and Taylor, 2003). The study found that lead concentrations were noticeably higher in the St. John's Group, with many samples ranging from 1 ppm to 89 ppm, a few over 100 ppm, and three samples over 200 ppm. Lead concentrations for the till of the Signal Hill and Conception groups were lower; most samples were under 21 ppm, with only an occasional sample between 21 ppm and 89 ppm. Data for subsurface soil lead over the three geological groups sampled in St. John's fall within the

ranges recorded on the Bay de Verde Peninsula. The fact that high lead concentrations were found associated with the St. John's Group on the wider peninsula may help explain the higher concentrations found in background soil samples overlying the St. John's Group in urban settings.

It appears that in the absence of local anthropogenic sources of lead, subsurface and surface samples have similar concentrations, although surface soil values may be slightly elevated (on average by 20 ppm) due to long-range transport and deposition of air pollution, which has been noted in rural lake sediments in eastern and south-central Newfoundland (Christopher *et al.*, 1993; Davenport *et al.*, 1992). Proximity to historic roadways may also result in slightly higher surface lead concentrations in rural areas.

On the basis of strictly undisturbed subsurface samples in urban settings, the mean background soil lead concentration in St. John's is 37 ±18 ppm. Assuming a long-range pollution effect extrapolated from rural settings (+20 ppm), then urban surface soils should have a mean lead concentration value of 57 ppm, perhaps as high as 75 ppm. For surface samples overlying the St. John's Group, the occasional sample may exceed 100 to 200 ppm, which means that in rare circumstances natural lead concentrations in surface soil in St. John's may be above the CCME guideline of 140 ppm.

City-Wide Soil Lead Patterns

The geometric mean soil lead concentration for the complete St. John's dataset is 162 ±12 ppm (95% confidence interval) and the median value is 148 ppm, 55 ppm lower than the median reported in the preliminary study by Bell (2003). Bell's study collected more samples from the downtown core where soil lead values are much higher than the city in general. The maximum soil lead concentration in this study is also higher than the preliminary study (24 477 versus 7047 ppm), indicating that the local exposure risk is larger than expected on some individual properties.

Notwithstanding differences in sampling strategy, the mean and median soil lead concentrations for the city of St. John's are higher than other comparative Canadian cities without an industrial lead pollution source, such as Victoria, Sudbury, Ottawa, and Iqaluit (Table 2.11). St. John's soil lead concentrations are either similar to or lower than Canadian communities impacted by industrial sources of lead (Table 2.11).

Lead concentrations are highest around the harbour front and in the downtown core of St. John's and decrease with distance from the city centre. A similar distance-decay pattern has been found in New Orleans and other cities in Minnesota (Mielke, 1994; Mielke *et al.*, 1984/85) and has been explained by a number of factors. First, inner cities have a larger proportion of older houses with leaded paint and second, there is a history of higher traffic volume and more buildings which trap leaded gasoline emissions (Mielke, 1994). Soil surveys in England indicate that heavy metal associations in contaminated

soil match those in coal and they identify two distinct pathways along which coal contamination has entered the soil; first through the atmospheric deposition of aerosols from domestic and industrial coal combustion, and second by the spreading of coal ash on back gardens as a soil amendment or for waste disposal purposes (Rawlins *et al.*, 2002; Kelly and Thornton, 1996). These sources and pathways have also been proposed for St. John's (Bell, 2003).

Table 2.11. A comparison of soil lead concentrations for other Canadian cities.

City	Population	Metric	Lead (ppm)	Sample Type
St. John's, NL	95 000	Geometric Mean	162	Residential soil collected from open spaces, along foundations, and by roadsides.
Belledune, NB [1]	1 711	Median	43-136	Range of medians for garden soil in five sub-areas
Sydney, NS [2]	24 115	Geometric Mean	297	Residential soil collected away from buildings and roads near the coke ovens
Victoria, BC [3]	75 000	Median	90	Boulevards, parks, school yards
Trail, BC [4]	7 575	Geometric Mean	756	Residential soil collected from two to three areas of exposed soil where children play
Port Colborne, ON [5]	18 600	Median	167	Residential topsoil collected at least one metre away from driveways, walkways, buildings, and fences
Sudbury, ON [6]	157 857	Mean	30	Various locations downwind from three Ni and Cu smelters
Ottawa, ON [7]	774 000	Geometric Mean	42	Residential soil collected from five locations in yard
Iqualuit, NU [8]	4 220	Range	26-217	Commercial and residential sites sampled at grid intersections and also targeted samples from playgrounds, roads, and culverts

[1] Government of New Brunswick, 2005; [2] Lambert and Lane, 2004; [3] Bowman and Bobrowsky, 2003; [4] Hilts, 2003; [5] Ontario Minstry of the Environment, 2002; [6] Adamo *et al.*, 2002; [7] Rasmussen *et al.*, 2001; [8] Peramaki and Decker, 2000

Despite the general city-wide trends there are, however, some high soil lead concentrations in outlying areas and low concentrations in the urban core. High concentrations in peripheral areas of the city tend to be associated with older properties or near older roads around which the city has grown. It is plausible that sources associated with high soil lead in the inner city, such as leaded paint and coal and gasoline combustion emissions, also apply to these sites. Additionally, high concentrations in ambient samples outside the downtown may be due to sample proximity to old structures (sheds or garages) that have been removed from the properties. Low soil lead concentrations in the downtown core may result from the use of imported or amended soil and sod for landscaping and renovation purposes. Imported fill is also used in the construction of road medians and side walks.

Neighbourhood Soil Lead Patterns

One of the disadvantages of aggregating soil values for large neighbourhood areas is that the average value is not necessarily representative of the whole neighbourhood. This is particularly obvious for peripheral neighbourhoods of St. John's. Because these areas are sparsely populated the neighbourhood size is quite large in order to encapsulate a population of 1000. These neighbourhoods therefore would have a large amount of undeveloped land with presumably much lower soil lead concentrations than the sampled properties. Notwithstanding this issue of representation in peripheral neighbourhoods, the neighbourhood soil lead patterns are broadly similar to the city-wide patterns based on point data only. Once again the development history of St. John's has an influence on

soil lead levels with neighbourhoods along the harbour front and old road ways having higher concentrations. In conclusion, neighbourhood geometric means may not be as effective as age of housing stock in characterizing the spatial pattern of soil lead in St. John's and therefore would not be appropriate for a human health risk assessment.

Property Level Soil Lead Patterns

Differences in soil lead concentrations at a local scale depend on where samples were taken on the property. In this study, geometric mean soil lead values were highest for dripline locations (219 ppm) compared to ambient and roadside sampling sites (154 ppm and 136 ppm, respectively), reflecting the influence of past and/or present leaded paint deterioration on exterior wooden clapboard. Road samples have lower concentrations than might be expected which may reflect the relatively small size of St. John's, the corresponding low traffic density, and the relatively new road network in the city, postdating the removal of lead from gasoline. Not surprisingly then roadside locations in suburban residential areas have concentrations similar to nearby ambient samples.

Mielke (1994) used a similar sampling strategy for soil lead characterization of inner city, mid city, and suburban locations in New Orleans. Despite the significant difference in population size, soil lead concentrations for ambient, dripline, and roadside samples in St. John's for the most part exceeded those for New Orleans (Table 2.12). Ambient and dripline samples for St. John's consistently exceeded those for New Orleans, by as much as 500% in the case of ambient values for the inner city, but the differences in concentrations between the two cities generally decreased from the inner city to the

suburbs. The marked difference in ambient soil lead values in the inner city may reflect the close proximity of buildings in downtown St. John's and the compounding factor of multiple lead sources in small gardens and open areas. Dripline and ambient samples have higher median soil lead concentrations in mid city areas of St. John's, possibly due to a higher lead paint contribution from painted clapboard houses in these areas in St. John's, or perhaps a younger housing stock in the mid city of New Orleans compared to St. John's.

Table 2.12. A comparison of soil lead concentrations in three different locations on properties in New Orleans and St. John's.

Location in City	Sample Location	New Orleans Median (ppm)	St. John's Median (ppm)
Inner City (pre 1926)	All Samples		981
	Ambient	212	1077
	Dripline	840	1253
	Road	342	376
Mid City (1926-1960)	All Samples		224
	Ambient	40	201
	Dripline	110	412
	Road	110	168
Suburban (post 1960)	All Samples		58
	Ambient	28	49
	Dripline	50	62
	Road	86	64

The Effect of Property Age on Soil Lead Concentrations

Soil lead concentrations are high for all samples taken on properties developed before 1926 and dripline concentrations remain high on properties dating from the late 1940s. The age of these properties correspond to the period when lead concentrations were highest in paint, up to 50% by weight (CMHC, 2007). It also corresponds with a period of widespread airborne pollution and ash disposal from coal combustion. Geometric mean dripline and ambient soil lead concentrations are below the CCME guideline of 140 ppm on properties developed after 1961, mirroring the decline in the amount of lead used in paints. This year is mentioned specifically by the Canadian Mortgage and Housing Corporation (2007) as the building date after which homeowners should be less concerned about leaded paint in homes. The use of coal for residential heating also declined in St. John's during the 1950s which may have also contributed to the low soil lead concentrations.

Geometric mean roadside lead levels are only slightly above CCME guidelines on properties built before 1948 which may simply reflect the smaller vehicular traffic volume in St. John's in the first half of the century, or the dilution or replacement of lead contaminated roadside soils in the last 50 to 60 years during infrastructure improvements.

Indoor Dust

Sampling Location in Home

Geometric mean window sill dust lead loadings are much higher than loadings on entrance and kitchen floors in homes in St. John's (24.3, 6.7, and 3.3 $\mu g/ft^2$ respectively). This is most likely due to the presence of leaded paint on exterior and interior windows. Window sill lead loadings were also elevated compared to floor samples in a study in Rochester, New York (geometric means of 393 - 476 $\mu g/ft^2$ compared to 8 $\mu g/ft^2$; Lanphear *et al.*, 1999). Window sill loadings in Rochester are much higher than those reported here, which probably reflects a sampling concentration on urban homes in the Rochester study compared to both urban and suburban homes in St. John's. Entrance floors have only slightly higher loading values than kitchen floors in St. John's, which contrasts with results from Sydney, Nova Scotia, where doorway loadings were found to be an order of magnitude higher than kitchen floors (Lambert and Lane, 2004). Entrance floor loadings are expected to be higher than those on kitchen floors because of tracking in of contaminated soil on shoes or pets and the reported more frequent cleaning of kitchen surfaces.

Relationship between Soil Lead and Indoor Dust Lead

In general, dust lead loadings in sampled houses in St. John's are more strongly correlated with outdoor soil lead concentrations from dripline sample sites. This relationship is strongest for entrance floors and window sills, whereas kitchen floors have a stronger association with the geometric mean soil lead concentration for the property as

a whole. Houses where elevated dust lead values were recorded all had dripline soil lead concentrations above 900 ppm. Entranceway floors and window sills are expected to have a stronger correlation with dripline soil samples because of their immediate proximity and exposure to these sample sites. Lambert and Lane (2004) found that soil was a major source of lead in entrance ways through the tracking in of soil on footwear. However, investigation of the entire dust dataset shows that many sites with dripline soil lead concentrations higher than 900 ppm did not have elevated dust lead loadings. Other factors including the movement of soil in the home, the cleaning regime, or the presence or absence of leaded paint from within the home must also be important.

Housing Age and Indoor Dust Lead

In St. John's overall geometric mean dust lead loadings decline with decreasing housing age prior to 1948, at which point they level off. This date was also used to distinguish between homes with high and low dust lead levels in Ottawa (Rasmussen *et al.*, 2001). Around this time restrictions on lead concentration in paint began to be implemented while interior paint became lead free in the late 1970s. Surprisingly, there does not appear to be a dramatic drop in dust lead loadings for houses built after the 1970s; perhaps renovations and remodelling blurs age-specific trends for indoor dust lead in older homes.

Dust lead loadings are much higher in sampled houses built before 1926 and are always associated with dripline soil lead concentrations above 900 ppm. These relationships

suggest that extremely high soil lead concentrations in dripline locations, which are primarily a product of deteriorating outdoor leaded paint on clapboard, are an important contributing factor to elevated indoor dust lead loadings. Alternatively, it can be argued that elevated indoor dust lead loadings are strongly influenced by window sill dust lead values, which are primarily a product of deteriorating indoor leaded paint on the window and to a lesser degree outdoor paint dust blown in through open windows. Rasmussen *et al.* (2001) argued that soil lead concentrations have little to do with indoor dust lead concentrations in Ottawa. Their study found very low soil lead concentrations associated with high dust lead concentrations, particularly in homes built before 1950.

Health Risk

Soil Lead Concentrations and Soil Lead Guidelines

There is reason to be concerned about soil lead levels in St. John's as 51% of the samples collected and 43% of the neighbourhoods sampled have geometric mean lead concentrations above the CCME residential soil lead guideline of 140 ppm. There is reason to be concerned about soil lead levels in St. John's as 51% of the samples collected and 43% of the neighbourhoods sampled have geometric mean lead concentrations above the CCME residential soil lead guideline of 140 ppm. Most of these houses are in the older, more central districts.In order for children to be exposed to lead they need to be living in those older homes. As a rough indicator of risk Figures 2.30 and 2.31 show the percentage of housing built before 1946 and 1960 for each neighbourhood.

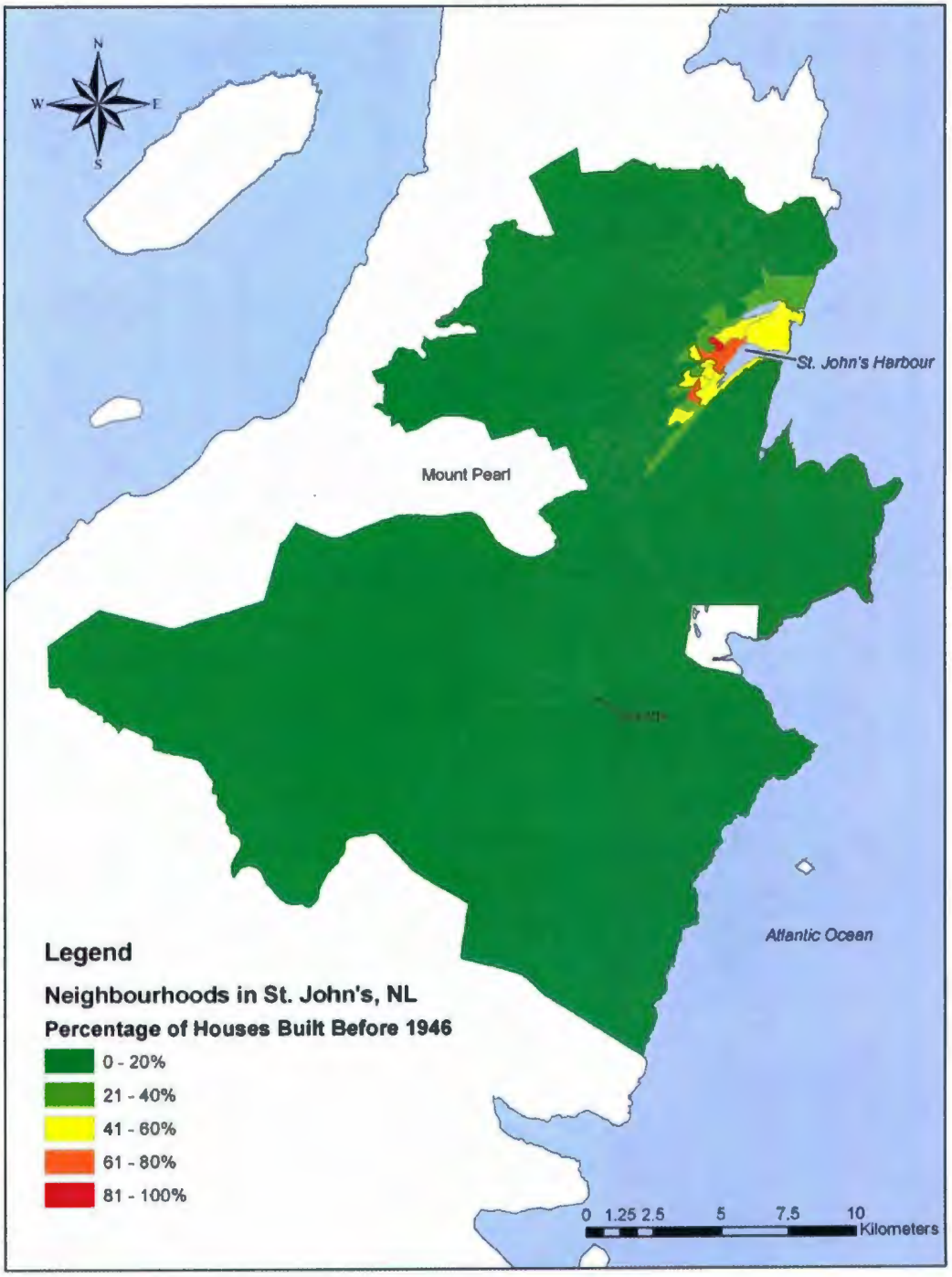


Figure 2.27. The percentage of neighbourhood houses built before 1946 (Community Accounts, 2007).

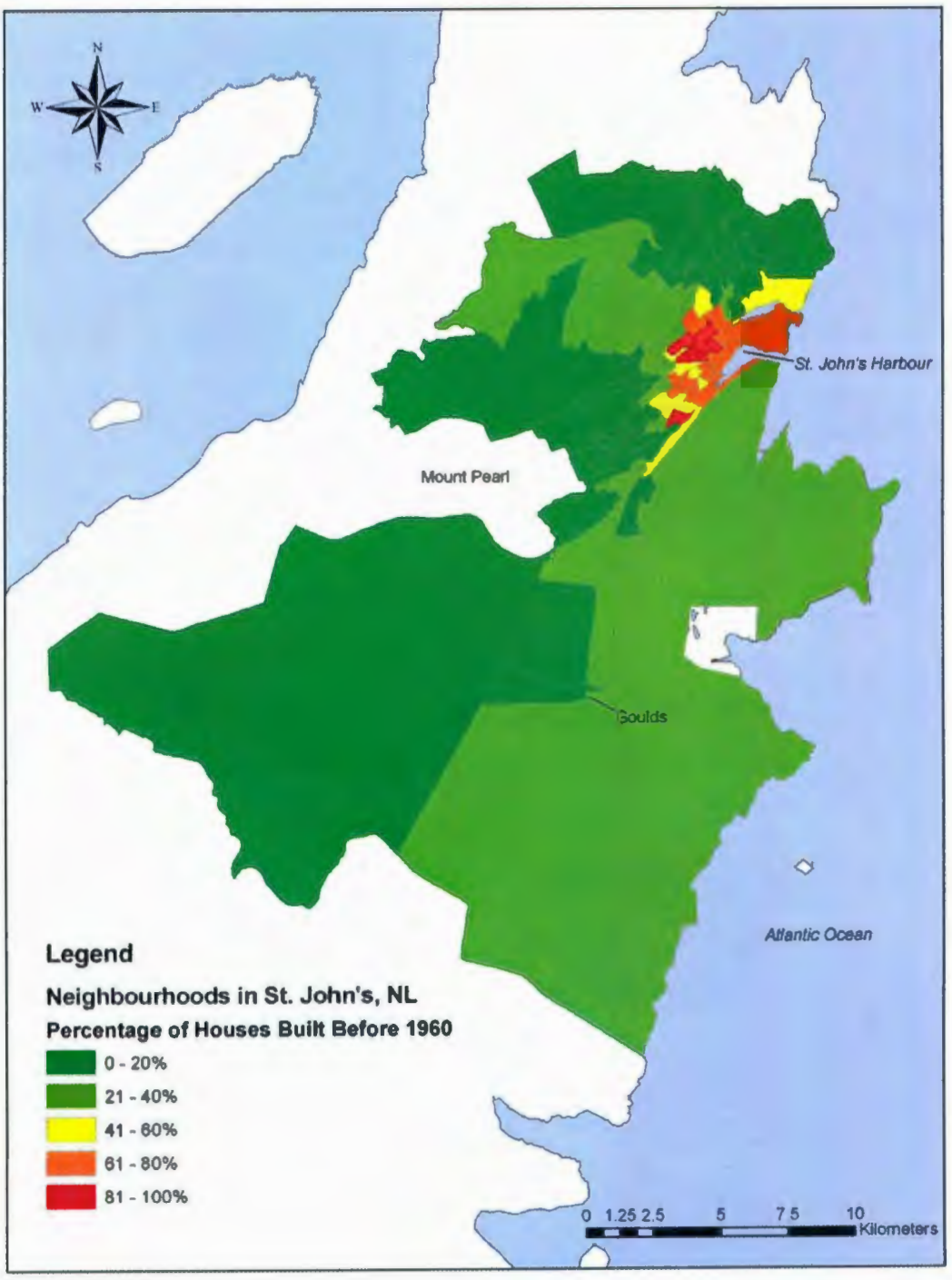


Figure 2.28. The percentage of neighbourhood houses built before 1960 (Community Accounts, 2007).

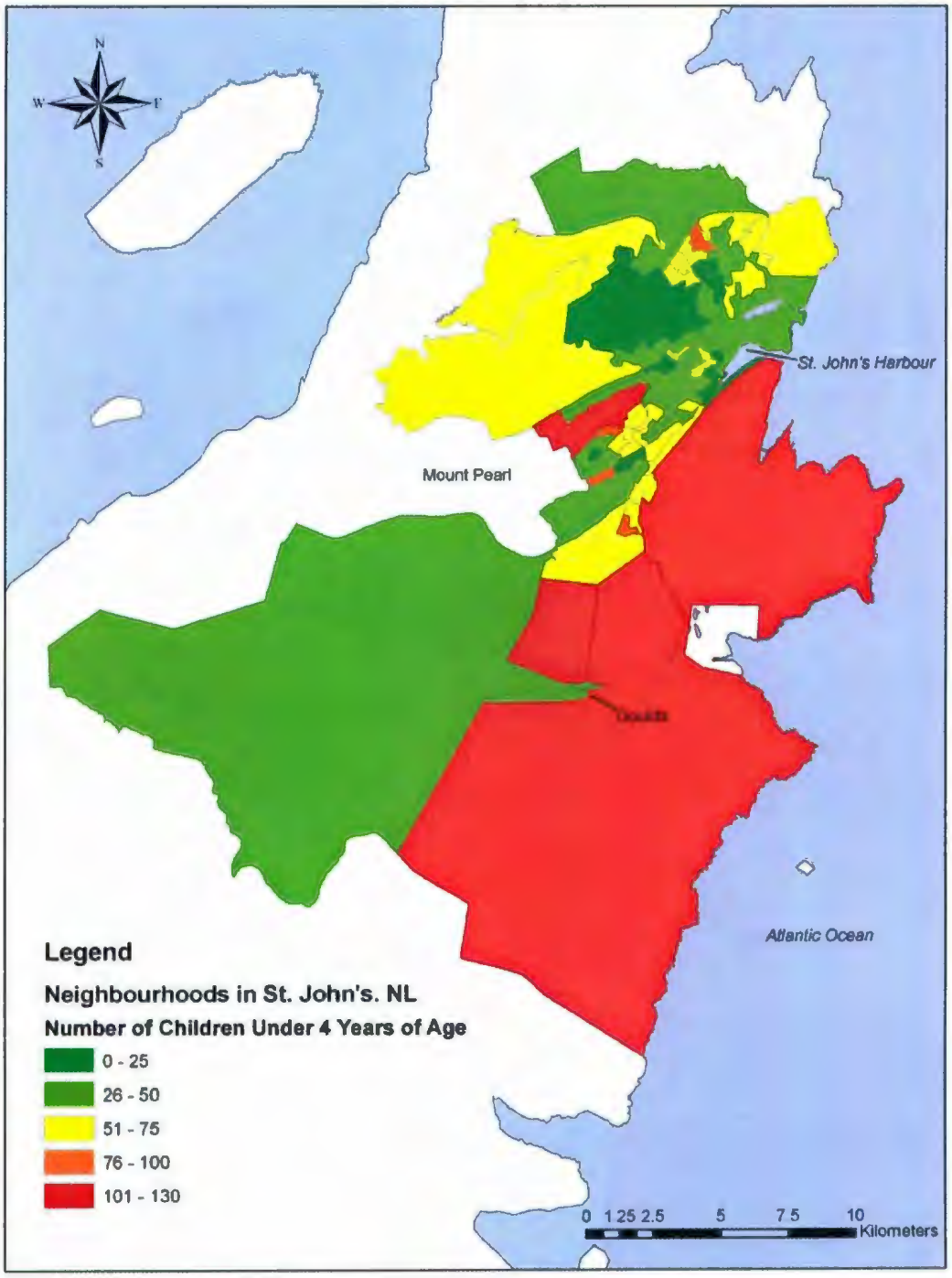


Figure 2.29. The number of children in each neighbourhood under 4 years of age (Community Accounts, 2007).

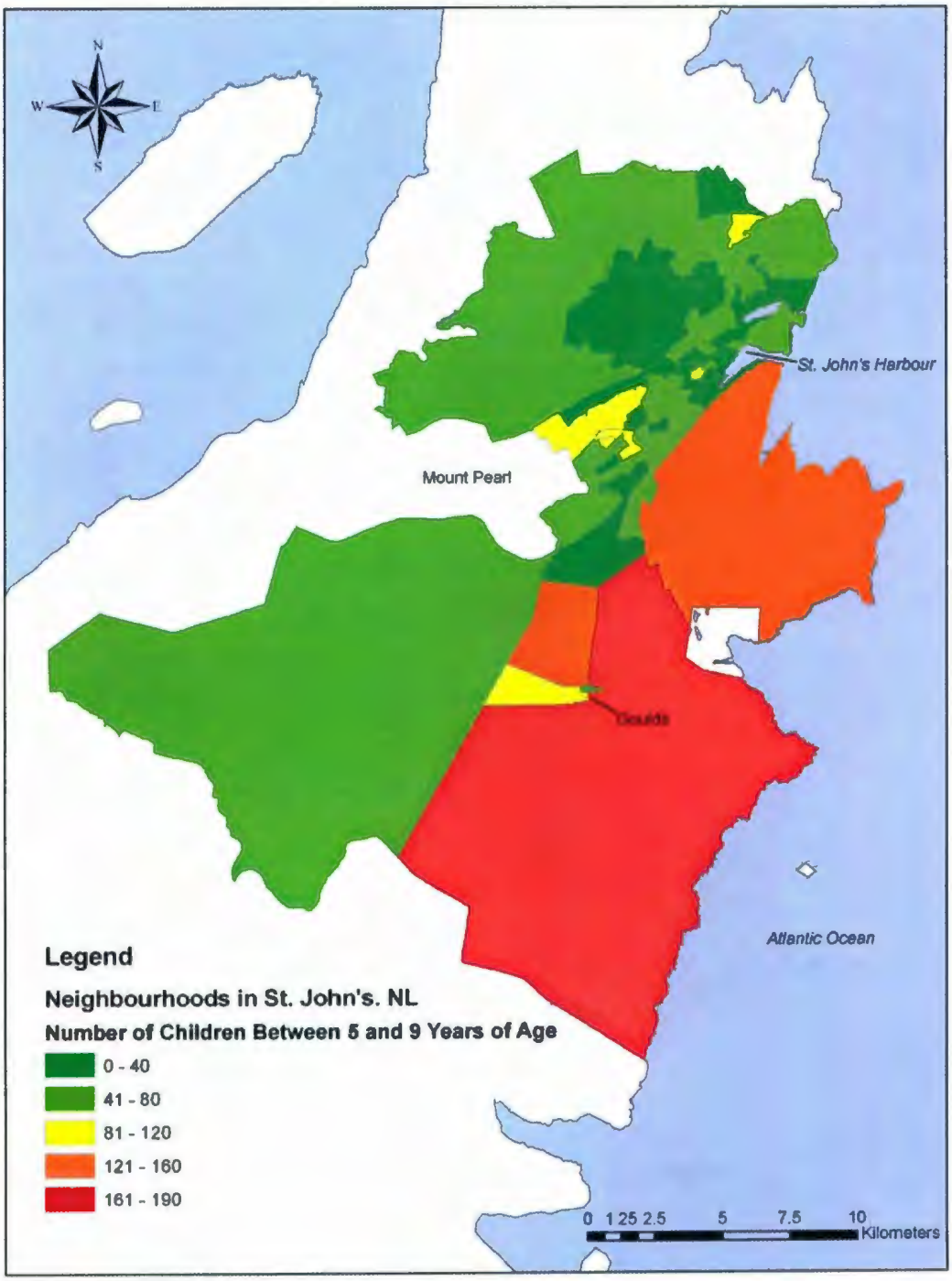


Figure 2.30. The number of children in each neighbourhood between five and nine years of age (Community Accounts, 2007).

Dust Lead Loadings and US EPA Guidelines

The US EPA has set maximum acceptable dust lead loadings at 40 $\mu g/ft^2$ for interior floors and 250 $\mu g/ft^2$ for interior window sills. Twelve percent of dust samples taken in the St. John's pilot dust study exceed these guidelines, most are from window sills and entrance ways. Of the seven houses where these 11 elevated dust samples were taken, all were built before 1948 and had dripline soil lead concentrations greater than 900 ppm; however, not all houses that share these characteristics have elevated dust lead loadings. Other factors, such as the type and condition of sampled surfaces and the elapsed time since the last cleaning, do not exhibit a consistent pattern at the elevated sites. On the basis of other studies in Ottawa and elsewhere, it seems highly likely that the presence of deteriorating leaded paint, either indoors, outdoors, or both, may be contributing significantly to indoor dust lead loadings in these pre-1948 aged houses. Further study is needed to confirm these sources and pathways for indoor dust lead in St. John's.

Estimated Blood Lead Levels

On the basis of a pooled analysis of 12 epidemiologic studies, Lanphear *et al.* (1998) used a multivariate regression model to generate a simple predictive relationship for blood lead concentrations from a suite of variables, including interior floor dust lead loading, exterior soil or dust lead concentrations, maximum interior paint lead content, household water lead concentration, indication of damaged paint, and race, age, socioeconomic status, and mouthing behaviour of residents. Using geometric mean

values for soil lead concentration and indoor dust lead loading from this study, together with standard values for the remaining variables, preliminary estimates of mean blood lead concentrations in children between 6 and 36 months are generated for St. John's (Table 2.13). Predicted geometric mean blood lead concentrations range between 6.5 and <2.6 μg/dL, while the probability of concentrations exceeding the clinical threshold for lead poisoning of 10 μg/dL are 21 and ≤2.2% for pre-1926 and post 1950s housing, respectively. These preliminary estimates suggest that while the wider St. John's community has a potentially low risk for blood lead poisoning from lead contaminated soil and indoor dust, there is a potential exposure risk for toddlers living in pre-1950s housing. Results from a preliminary blood lead study in St. John's and a retrospective chart review in the province indicate that children's geometric mean blood lead concentrations are under 3 μg/dL, with only 4% predicted to have concentrations above 10 μg/dL (Allison, 2006; O'Brien, 2006). This may be because the number of toddlers living in pre-1950s housing is potentially relatively small, only 9% of children under four based on rough calculations of the number of children living in neighbourhoods with greater than 50% of the housing built before 1946. Fortunately, most pre-1950s housing is spatially restricted to downtown and surrounding neighbourhoods, which could easily be targeted for an educational program that mitigates soil and dust exposure risk.

Table 2.13. Predicted blood lead concentrations for children between 6 and 36 months in St. John's based on data from Lanphear *et al.* (1998).

Property Age	Measured GMean Soil Lead Concentration (ppm)	Measured GMean Floor Dust Lead Loading (µg/ft^2)	Predicted GMean Blood Lead (µg/dL)	Predicted Probability >10 µg/dL (%)
Pre 1926	901	15.5	6.5	21.3
1926 - 1948	334	6.1	4.6	7.7
1949 - 1960	191	1.0	3.0	1.6
1961 - 1976	95	2.1	3.2	2.2
1977 - 1992	49	0.2	< 2.6	< 0.8
1993 - Present	38	1.9	2.8	1.3

Conclusions

The historical use of leaded paint and combustion of coal and leaded gasoline has left a geochemical mark on many cities around the world. In downtown St. John's these lead sources have been exacerbated by the high density of wooden clapboard houses and the frequency of devastating fires. The result is that 51% of surface soil samples taken across the city exceed the CCME guideline of 140 ppm. In houses built before 1926, which represents most of the downtown core, 98% of soil samples exceed this level, 66% by an order of magnitude. Considering the considerable number of studies that have linked areas of high soil lead with elevated blood lead levels, this is a concern for residents of St. John's.

While a large proportion of soil samples have high soil lead, only 12% of houses sampled in a pilot dust lead study had dust lead loadings above US EPA standards. Of the houses

that exceeded the guidelines all were built before 1948 and had dripline soil lead concentrations over 900 ppm. Contaminated indoor dust may be less of a concern for the majority of residents; however, for those living in older homes with extremely elevated soil lead, indoor dust lead may be an additional source of exposure.

Environmental lead levels have been found to be elevated on properties developed before 1948. These levels could result in geometric mean blood lead levels of 4.6 to 6.5 μg/dl. in children under three years of age, and 8 to 21% of children living with these levels of lead may exceed have blood lead levels above 10 μg/dL. In practice, there is no evidence of elevated blood lead levels in the children of St. John's based on a chart review of children admitted to the Janeway Children's Hospital. However, the chart review is based on a small number of children from all over the province and not a comprehensive screening of high risk areas. It remains possible that a portion of the children in this community are at risk and further investigation should be considered.

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Chapter 3: Environmental Lead Exposure in St. John's, Newfoundland: Preliminary Human Health Risk Assessment

Introduction

Exposure to environmental lead can have both acute and chronic health effects. For most children acute exposure to lead is no longer a problem as the removal of lead from gasoline, domestic paint, and solder in food cans and water pipes has reduced blood lead levels (Meyer, 2001). Instead it is the health effects brought about by continual exposure to low levels of lead that are of concern. Research has shown that chronic exposure to lead causes neuro-developmental effects including decreased scores on IQ and other intelligence and development tests (Schwartz, 1994). Children are at greater risk because they absorb 30-40% more lead through the intestine compared to adults (OMEE, 1994; Bellinger, 2004). Additionally, children tend to have more direct contact with sources of lead through hand-to-mouth behaviour (Viverette *et al.*, 1996; Bellinger, 2004; Moya *et al.*, 2004).

A blood lead concentration of 10 µg/dL (0.48 µmol/L) is defined in the United States as the level at which intervention may be required. In recent years lead has been shown to be associated with neuro-developmental concerns at lower levels (Federal Provincial Committee on Environmental and Occupational Health Lead Working Group, 1994; Canfield et al 2003; Bellinger and Needleman 2003). In response, the United States has

established large scale screening programs, particularly in large urban areas where the persistence of environmental lead is recognised (Mielke *et al.*, 1984/85; Bellinger *et al.*, 1985; Ernhart *et al.*, 1985; Dietrich *et al.*, 1987; Mielke, 1994; Lanphear *et al.*, 1998; Mielke *et al.*, 1999; Johnson and Bretsch, 2002), but there are no such programs in Canada. Instead community level interventions are to be undertaken when blood lead concentrations from a sample of children exceed the mean from the general population plus three standard deviations, or when the percentage of children with values above 10 µg/dL is double that seen in the general population (Federal Provincial Committee on Environmental and Occupational Health Lead Working Group, 1994). This may explain why most studies in Canada have focused on residential areas around industrial sources of lead (Langlois *et al.*, 1996; OME, 2002; Hilts, 2003; Lambert and Lane, 2004; Government of New Brunswick, 2006), with only occasional studies on urban areas without point sources of pollution (Peramaki and Decker, 2000; Rasmussen *et al.*, 2001; Bowman and Bobrowsky, 2003).

There is conflicting evidence regarding the connection between elevated blood lead concentrations and high lead levels in environmental media. Many studies have shown a relationship with soil and dust, both of which can act as a sink for historic inputs of lead into the environment (Lanphear *et al.*, 1998; Mielke *et al.*, 1997). Other studies have found no such correlation, including the Lead Screening Report recently released for the Eastside Community in Port Colborne, Ontario (Decou *et al.*, 2001), and several soil lead abatement projects (Langlois *et al.*, 1996; Weitzman *et al.*, 1993; Farrell *et al.*, 1998).

In contrast, a recent assessment of high lead levels in residential soil and indoor dust in St. John's, Newfoundland, suggests that while the wider community has a potentially low risk for blood lead poisoning, there is a potential exposure risk for toddlers living in pre-1950s housing (Campbell 2008). Fifty-one percent of surface soil samples (n=1231) exceeded the Canadian Council of Ministers of the Environment (CCME) guidelines for residential soil (140 ppm), and 12% exceeded the US EPA guidelines for indoor dust (40 $\mu g/ft^2$ for floors and 250 $\mu g/ft^2$ for window sills). This paper explores the potential human health risk associated with these environmental lead levels as a precursor to any blood screening program, especially since recent Canadian studies indicate that geometric mean blood lead concentrations in children are below the guideline of 10 µg/dL (Langlois *et al.*, 1996; Decou *et al.*, 2001; Hilts *et al.*, 2001; Nova Scotia Department of Health and the Cape Breton District Health Authority, 2001).

As soil and indoor dust lead are the focus of this risk assessment ingestion, inhalation, and dermal contact with these media are predicted to be the major contributors to total lead intake. Additionally, the consumption of home-grown produce from contaminated soil is another potential pathway for those individuals with gardens. Other lead exposure routes, including drinking water, consumer food products, and urban air, are less of a concern since the reduction or elimination of lead in water pipes, solder and gasoline; nevertheless, they are incorporated in the risk assessment for completeness.

The risk assessment for St. John's was conducted in two stages. First, a screening-level risk assessment was conducted using the federal government's *Preliminary Quantitative Risk Assessment* (PQRA) procedures (Health Canada, 2004). The PQRA standardizes screening-level risk assessment methodology for federal contaminated sites to facilitate comparisons between sites and to help establish priority areas for remediation (Health Canada, 2004). Initially a worst-case scenario was investigated using 95th percentile soil and dust lead concentrations along with other high estimates of exposure (Appendix C). If no health risks were found in the worst-case scenario then no further exploration would be needed; however since unacceptable health risks were found an average-case risk assessment was conducted using 50th percentile soil and dust lead concentrations along with more moderate exposure parameters. The PQRA for St. John's addresses the following questions: (i) What are the estimated daily intakes (EDIs) for the various receptor life stages; infant, toddler, child, teenager, and adults? (ii) What is the cumulative daily intake (CDI) of lead averaged over the lifetime of the most exposed receptor and the moderately exposed receptor? (iii) How do the CDIs and EDIs compare to the oral lead tolerable daily intake (TDI) of 0.0036 mg/kg/day (Health Canada, 2004)? (iv) What sources of lead contribute the most to the total daily lead intake? (v) How does the daily intake vary with exposure to different levels of soil lead contamination as a function of property age? (vi) Based on housing stock age, which neighbourhoods are potentially at risk from environmental lead in St. John's?

Second, the US EPA *Integrated Exposure, Uptake and Biokinetic (IEUBK) Model for Lead in Children* was used to estimate exposure to lead from birth to seven years and generate a distribution of blood lead values (US EPA, 2002; 2005). The model is based on three different components: exposure to different media (air, water, diet, soil, dust, other ingested media, and maternal blood lead concentration); uptake of the lead based on differing bioavailability in the lungs and gastrointestinal tract, and a biokinetic component that models the storage, transportation and excretion of lead within the body. Output from the IEUBK model run for St. John's data was used to answer the following two questions: (i) What are the predicted mean blood lead concentrations for children with average exposure to environmental lead? (ii) What are the probabilities that children with an average exposure to environmental lead will have blood lead concentrations greater than 10 μg/dL?

Early exploratory runs of the PQRA and IEUBK models indicated that the consumption of garden produce was a significant contributor to total daily lead intake and blood lead concentrations. Because not all residents of St. John's eat fruits and vegetables from a backyard garden the first scenario was run without garden produce as an additional source of lead intake. For those residents who do eat garden produce two additional scenarios were evaluated, each with a different plant uptake factor. This was done because there were conflicting recommendations regarding this parameter in the literature and the impact of changing the uptake factor greatly affected the final lead concentration estimated for the plant. One set of uptake factors from the Multimedia, Multipathway,

and Multireceptor Risk Assessment (3MRA) Modeling System (US EPA, 2003a) was recommended to Health Canada by a consulting firm (Health Canada, 2005a), and another set was taken from Boyd *et al.* (1999) because the pattern reflected plant uptake in other studies (Finster *et al.*, 2004).

Methods

PQRA Assessment

Risk was assessed for five standard PQRA receptor categories: infants (0 to 6 months), toddlers (7 months to 4 years), children (5 to 11 years), adolescents (12 to 19 years), and adults (20+ years), living in six property age categories: pre 1926, 1926-1948, 1940-1960, 1961-1976, 1977-1992, and 1993 to the present. The property age categories were derived from an analysis of soil lead concentration and property age in St. John's by Campbell (2007a).

The following sections briefly describe the calculations, assumptions and values used in the average risk scenario run of the PQRA model for St. John's. More detailed explanations and justifications of model parameters and values for both the average risk and conservative risk scenarios are available in Appendix B.

Receptor Characteristics

Standard PQRA data for receptor characteristics such as body weight, surface area and exposure rates were used in the risk assessments (Table 3.1). Receptors were understood to be exposed 24 hours a day, 364 days per year, as per PQRA residential exposure assumptions. Newfoundland has the highest prevalence of obesity in Canada (Twells, 2005) and therefore the use of standard Canadian receptor body weights may result in an overestimation of the daily lead intake for all pathways.

Table 3.1. Receptor characteristics used in the risk assessment calculations.

Receptor Characteristic	Units	Infant	Toddler	Child	Teen	Adult
Age[1]	yrs	0 -6 m	7 m - 4 y	5 - 11 y	12-19 y	> 20 y
Body Weight[2]	kg	8.2	16.5	32.9	59.7	70.7
Soil Ingestion Rate[3,4]	g d	0.02	0.08	0.02	0.02	0.02
Inhalation Rate[2,5]	m^3 d	2.1	9.3	14.5	15.8	15.8
Water Ingestion Rate[2]	L d	0.3	0.6	0.8	1	1.5
Skin Surface Area[2]	cm^2					
Hands		320	430	590	800	890
Forearms		248	401	666	1004	1125
Lower Legs		364	676	1228	1988	2288
Feet		250	430	720	1080	1190
Entire Body		1780	3010	5140	8000	9110
Food Ingestion[2]	g d					
Root vegetables		83	105	161	227	188
Other vegetables		72	67	98	120	137
Fruits and Juices		136	234	268	258	245

[1] Health Canada (1994)
[2] Richardson (1997)
[3] CCME (1996)
[4] MDEP (2002)
[5] Allan and Richardson (1998)

Ingestion of Outdoor Soil and Indoor Dust

The total daily intake of lead from the ingestion of soil was calculated using Equation 1 (Health Canada, 2006) and data from Table 3.2.

$$ING_{Soil\ \ Dust}(mg \cdot kg \cdot day) = \frac{C_{Soil\ \ Dust} \times IR_{Soil\ \ Dust} \times F_{Soil\ \ Dust} \times RAF_{GI} \times D_1 \times D_2}{BW} \qquad (1)$$

Table 3.2. Model parameters used for the ingestion of soil and dust exposure pathways in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Soil Lead Concentration	C_{Soil}	mg/kg	Geometric mean soil concentration derived from all soil samples (ambient, dripline, roadside)	Health Canada, 2004
Dust Lead Concentration	C_{Dust}	mg/kg	Two times the geometric mean soil lead concentration	Healey, 2007
Soil Ingestion Rate	IR_{Soil}	kg/day	45% of PQRA soil ingestion rates	Walker and Griffin, 1998; OME, 2002; US EPA, 2005
Dust Ingestion Rate	IR_{Dust}	kg/day	55% of PQRA soil ingestion rates	Walker and Griffin, 1998; OME, 2002; US EPA, 2005
Relative Gastrointestinal Absorption for Lead in Soil and Dust	RAF_{GI}	unitless	0.8	Health Canada, 2006
Exposure Duration	D_1	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004
	D_2	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004
Body Weight	BW	kg	Standard PQRA values (Table 3.1)	Richardson, 1997

Outdoor Soil and Indoor Dust Inhalation

Outdoor dust generated from lead contaminated soil may potentially be inhaled by receptors. Indoor dust may also be re-suspended into the air and subsequently inhaled. PQRA assumes that the inhalation of contaminated soil and dust is minimal compared to the ingestion and dermal contact pathways. In this study the inhalation of contaminated soil and dust is considered as an additional pathway of exposure, at least in the initial run stages, to assess its impact on the overall lead intake. It was calculated using Equation 2 and data from Table 3.3 (Health Canada, 2004):

$$INH_{Soil\ \ Dust}(mg/kg \cdot day) = \frac{C_{Soil\ \ Dust} \times P_{Air} \times IR_{Air} \times RAF_{Inh} \times D_1 \times D_2 \times D_3}{BW} \qquad (2)$$

Dermal Contact with Soil and Indoor Dust

Direct contact of contaminated soil or dust with skin can cause a very small transfer of lead into the blood stream. The PQRA and US EPA risk assessment protocols assume one dermal contact event per day; however, PQRA does not account for exposure to contaminated indoor dust. The risk assessment conducted in Port Colborne (OME, 2002) incorporated dust exposure by weighting the one daily dermal event by the proportional time spent outdoors and indoors. This approach was adopted for the St. John's risk assessment using Equation 3 and data from Table 3.4 (Health Canada, 2004).

$$DERM_{Soil\ \ Dust}(mg\ \ kg \cdot day) = \frac{C_{Soil\ \ Dust} \times A_{Soil\ \ Dst} \times F_{Out\ \ In} \times RAF_{Derm} \times EF \times D_1 \times D_2}{BW} \qquad (3)$$

Table 3.3. Model parameters used for the inhalation of soil and dust exposure pathways in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Soil Lead Concentration	C_{Soil}	mg/kg	Geometric mean soil concentration derived from all soil samples (ambient, dripline, roadside)	Health Canada, 2004
Dust Lead Concentration	C_{Dust}	mg/kg	Two times the geometric mean soil lead concentration	Healey, 2007
Particulate Concentration in air	P_{Air}	µg/m3	0.76	US EPA, 1992a
Receptor Air Intake Rate	IR_{Air}	m^3/hour	Standard PQRA intake rates (Table 3.1)	Health Canada, 2004
Relative Inhalation Absorption for Lead in Soil and Dust	RAF_{Inh}	unitless	1	Health Canada, 2004
Soil Exposure Duration	D_1	hours/day	1.5	Modification of Richardson, 1997
	D_2	days/7days	7 days per week exposed/ 7 days	Health Canada 2004
	D_3	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada 2004
Dust Exposure Duration	D_1	hours/day	22.5	Modification of Richardson, 1997
	D_2	days/7days	7 days per week exposed/ 7 days	Health Canada 2004
	D_3	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada 2004
Body Weight	BW	kg	Standard PQRA values (Table 3.1)	Richardson, 1997

Ingestion of Home Grown Produce

A questionnaire given to homeowners participating in the indoor dust survey of St. John's included questions on gardening practices. Fifteen of the 32 houses had a garden, in which herbs, strawberries, raspberries and tomatoes were commonly grown; fewer households grew root vegetables. Participants indicated that they consumed garden produce typically for one to three months of the year and during these months they ate on average four meals per week that contained garden produce.

Table 3.4. Model parameters used for the dermal contact soil and dust exposure pathways in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Soil Lead Concentration	C_{Soil}	mg/kg	Geometric mean soil concentration derived from all soil samples (ambient, dripline, roadside)	Health Canada, 2004
Dust Lead Concentration	C_{Dust}	mg/kg	Two times the geometric mean soil lead concentration	Healey 2007
Annual Average Amount of Soil or Dust Dermal Contact	$A_{Soil\ Dust}$	kg	Surface areas for each body part were multiplied by the corresponding soil or dust loading factor, weighted by the number of months exposed (four for the warm months and eight for the cool months) and then divided by 12	Modification of OME, 2002
Exposed Skin Surface Area		cm2	Standard surface areas for hands, forearms, lower legs, and feet assumed to be exposed from June to September. Only hands assumed to be exposed for the rest of the year.	Health Canada, 2004; and a modification of OME, 2002
Soil Loading Factor		mg/cm2	Geometric mean soil loadings for children in dry soil were used for infants, toddlers, and children for all body parts except feet for which no loading factor was available. Instead the soil loading for feet for daycare children with both outdoor and indoor exposures was used. Soil loading factors for gardeners were applied to teenagers and adults for all body parts.	OME, 2002;US EPA, 2004
Dust Loading Factor		mg/cm2	Geometric mean dust loading factors for children indoors were used for all age groups because of the lack of comparable data for teenagers and adults	OME, 2002;US EPA, 2004
Fraction of Time Spent Outdoors	F_{Out}	unitless	1.5 hours/ 24 hours (0.0625)	
Fraction of Time Spent Indoors	F_{In}	unitless	22.5 hours/ 24 hours (0.9375)	
Relative Lead Dermal Absorption Factor	RAF_{Derm}	unitless	0.006	Health Canada, 2004
Exposure Frequency	EF	events/day	1	
Dermal Exposure	D_1	days 7days	7 days per week exposed/ 7 days	Health Canada, 2004
	D_2	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004
Body Weight	BW	kg	Standard PQRA values (Table X)	Richardson, 1997

Richardson (1997) calculated the average total daily vegetable and fruit intake by Canadians (Table 3.1). In this study the daily intake was divided into three to give the average amount of fruit and vegetables consumed per meal. It was assumed that garden produce contributed to all the fruits and vegetables eaten for four meals per week during a three-month growing season.

The concentration of lead in garden produce was estimated from soil lead concentrations. Only uptake from roots was considered because lead does not occur in gaseous form, which eliminates direct uptake from the air, and because the deposition of contaminated particulate matter in St. John's is unknown. Two sets of bioconcentration factors (BCFs) were used to investigate how influential the choice of factor is on lead intake. A review for Health Canada indicated that the best models for estimating heavy metal concentrations in backyard produce are the *Multimedia, Multipathway, and Multireceptor Risk Assessment (3MRA) Modeling System* (US EPA, 2003a) and the *Guidance for Developing Ecological Soil Screening Levels* (US EPA, 2003b; Health Canada 2005a). One set of BCFs for root vegetables, other vegetables, and fruit was taken from the 3MRA model (US EPA, 2003a). The other set was from Boyd *et al.* (1999) in which the BCFs were highest in the root of plants and decreased with distance from the soil, a pattern supported by the results of other studies (Finster *et al.*, 2004), but not the 3MRA model. BCFs from Boyd *et al.* (1999) that matched those fruits and vegetables grown in St. John's gardens were averaged for each of the three produce categories.

The amount of lead in home grown produce ingested by receptors was calculated using Equations 4 and 5 and parameter values from Table 3.5.

$$ING_{Produce} = ING_{Root} + ING_{Other} + ING_{Fruit} \quad (4)$$

where:

$ING_{Produce}$ = daily intake of ingested lead from home grown produce (mg/kg*day)

ING_{Root} = daily intake of ingested lead from home grown root vegetables (mg/kg*day)

ING_{Other} = daily intake of ingested lead from home grown other vegetables (mg/kg*day)

ING_{Fruit} = daily intake of ingested lead from home grown fruit (mg/kg*day)

$$ING_{Root\ Other\ Fruit} = \frac{CSoil \times BCF_{Root\ Other\ Fruit} \times IR_{Root\ Other\ Fruit} \times AF_{GI} \times D_1 \times D_2}{BW} \quad (5)$$

Ingestion of Store-Bought Food

The Canadian Total Diet Study Report provides an overall total daily dietary lead intake based on the collection and preparation of supermarket food from eight Canadian cities (Health Canada, 2005b). Dietary lead intake values for sixteen age groups from the Health Canada report were combined according to the five PQRA age categories using age-weighted averages for both the conservative and average risk scenarios (Table 3.6).

3.5. Model parameters used for the ingestion of garden produce exposure pathway in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Soil Lead Concentration	C_{Soil}	mg kg	Geometric mean soil concentration derived from all soil samples (ambient, dripline, roadside)	Health Canada, 2004
Plant Lead Bioconcentration Factor I	$BCF_{Root\ Other\ Fruit}$	unitless	0.03 for root vegetables, 0.038 for other vegetables, and 0.15 for fruit (dry plant weight dry soil weight)	US EPA, 2003
Plant Lead Bioconcentration Factor II	$BCF_{Root\ Other\ Fruit}$	unitless	0.033 for root vegetables, 0.010 for other vegetables, and 0.00004 for fruit (wet plant weight/dry soil weight)	Boyd *et al.*, 1999
Garden Produce Consumption Rates	$IR_{Root\ Other\ Fruit}$	kg day	Assumed that one entire meal of garden produce was consumed (one third of daily root, other vegetable, or fruit intake as reported by Richardson, 1997) for four meals a week over three months of the year. This amount was averaged over 365 to provide a daily intake.	Modification of Richardson, 1997 using data from St. John's questionnaire
Relative Gastrointestinal Absorption for Lead in Food	RAF_{GI}	unitless	1	Health Canada, 2004
Exposure Duration	D_1	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004
	D_2	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004
Body Weight	BW	kg	Standard PQRA values (Table X)	Richardson, 1997

Table 3.6. Total dietary lead intake for specific age groups of Canadians modified from the Canadian Total Diet Study Report (Health Canada, 2005b).

Age (years)	Dietary Lead Intake (mg/kg*day)
0 - < 0.5	0.000479
0.5 - < 5	0.000487
5 - < 12	0.000368
12 - < 20	0.000224
20 <	0.000187

Ingestion of Drinking Water

The ingestion of lead in drinking water was calculated using Equation 6 and data from Table 3.7 (Health Canada, 2004):

$$ING_{Water}(mg/kg \cdot day) = \frac{C_{Water} \times IR_{Water} \times RAF_{GI} \times D_1 \times D_2}{BW} \quad (6)$$

Table 3.7. Model parameters used for the ingestion of drinking water exposure pathway in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Drinking Water Lead Concentration	C_{Water}	mg/L	0.0048, median from 40 houses sampled in Ontario	Graham, 1988; Health Canada, 1992
Water Ingestion Rate	IR_{Water}	L/day	Standard PQRA values	Richardson, 1997
Relative Gastrointestinal Absorption for Lead in Drinking Water	RAF_{GI}		1	Health Canada, 2004a; US EPA, 2005
Exposure Duration	D_1	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004
	D_2	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004
Body Weight	BW	kg	Standard PQRA values (Table X)	Richardson, 1997

Dermal Contact with Water during Bathing

Equation 7 and data from Table 3.8 were used to estimate the daily intake of lead that passes through the skin while bathing (US EPA, 2004; US EPA, 1997):

$$DERM_{Water}(mg/kg \cdot day) = \frac{C_{Water} \times CF \times PC \times SA_{Total} \times EF \times D_1 \times D_2}{BW} \quad (7)$$

Table 3.8. Model parameters used for the dermal contact with bathing water exposure pathway in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Drinking Water Lead Concentration	C_{Water}	mg L	0.0048	Graham, 1988; Health Canada, 1992
Volumetric Conversion of Water	CF	L/cm^3	0.001	
Dermal Permeability Coefficient for Lead	PC	cm/hour	0.0001	US EPA, 2004
Surface Area	SA_{Total}	cm2	Total body surface area (Table X)	Richardson, 1997
Length of Bathing Time		hours/day	0.33 (50^{th} percentile)	US EPA, 1997
Dermal Exposure	D_1	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004
	D_2	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004
Body Weight	BW	kg	Standard PQRA values (Table X)	Richardson, 1997

Inhalation of Urban Air

Equation 11 was used to calculate the daily intake of lead from inhaled outdoor and indoor urban air (Health Canada, 2004a).

$$INH_{urb\,air\_In} = \frac{C_{urb\,air\_In} \times IR_{air} \times RAF_{inh} \times D_1 \times D_2 \times D_3}{BW} \qquad (8)$$

Table 3.9. Model parameters used for the inhalation of outdoor and indoor air exposure pathway in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Outdoor Air Lead Concentration	C_{AirOut}	µg m3	0.06	Health Canada, 1992
Indoor Air Lead Concentration	C_{AirIn}	µg m3	0.045 (75% of the concentration of outdoor air)	OME, 2002 (based on Roberts *et al.*, 1974)
Receptor Air Intake Rate	IR_{Air}	m^3/hour	Standard PQRA intake rates (Table X)	Health Canada, 2004
Relative Inhalation Absorption for Lead in Air	RAF_{Inh}		0.64 (absolute AF of 32%)	Health Canada, 2004a; US EPA, 2005
Outdoor Air Exposure Duration	D_1	hours day	1.5	Modification of Richardson, 1997
	D_2	days 7days	7 days per week exposed 7 days	Health Canada 2004
	D_3	weeks 52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada 2004
Indoor Air Exposure Duration	D_1	hours day	22.5	Modification of Richardson, 1997
	D_2	days 7days	7 days per week exposed/ 7 days	Health Canada 2004
	D_3	weeks/52 weeks	52 weeks per year exposed 52 weeks	Health Canada 2004
Body Weight	BW	kg	Standard PQRA values (Table x)	Richardson, 1997

Hazard Assessment and Risk Characterization

The Estimated Daily Intake (EDI) of lead was calculated for each receptor age group in each housing age category by summing the intake for all pathways as follows:

$$EDI_{(mg\ kg\ day)} = Ing_{(Soil\ +\ Dust\ +\ Water\ +\ Produce\ +\ Food)} + Inh_{(Soil\ +\ Dust\ +\ Urb.Air)} + Derm_{(Soil\ +\ Dust\ +\ Water)} \quad (9)$$

where:

Ing = ingestion
Inh = inhalation
Derm = dermal contact

Health Canada has set the oral tolerable daily intake for lead (TDI) at 0.0036 (mg/kg·day) for PQRA (2004). Because there are no lead toxicological reference values for the inhalation and dermal exposure pathways, these intakes were adjusted for comparison to the oral pathway by multiplying by the relative bioavailability factors in the exposure assessment (Health Canada, 2004). The EDI for each scenario was divided by the TDI to determine how many times higher or lower the estimated intake was compared to the allowed intake (Equation 10). This is known as the Hazard Quotient (HQ).

$$HazardQuotient = \frac{EDI(mg/kg \cdot day)}{TDI(mg/kg \cdot day)} \quad (10)$$

Because TDIs represent the total intake that a receptor can be exposed to on a daily basis over a full lifetime without deleterious effects, exceeding the TDI (HQ >1) for a specific age category does not necessarily mean undue health risk, especially if it only applies to a small proportion of the total lifespan (Health Canada, 1996). Consequently, intakes were averaged over the entire lifetime using Equation 11 to produce the Cumulative Daily Intake (CDI) for each risk scenario.

$$TotalCDI = \sum_{1}^{n}\left(\frac{EDI_{1\ n} \times Time_{1\ n}}{70\,years}\right) \qquad (11)$$

where:

$EDI_{1\ n}$ = Estimated Daily Intake of age group n (mg/kg/day)
$Time_{1\ n}$ = time spent in each age group (years)
n = age group

Estimation of Blood Lead Using the IEUBK Model

The IEUBK model software provides a set of default values for all parameters based on expected exposure for urban residents who do not have any unusual lead exposure. The IEUBK model was run using a combination of IEUBK default parameters and parameters used in the PQRA average risk scenario (Table 3.10). IEUBK model default values were retained for maternal blood lead concentration, which is used to determine the lead levels in the child's organs at birth, absorption factors and ventilation and water intake rates. This was done because either specific data were not available for St. John's or the because the default values were considered good estimates. Values for all other model parameters were derived from the PQRA analysis.

Table 3.10. Parameter values applied from the PQRA analysis to the IEUBK model for the average risk assessment in the St. John's study.

Age (years)	Outdoor Air (µg/m³)	Indoor Air[1] (µg/m³)	Time Outdoors (h/d)	Ventilation Rate (m³/d)	Lung AF	Dietary Intake[2] (µg/d)	Dietary AF
0-1	0.06	0.045	1.5	2	0.32	4.18±G	0.5
1-2	0.06	0.045	1.5	3	0.32	5.50±G	0.5
2-3	0.06	0.045	1.5	5	0.32	6.48±G	0.5
3-4	0.06	0.045	1.5	5	0.32	7.45±G	0.5
4-5	0.06	0.045	1.5	5	0.32	8.47±G	0.5
5-6	0.06	0.045	1.5	7	0.32	7.25±G	0.5
6-7	0.06	0.045	1.5	7	0.32	8.32±G	0.5

Indoor Air[1] 75% of outdoor air
Dietary Intake[2] - Intake from supermarket food with or without intake from garden produce (G) which is based on soil lead concentration

Age (years)	Water Conc (µg/L)	Water Intake (L/d)	Water AF	Soil Conc[4] (µg/g)	Dust Conc[5] (µg/g)	Soil and Dust Intake (g/d)	Soil and Dust AF	Maternal Blood Lead (µg/dL)
0-1	4.8	0.20	0.5	901	1802	0.050	0.3	2.5
1-2	4.8	0.50	0.5	901	1802	0.080	0.3	2.5
2-3	4.8	0.52	0.5	901	1802	0.080	0.3	2.5
3-4	4.8	0.53	0.5	901	1802	0.080	0.3	2.5
4-5	4.8	0.55	0.5	901	1802	0.080	0.3	2.5
5-6	4.8	0.58	0.5	901	1802	0.020	0.3	2.5
6-7	4.8	0.59	0.5	901	1802	0.020	0.3	2.5

Soil Concentration[4] 50th percentile for the average assessment. Example in table taken from the pre-1926 housing category.
Dust Concentration[5] - Two time the soil concentration

In order to accommodate the receptor age categories used in the IEUBK model the following parameters had to be modified from the PQRA. For soil and dust intake for the IEUBK 0-1 age category the PQRA values for infants and toddlers were averaged, for IEUBK 1-5 year-olds the PQRA toddler rate was used, and for the IEUBK 5-7 year-old category the PQRA child rate was assumed to be appropriate.

Dietary lead intake from store-bought food was calculated from estimated daily intakes generated by the Total Diet Study (Health Canada, 2005b) multiplied by age-specific mean body weights (US EPA, 1997) and converted to µg/day. Just like for soil and dust intake, the dietary daily intake for the IEUBK 0-1 age category was calculated by averaging rates for infants and toddlers from the PQRA model run. For those scenarios including garden produce consumption daily lead intakes for garden produce types were combined and also multiplied by the mean body weight in each age category to generate age-specific lead intakes in µg/day. Because there was no specific pathway for dermal exposure and because the total lead intake for these parameters is so small, this pathway was not added to the model. Finally, the IEUBK model has a section for alternative sources of lead exposure but suggests it be used for the direct ingestion of lead-based paint (in addition to house dust), the use of leaded cosmetics or home remedies, or hobby or occupational exposures. None of these exposures were considered in this study. IEUBK 1-5 year-olds the PQRA toddler rate was used, and for the IEUBK 5-7 year-old category the PQRA child rate was assumed to be appropriate.

Dietary lead intake from store-bought food was calculated from estimated daily intakes generated by the Total Diet Study (Health Canada, 2005b) multiplied by age-specific mean body weights (US EPA, 1997) and converted to µg/day. Just like for soil and dust intake, the dietary daily intake for the IEUBK 0-1 age category was calculated by averaging rates for infants and toddlers from the PQRA model run. For those scenarios including garden produce consumption daily lead intakes for garden produce types were

combined and also multiplied by the mean body weight in each age category to generate age-specific lead intakes in µg/day. Because there was no specific pathway for dermal exposure and because the total lead intake for these parameters is so small, this pathway was not added to the model. Finally, the IEUBK model has a section for alternative sources of lead exposure but suggests it be used for the direct ingestion of lead-based paint (in addition to house dust), the use of leaded cosmetics or home remedies, or hobby or occupational exposures. None of these exposures were considered in this study.

Housing Age and Demographic Information for St. John's

The PQRA model calculated hazard quotients for designated property age categories in St. John's. Because housing stock age composition is available for each neighbourhood in St. John's, it was possible to map neighbourhood risk hazard (housing stock data maintained by the Community Accounts section of the Economics and Statistics Branch of the Province of Newfoundland and Labrador, 2007). Unfortunately, property age is not the same as housing age, as the former reflects the entire development history of the property, whereas the latter only represents the current building on the property. It is possible for a new house to be built on an old property with high soil lead concentrations and therefore have an elevated health risk. Despite the potential inaccuracy, property and housing age data were directly compared to facilitate neighbourhood hazard mapping (Table 3.12).

Table 3.11. A comparison of property age categories used in the PQRA risk assessment and dwelling age categories used to map neighbourhood risk.

Property Age Categories used in the PQRA	Dwelling Age Categories Available to Map Risk
Pre-1926	
1926 - 1948	Pre-1946
1949 - 1960	1946 - 1960
1961 - 1976	1961 - 1980
1977 - 1992	1981- 1990
Post-1993	1991 - 2001

Results

Average PQRA Exposure Assessment and Risk Characterization

When the 3MRA bioconcentration factors are used to estimate garden produce consumption in the average PQRA, hazard quotients (HQs) exceed unity for infants and toddlers living on all properties, children living on properties built before 1977 (soil lead concentration >95 ppm), and teenagers and adults living on properties built before 1961 (soil lead concentration >191ppm; Table 3.13). The HQs range from 0.35 for adults living on the newest properties, to 30.44 for infants living on the oldest properties. The HQs are lower when bioconcentration factors from Boyd *et al.* (1999) are used (Table 3.13). Hazard quotients exceed unity for infants living on all properties, toddlers and children living on properties built before 1977 (soil lead concentration >95 ppm), and teenagers and adults living on properties built before 1961 (soil lead concentration >191 ppm). In this scenario HQs range from 0.30 for adults living on the newest properties to 20.64 for infants living on the oldest properties. For the rest of the year when no garden

produce is eaten, or for those individuals who never consume garden produce, the HQs only exceed unity for infants living in pre-1926 housing (soil lead concentration >901 ppm) (Table 3.14). Without the consumption of garden produce the HQs range from 0.09 for adults living on the newest properties to 1.70 for toddlers living on the oldest properties. For all risk assessments the risk is higher for older properties and thus higher soil lead concentrations and also for younger receptors.

The percentage of lead intake from parameters associated with soil lead (ingestion of soil, dust, and garden produce) is highest on the oldest properties and progressively decreases on properties of younger age because the associated soil lead concentration also diminishes (Table 3.15). The upper range of values for soil and dust ingestion in older homes represents toddlers who potentially consume much more soil and dust than other receptors. All other parameters remain constant as property age decreases and thus their relative contribution to the total daily lead intake increases because soil related parameters are contributing less. Dermal contact and inhalation pathways contribute very little to the total daily lead intake and will not be discussed further (Table 3.15).

Table 3.12. Hazard quotients for the average risk assessment for the months where receptors are consuming garden produce.

	0 - < 6 months		5 months - < 5 years		5 years - <12 years		12 years - <20 years		20+ years	
Age of Property	Hazard Quotient (3MRA BCFs)	Hazard Quotient (Boyd *et al.* BCFs)	Hazard Quotient (3MRA BCFs)	Hazard Quotient (Boyd *et al.* BCFs)	Hazard Quotient (3MRA BCFs)	Hazard Quotient (Boyd *et al.* BCFs)	Hazard Quotient (3MRA BCFs)	Hazard Quotient (Boyd *et al.* BCFs)	Hazard Quotient (3MRA BCFs)	Hazard Quotient (Boyd *et al.* BCFs)
Pre-1926	**30.44**	**20.64**	**25.04**	**13.40**	**14.32**	**9.26**	**8.11**	**6.98**	**6.53**	**5.17**
1926 - 1948	**11.40**	7.77	**9.40**	**5.09**	**5.40**	**3.52**	**3.06**	**2.64**	**2.47**	**1.97**
1949 - 1960	**6.60**	**4.52**	**5.46**	**2.99**	**3.15**	**2.07**	**1.79**	**1.55**	**1.45**	**1.16**
1961 - 1976	**3.38**	**2.34**	**2.81**	**1.58**	**1.64**	**1.10**	0.93	0.81	0.76	0.62
1977 - 1992	**1.83**	**1.30**	**1.54**	0.91	0.91	0.64	0.52	0.46	0.43	0.36
1993 - Present	**1.46**	**1.05**	**1.24**	0.75	0.74	0.52	0.43	0.38	0.35	0.30

Note: all bolded HQs are greater than 1.00 and therefore represent elevated health risk.

Table 3.13. Hazard quotients for the average risk assessment for the months where receptors are not consuming garden produce.

		0 - < 6 months	5 months - < 5 years	5 years - <12 years	12 years - <20 years	20+ years
Age of Property	Geometric Mean Soil Lead Concentration (ppm)	Hazard Quotient	Hazard Quotient	Hazard Quotient	Hazard Quotient	Hazard Quotient
Pre-1926	901	0.95	**1.70**	0.33	0.19	0.17
1926 - 1948	334	0.47	0.75	0.21	0.13	0.12
1949 - 1960	191	0.35	0.51	0.18	0.11	0.10
1961 - 1976	95	0.27	0.35	0.16	0.10	0.09
1977 - 1992	49	0.23	0.27	0.15	0.09	0.09
1993 - Present	38	0.22	0.25	0.15	0.09	0.09

Note: all bolded HQs are greater than 1.00 and therefore represent elevated health risk

Table 3.14. The contribution of each average risk parameter to the percent of total daily lead intake. The percentage range represents the array of values for different ages of receptors.

	Including Garden Produce				Not Including Garden Produce	
Risk Parameter	Pre-1926 Housing (3MRA)	Pre-1926 Housing (Boyd *et al.)*	Post 1993 Housing (3MRA)	Post 1993 Housing (Boyd *et al.*)	Pre-1926 Housing	Post 1993 Housing
Ingestion of Fruit	81 - 75%	<1%	68 - 58%	<1%	n/a	n/a
Ingestion of Root Vegetables	13 - 7%	84 – 73%	11 - 6%	65 – 55%	n/a	n/a
Ingestion of Other Vegetables	11 - 6%	20 – 14%	9 - 5%	17 – 11%	n/a	n/a
Ingestion of Dust	4 - 1%	8 - 1%	4 - 1%	6 - 1%	63 - 36%	18 - 3%
Ingestion of Water	2 - 1%	1 - <1%	8 - 3%	10 - 7%	16 - 3%	33 - 19%
Ingestion of Soil	2 - <1%	3 - <1%	2 - <1%	3 - <1%	26 – 15%	7 - 1%
Ingestion of Supermarket Food	1 - <1%	1%	15 - 9%	20 - 13%	31 - 8%	69 - 53%
Dermal Contact with Dust	<0.1%	<0.1%	<0.1%	<0.1%	1 - <1%	<0.1%
Inhalation of Outdoor Air	<0.01%	<0.01%	<0.1%	<0.1%	<1%	<1%
Dermal Contact with Soil	<0.01%	<0.1%	<0.01%	<0.1%	<1%	<0.1%
Dermal Contact with Water	<0.001%	<0.001%	<0.01%	<0.01%	<0.01%	<0.1%
Inhalation of Indoor Air	<0.001%	<0.01%	<0.01%	<0.01%	2 - <1%	4 - 1%
Inhalation of Dust	<0.001%	<0.01%	<0.001%	<0.01%	<0.1%	<0.01%
Inhalation of Soil	<0.0001%	<0.0001%	<0.0001%	<0.0001%	<0.01%	<0.001%

n/a - parameter not included for this risk assessment

Garden produce consumption is the largest contributor to daily lead intake on older properties with high soil lead concentrations; however, the 3MRA model predicts that fruit is the largest contributor to daily lead intake, followed by root vegetables, and then other vegetables, whereas the model based on Boyd *et al.* (1999) predicts that root vegetables will contribute the most, other vegetables less, and fruit very little (Table 3.15). The ingestion of indoor dust is the next highest contributor for both models, followed by the ingestion of soil, water, and supermarket food.

On newer properties with lower soil lead concentrations the influence of soil related parameters on the daily lead intake declines. Garden produce still contributes the most, but the ingestion of supermarket food and water become more influential than the ingestion of dust and soil (Table 3.15).

For those residents on older properties who do not eat garden produce the most important pathway of lead exposure is dust ingestion, followed by the ingestion of supermarket food, soil, and drinking water. On newer properties supermarket food contributes the most to daily lead intake, while drinking water and dust and soil ingestion contribute less. The inhalation of indoor air also contributes a small amount to daily lead intake.

When the estimated daily intake of lead is averaged over a lifetime, hazard quotients are below unity for those individuals who do not eat garden produce (Table 3.16). Hazard

quotients do exceed unity for those receptors that consume garden produce and live on pre-1926 properties (Table 3.16).

Table 3.15. The cumulative daily intake of lead averaged over a lifetime of exposure for residents living on properties of different ages for the average risk scenario.

	Consuming Garden Produce for Three Months of the Year		Not Consuming Garden Produce
Age of Property	Hazard Quotient (3MRA)	Hazard Quotient (Boyd *et. al.*)	Hazard Quotient
Pre-1926	**2.43**	**1.83**	0.29
1926 - 1948	**0.96**	**0.74**	0.17
1949 - 1960	**0.59**	**0.46**	0.14
1961 - 1976	**0.34**	0.28	0.12
1977 - 1992	0.22	0.19	0.11
1993 - Present	0.20	0.17	0.11

Note: all bolded HQs are greater than 1.00 and therefore represent elevated health risk.

IEUBK Model Blood Lead Predictions

Predicted geometric mean blood lead concentrations were at or above 10 µg/dL for children living on properties developed before 1993, consuming garden produce, and having a bioconcentration factor defined by the 3MRA model. Similar results were obtained for children living on properties developed before 1977 and consuming garden produce according to the Boyd *et al.* model (Table 3.17). In contrast, no children had predicted geometric mean blood lead concentrations above 10 µg/dL when garden produce consumption was not part of the model.

The blood lead concentrations discussed above are predicted averages only, and there still may be children who have higher concentrations based on individual variations in behaviour and physiology. For example, as many as 41% of children who do not consume garden produce, living in pre-1926 housing, may exceed the safe guideline of 10 µg/dL, even though mean blood lead concentration in this population of children is below this level (Table 3.17).

Table 3.16. The IEUBK predicted geometric mean blood lead concentrations for children 0 to 7 years and the percentage of children above 10 µg/dL.

	Including Garden Produce (3MRA)		Including Garden Produce (Boyd *et al.*)		Not Including Garden Produce	
Age of Property	GM Blood Lead (µg/dL)	Percent Above 10 µg/dL	GM Blood Lead (µg/dL)	Percent Above 10 µg/dL	GM Blood Lead (µg/dL)	Percent Above 10 µg/dL
Pre-1926	**60.53**	99.99	**43.90**	99.92	9.00	41.15
1926 - 1948	**34.67**	99.59	**24.91**	97.39	4.72	5.50
1949 - 1960	**25.10**	97.49	**17.56**	88.46	3.46	2.20
1961 - 1976	**16.19**	84.72	**10.99**	57.94	2.56	0.19
1977 - 1992	**10.30**	52.50	6.95	21.96	2.12	0.05
1993 - Present	8.67	38.03	5.87	12.88	2.01	0.03

Note: all bolded HQs are greater than 1.00 and therefore represent elevated health risk.

Spatial Patterns of Health Risk in St. John's

There is a strong association between geometric mean soil lead concentration and property age in the City of St. John's (Campbell, 2007a) and therefore for those health risk scenarios that are closely linked to soil lead concentrations, the spatial distribution of housing stock by age is a good proxy for the spatial pattern of related health risk. In Figures 3.1 to 3.4, the percent housing stock of a specific age is mapped by city

neighbourhood. Each map shows the potential health risk by neighbourhood for a specific group of receptors with certain behaviours. For example, Figure 3.1 shows the percent of housing stock built before 1946 in each neighbourhood, which roughly represents the increased lifetime CDI risk when garden produce is consumed by receptors (using either 3MRA or Boyd *et al.* bioconcentration models). It is also a conservative proxy for increased EDI risk in toddlers who do not eat garden produce. In this example, the mapped risk values are described as conservative because the risk scenarios are specifically associated with pre-1926 properties, data on which are not available by neighbourhood, and so the next oldest property age category is mapped instead. The percent of houses built before 1960 illustrates the increased EDI health risk for teenagers and adults who consume garden produce (using either 3MRA or Boyd *et al.* bioconcentration models), as well as elevated blood lead levels for children under 7 as predicted by the IEUBK model that uses garden produce values based on Boyd *et al.* (Figure 3.2). The percent of houses built before 1980 which represents increased EDI risk for children (using either 3MRA or Boyd *et al.* bioconcentration models) and toddlers (Boyd *et al.* model only) who consume garden produce (Figure 3.3), and the percent of houses built before 1990 which may be associated with elevated blood lead levels in children under seven according to the IEUBK model using 3MRA garden produce data (Figure 3.4). All houses may produce an increased EDI health risk for infants (using either 3MRA or Boyd *et al.* bioconcentration models) and toddlers (3MRA model only) consuming garden produce. This was not mapped as it includes the entire city.

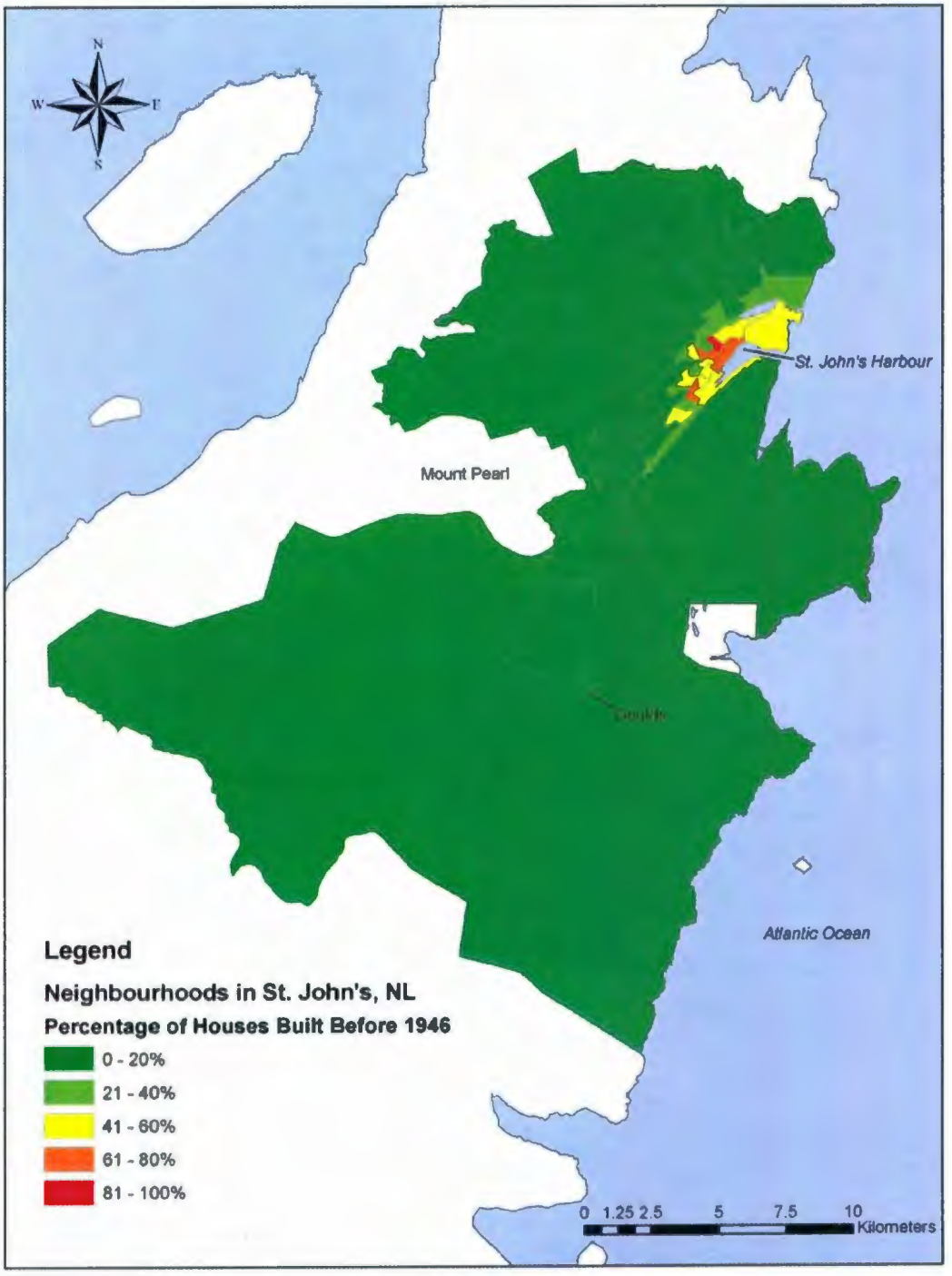

Figure 3.1. The percentage of neighbourhood houses built before 1946 (Community Accounts, 2007).

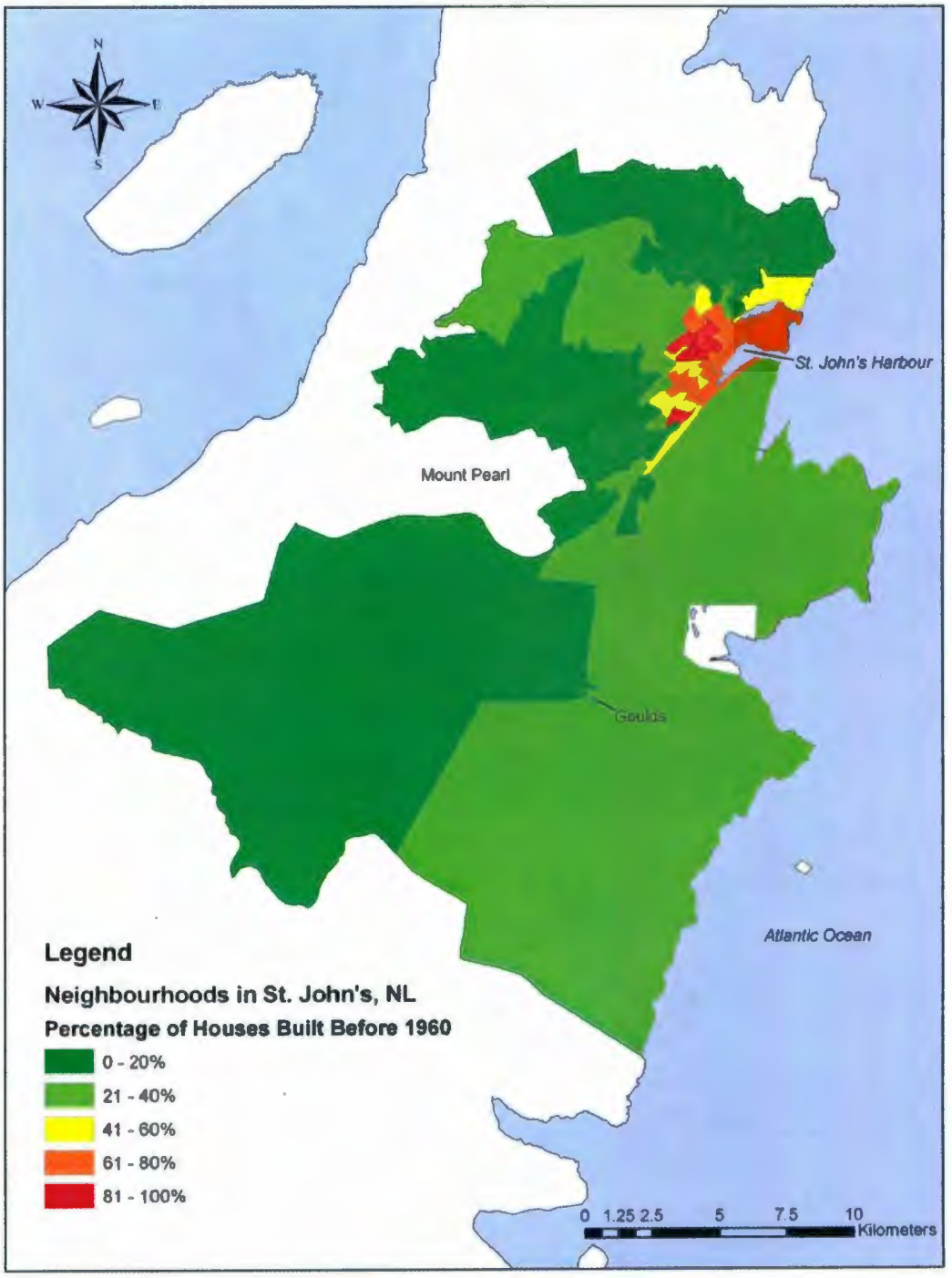

Figure 3.2. The percentage of neighbourhood houses built before 1960 (Community Accounts, 2007).

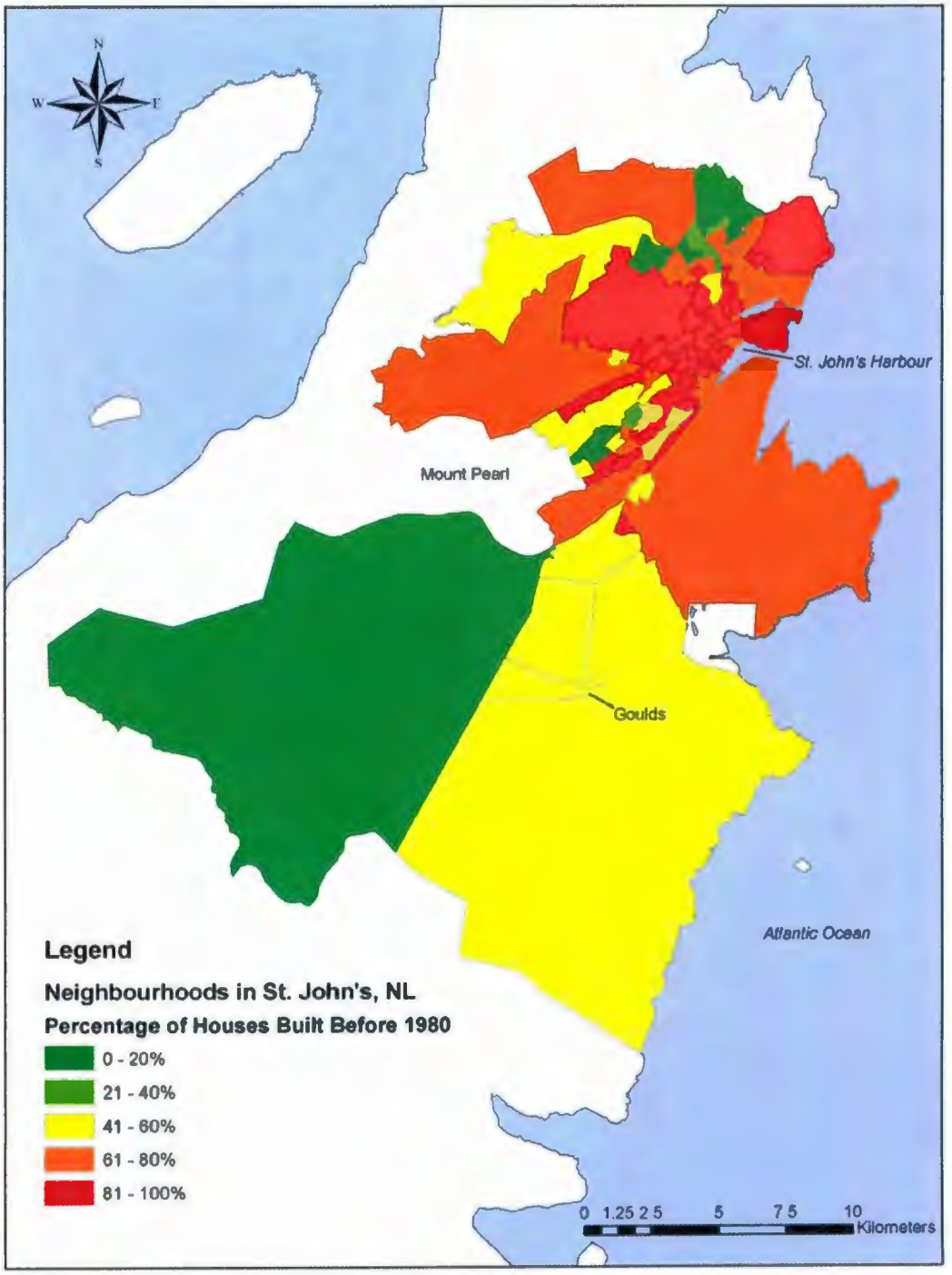

Figure 3.3. The percentage of neighbourhood houses built before 1980 (Community Accounts, 2007).

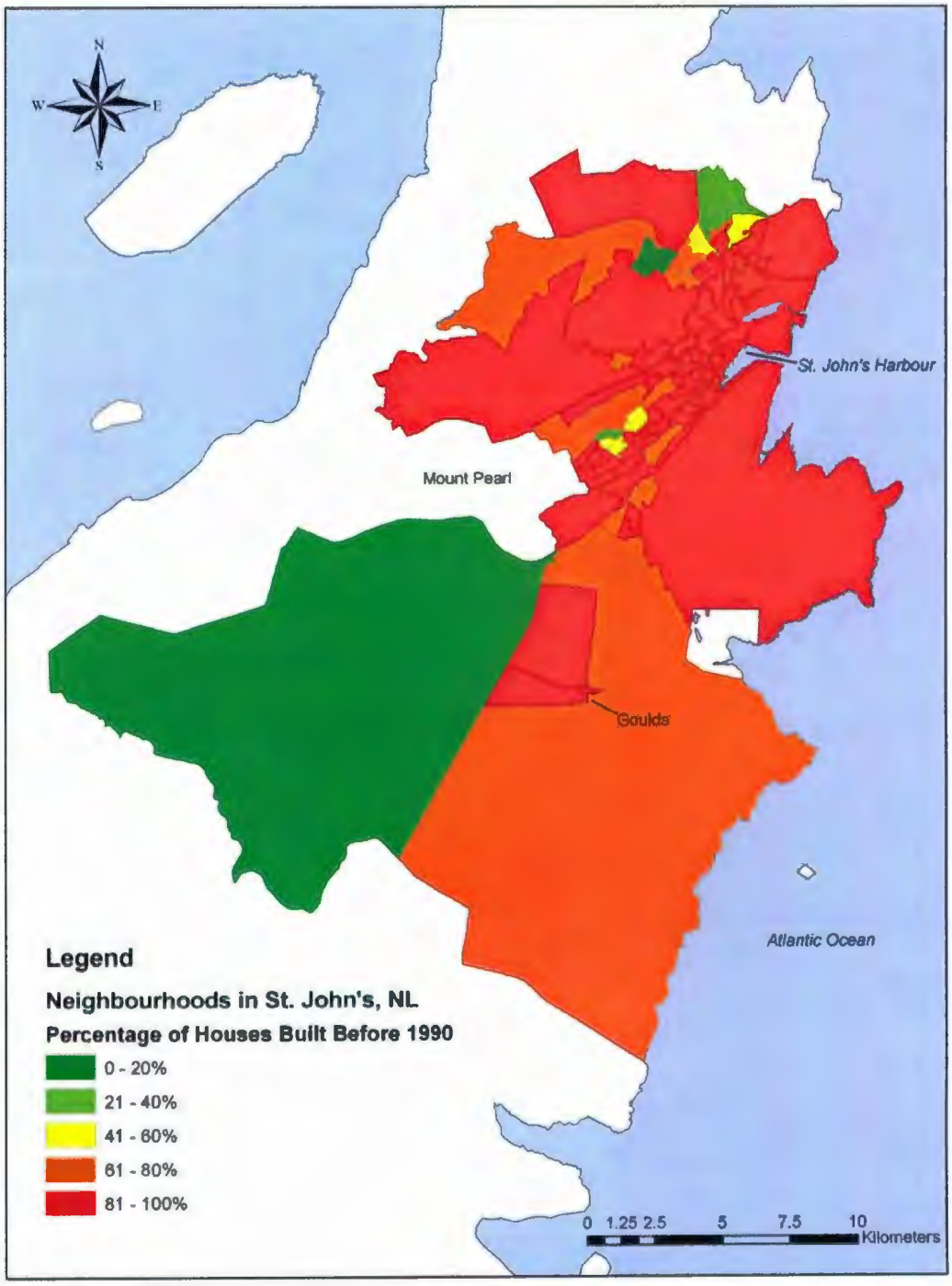

Figure 3.4. The percentage of neighbourhood houses built before 1990 (Community Accounts, 2007).

Discussion

Preliminary Quantitative Risk Assessment

When average risk scenario intakes were averaged over an entire lifetime the CDIs of lead only exceeded the TDI for receptors living on pre-1926 properties who consume garden produce for three months of the year (soil lead concentration greater than 901 ppm). This scenario may be unrealistic as most individuals will move residences and thus have changing soil exposure over their life span, and many may not consistently eat garden produce, this potentially overestimates the lifetime CDI risk. Risk was not elevated for the other property/soil lead categories because the consumption of garden produce was only considered for three months of the year, and therefore the high HQs associated with produce ingestion had a lesser effect on the overall CDI.

While examination of elevated age-specific EDI hazard quotients may not provide a true indication of increased health risk, it may indicate temporary increases in the body burden of lead. This may be important for young children who are most susceptible to lead's chronic low-level effects, especially since these are the receptors with the highest hazard quotients in all the risk scenarios. For EDIs that did not include garden produce ingestion elevated risk occurs only for toddlers on pre-1926 properties (soil lead concentration 901 ppm), but for the three months when garden produce was included elevated risks occurred on much newer properties with much lower soil lead concentrations. In fact during the growing season all properties in St. John's may pose a

risk for infants and toddlers, children may be at risk in houses built before 1977 and teenagers and adults may be a risk in houses built before 1961.

The impact of garden produce was also evident in the risk assessment conducted in Belledune, New Brunswick (Government of New Brunswick, 2005). In general lead intake was dominated by wild mussels and local fish, but for the infants and toddlers at the first site with elevated HQs lead intake was primarily from garden vegetables and secondarily from soil exposure. For the toddlers at the second site with elevated HQs lead exposure was mainly due to soil intake and only at upper bound soil lead concentrations. Otherwise soil ingestion, dermal exposure and supermarket food contributed very little to the overall exposure. The importance of garden vegetable and soil intake for infants and toddlers in particular was mirrored in St. John's although garden produce also was a large contributor to lead intake for the other receptors. This might be because of the much larger soil lead concentrations in St. John's, or it could also be that the garden produce models used in the St. John's risk assessment overestimated lead uptake since unlike the Belledune assessment no actual produce samples were analyzed.

Infants were at the most risk when garden produce was entered into the risk assessment, perhaps because their very low body weights causes a larger lead body burden per kilogram. Of course whether or not infants consume garden produce may be questionable. For much of the time they would consume only breast milk or formula, with only a slight chance that parents would make their own baby food out of garden

produce. Perhaps the large hazard quotients for infants consuming garden produce should be questioned. When garden produce consumption was not considered in the risk assessments, toddlers had the highest risk of negative health effects. This may be because without the large impact of garden produce ingestion, a toddler's high soil and dust intake rates may have the greatest impact on lead intake.

The CCME has set the residential soil lead guideline at 140 ppm. When garden produce is not considered potential health risks occur closer to 1000 ppm (901 in the average risk assessment and 1011 ppm in the conservative risk assessment). However when the risk assessment includes the ingestion of backyard produce during the three month growing season health risks may occur for infants and toddlers at soil lead concentrations as low as 38 ppm according to the average risk scenario. This risk is only seasonal and may diminish over the course of the year.

IEUBK Blood Lead Predictions

The predicted IEUBK geometric mean blood lead concentrations that did not exceed 10 µg/dL when garden produce consumption was not considered, although there was a 41% chance that children living in pre-1926 housing may exceed this limit. Predicted blood lead concentrations were much higher when the consumption of garden produce was included in the model. Concentrations were elevated for children living on properties developed before 1993 according to 3MRA data and before 1977 according to Boyd *et al.* (1999) data. An evaluation of the most important pathways once again point to diet

(garden produce and supermarket food) and to a much lesser extent dust and soil ingestion. Blood lead concentrations were predicted to be as high as 61 µg/dL for the three months children living in pre-1926 housing consume garden produce (soil lead concentration >901 ppm). This seems very high as children with this blood lead would present clinically with acute lead poisoning and this has not been identified as a problem in St. John's.

For those properties where geometric mean blood lead levels did not surpass 10 µg/dL there is still a chance that some children may exceed this guideline due to variability in individual behaviour and physiology. Risk management protocol in the US EPA aims to limit the risk of a child exceeding 10 ug/dL to less than 5%. This means mitigative measures might need to be undertaken on properties where this percentage is surpassed. In St. John's all properties would need to undergo mitigation if garden produce is being consumed and mitigation might also be necessary for all properties developed before 1949 when garden produce consumption is not an issue. Suggested measures to reduce exposure are discussed in Chapter 4.

The plausibility of the average predictions was determined by comparing them to values generated using other established models, preliminary blood lead data for St. John's, and to blood lead levels measured in other Canadian cities

Geometric mean soil lead concentrations and dust lead loadings measured on properties in St. John's were entered into the model created by Lanphear *et al.* (1998). Geometric mean blood lead concentrations were found to be either slightly higher or slightly lower than those predicted by the IEUBK model for the average risk scenario when garden produce was excluded, making the IEUBK predictions plausible (Table 3.18). Values generated by the IEUBK model may differ from the Lanphear *et al.* (1998) predictions because the IEUBK model uses dust concentrations estimated from the observed soil lead concentrations whereas the Lanphear model uses actual measured dust lead loadings.

In contrast, results from a preliminary blood lead study in St. John's and a retrospective chart review indicate that the blood lead levels of children in St. John's are much lower than predicted by either model.

Table 3.17. Geometric mean blood lead concentrations and the probability of exceeding 10 µg/dL as predicted by the model generated by Lanphear *et al.* (1998) using soil and dust values from St. John's compared to the same parameters estimated from the IEUBK model.

Property Age	GMean Soil Lead Concentration (ppm)	GMean Floor Dust Lead Loading (µg ft^2)	Lanphear GM Blood Lead (µg dL)	IEUBK GM Blood Lead (µg dL)	Lanphear Probability >10 µg dL (%)	IEUBK Probability >10 µg dL (%)
<1926	901	15.5	6.5	9.0	21.3	41.2
1926 - 1948	334	6.1	4.6	4.7	7.7	5.5
1949 - 1960	191	1.0	3.0	3.5	1.6	2.2
1961 - 1976	95	2.1	3.2	2.6	2.2	0.2
1977 - 1992	49	0.2	< 2.6	2.1	< 0.8	0.1
1993 - Present	38	1.9	2.8	1.8	1.3	0.0

The pilot blood lead study gained access to 113 anti-coagulated blood samples collected for other clinical purposes from children aged 6 months to 6 years of age (Allison, 2006). The samples were stripped of personal information except for an identification number, the postal code, age of child, and health care number. Samples were analyzed using atomic absorption spectrometry and the arithmetic mean for all samples was 0.156 µmol/L (3.25 µg/dL) and the geometric mean was 0.098 µmol/L (2.04 µg/dL). It was estimated that approximately 4-5% of children may have elevated blood lead levels which is slightly higher than the 2% estimated by combining the IEUBK predicted blood lead levels and neighbourhood demographic information for the entire city, but less than the percentage predicted for individual neighbourhoods in the downtown core, which range from 4 to 10%.

The postal codes of the samples were matched to 59 of the city's 95 neighbourhoods and the blood lead concentrations were compared to the soil concentration for the neighbourhood. Levels below 140 ppm were deemed acceptable, while levels above this value were considered high. In the analysis no association between blood lead and soil lead or dwelling age was found.

A chart review was also conducted which looked at records in the Meditech system for the province of Newfoundland and Labrador from May 1993 to May 2004 (O'Brien, 2006). 1028 sets of blood lead data were retrieved with 263 repeats (Table 3.19). The geometric mean for children under 12 was 0.15 µmol/L (3.13 µg/dL), for children between 13 and 15 it was 0.13 µmol/L (2.71 µg/dL), for teens 16 to 18 it was 0.09

µmol/L (1.88 µg/dL), and for adults over 18 it was 0.2 µmol/L (4.17 µg/dL). The geometric mean for children under twelve was slightly higher than the value obtained in the pilot study for children under six, but the chart audit only examined blood samples that had already been targeted for lead testing which may have overestimated the true blood lead concentration for the region.

Overall 4.2% of children under 18 in the province had blood lead levels above the threshold of 0.48 µmol/L (10µg/dL), which corresponds to the estimate given for children under six in St. John's. Of those children with elevated results the majority were for males (6 out of 7), although the geometric mean for both sexes was not significantly different. For the other samples with a known location, blood lead concentrations were higher in Health Regions for Western and Central Newfoundland as well as Labrador (mean = 0.24 µmol/L / 5.00 µg/dL) then for St. John's (mean = 0.15 µmol/L / 3.13 µg/dL) or the Eastern Health Region (mean = 0.13 µmol/L/ 2.71 µg/dL).

Table 3.18. The number of unique blood lead data sets in the chart review of Newfoundland and Labrador.

Age Range	n
0-3	49
4-6	29
7-9	25
10-12	20
13-15	28
15-18	17
>18	597
Total	765

Blood lead concentrations found in both the St. John's pilot study and the chart audit are similar to values measured in other Canadian cities (Table 3.20), even though all of the other monitored sites were associated with historical or current industrial point sources of pollution. It is not that St. John's blood lead concentrations are high for a non-industrial city; rather increased industrial pollution controls and the elimination of leaded gasoline and paint have decreased the lead burden across the country, even at those sites directly impacted by lead pollution.

Table 3.19. Recent children's blood lead concentrations measured in other Canadian cities.

Year	Location	Age Range	Blood Lead Level	Percent Over 10 µg/dL	Reference
1990	Murdochville,QC	6 m-5 yrs	Mean 5.83		Chagnon and Bernier, 1990
1990	Murdochville,QC	5-12 yrs	Mean 6.67		Chagnon and Bernier, 1990
1991	Trail, BC	<6 yrs	13.5	83%	Hilts et al, 2001
1991	St-Jean-sur-Richelieu, QC	6 m-10 yrs	6 m-10 yrs	GM 5.0	Goulet *et al.*, 1996
1992	South Riverdale, TO, ON	<6 yrs	GM 3		Langlois et al, 1996
2000	Trail, BC	<6 yrs	6.7	27%	Hilts et al, 2001
2001	Sydney, NS	1-5 yrs	GM 1.86	0%	NSDH and CBDHA, 2001
2001	Port Colborne, ON	<7 yrs	GM 2.3	0%	Decou et al, 2001

The predicted IEUBK blood lead concentrations for St. John's were similar to other Canadian studies, and only slightly higher than those found in the pilot study and chart review; however, when garden produce was included the predictions were much higher. Several other studies have found that IEUBK predictions were higher than actual blood lead measurements. In Trail, B.C. the IEUBK model successfully predicted blood lead levels in 1996, but in later analyses in 1999 and 2001 the model continued to predict at a

similar level while the actual levels dropped to less than half the 1996 levels (Hilts, 2003). The only major changes that took place were a significant drop in air lead levels due to the replacement of the old lead and zinc smelter with a new facility and the introduction of an educational initiative (Hilts et al, 2001). Air emission reductions have both direct and indirect effects on blood lead levels. It directly decreases lead inhalation, although this is only a small proportion of the total lead exposure, and it also indirectly decreases dust lead levels and therefore lowers ingestion of lead. Perhaps dust is a more important and complex exposure pathway than the IEUBK model accounts for. The model does incorporate a strong relationship between soil lead and blood lead concentrations, predicting a 7 μg/dL increase in blood lead for every 1000 ppm increase in soil lead. Other studies indicate that the link may not be so strong or not even present (Tsuji and Serl, 1996), especially for soil lead concentrations less than 1000 ppm, which are common values for non-industrial sites. These studies indicate that blood lead is indirectly related to soil lead via house dust, rather than directly. They also point out that air lead and paint lead may be other important contributors affecting house dust lead and ultimately blood lead via hand to mouth activity. In a discussion of the conceptual structure of the IEUBK model White *et al.* (1998) also suggest that future improvements to the model would need to address differences in indoor dust versus outdoor soil exposure, particularly the impact on dust levels if there are indoor sources of lead (ie. paint).

Variability

Natural variability of both environmental and receptor characteristics were dealt with in this study using a number of different strategies. Spatial differences in soil lead concentrations within properties were dealt with by averaging the values for all locations (roadside, dripline, and ambient). Because older properties were exposed to larger quantities of lead and for a longer period of time, this temporal difference was accounted for by creating six property age categories. Soil, dust, garden produce and water concentrations were varied according to these categories. Because current air concentrations are more evenly distributed spatially this variable was not modified based on property age but instead was partitioned into indoor and outdoor components. The plant-specific nature of lead uptake in garden produce was accounted for by averaging plant-specific values for those varieties grown in St. John's and by grouping them into root vegetables, other vegetables, and fruit.

The natural variability of receptors was accounted for by dividing them into five age categories as intake rates and body weights vary with age. Dividing receptors into different age categories was also important because young children are more susceptible to lead exposure due to higher soil/dust ingestion rates and loading factors. Lead exposure is also very seasonal with higher exposure to outdoor sources occurring in the warmer months. Seasonality was addressed in this risk assessment by calculating risk for the three summer months where garden produce is consumed separately from the risk for the rest of the year. Other high summer exposure values (body surface area exposed and

the time spent outdoors) were averaged with low winter values to produce a daily value that was representative of the year as a whole and not of specific seasons. This may mask seasonal increases in exposure. Inter-individual differences in receptors, besides age-related differences, are hard to incorporate into the risk assessment and for the most part only age-specific average intake rates and body weights were used.

Uncertainties and Limitations

Uncertainty in risk assessment refers to the lack of knowledge of factors that affect risk and can lead to inaccurate or biased estimates (US EPA, 1997). Uncertainty can be reduced by eliminating knowledge gaps. There are two ways to address uncertainty in risk assessment. The first is an uncertainty characterization which qualitatively discusses the thought process that lead to the selection or rejection of specific data, estimates, and scenarios (US EPA, 1992b), as discussed in Appendix B. Additionally, a qualitative exploration of the effect of assumptions on the predicted PQRA risk estimate was carried out in Appendices D and E. Alternatively, uncertainty can be quantitatively assessed with a sensitivity analysis, analytical uncertainty propagation, probabilistic uncertainty analysis, or classical statistical methods. This was deemed beyond this scope of this research project.

The following is a brief description of the major uncertainties in the PQRA and IEUBK risk assessments; for a more detailed analysis see Appendices D and E.

Uncertainty in the Preliminary Quantitative Risk Assessment

One of the major sources of uncertainty for the PQRA risk estimates in the St. John's study concerns those values for environmental parameters that were estimated from other studies. This included indoor dust, garden produce, drinking and bathing water, outdoor and indoor air, and supermarket food. Until local values are collected for St. John's, estimated values will always generate a level of uncertainty.

Uncertainties in the major contributing pathways to daily lead intake are the major sources of uncertainty for the overall risk assessment and should be continually reviewed to improve the accuracy of results. Regardless of property age or whether or not conservative or average assumptions were used, the contribution of the dermal and inhalation pathways were minimal, thus the uncertainties associated with these pathways are not a major concern. However, the impact of the consumption of garden produce was large; in fact it was large enough to warrant running the risk assessment with and without this parameter. Uncertainties associated with soil and dust exposure may also be important as these pathways were dominant when soil concentrations were high on older properties, and were especially influential when garden produce was not an issue. Uncertainties regarding drinking water and supermarket food ingestion may play a more important role in scenarios where soil lead concentrations were low.

For the most part parameters were chosen to overestimate risk in order to protect the most sensitive cohorts of the population. This was especially true for the conservative risk

assessment. Out of all the parameters dust and soil ingestion and garden produce consumption had the highest number of underestimated parameters. The soil and dust ingestion rate might have been underestimated because the soil intake was split in two to create soil as well as indoor dust components. Dust absorption might also have been underestimated because it was assumed to be the same as soil, but a high organic content may increase concentrations and the smaller particle size may make indoor dust more bioavailable (Rasmussen, 2004). Lead intake from garden produce might have been underestimated because it only considered root uptake, and didn't include direct deposition for above ground plants (although air concentrations were predicted to be minimal in St. John's). The exposure duration was also estimated for only three months, but there may be a few individuals who freeze or preserve their produce for consumption outside the growing season. In addition to a few potential underestimations garden produce had the most uncertainty because the impact of the two uptake models is unknown. Without actual garden produce sampling it is difficult to predict the lead concentrations in backyard produce, as so many factors are influenced by site-specific conditions.

Uncertainties in the IEUBK Predictions

The default values of the IEUBK model have been empirically determined to provide the best prediction of blood lead when no site specific data are available. The model has also been independently verified, so making changes to the defaults may have influenced the accuracy of the predicted results.

In general the values chosen for most parameters were similar to those used as defaults; however, several parameters differed considerably and may have potentially overestimated risk (Table E.1). First, very high soil lead concentrations were used for older properties, and based on the plant uptake models this caused a very high dietary intake. Second, indoor dust concentrations were also much higher because they were estimated to be twice the soil concentrations instead of the IEUBK recommended 70%. Last, water concentrations were estimated to be higher than the suggested values for older properties to account for the potential presence of leaded pipes or solder. As previously discussed, the IEUBK model does not make accurate blood lead predictions if extreme values are used, which may have caused some exaggeration of blood lead results and therefore the results may not useful for risk assessment purposes.

Conclusions

According to the PQRA the cumulative lifetime risk in the average risk scenario is negligible for all receptors not consuming garden produce. For those receptors who do consume garden produce, cumulative lifetime risk may be an issue for those living on pre-1926 properties (soil lead concentration >901 ppm). The age specific hazard quotients in the average risk scenario without garden produce indicates that there may be a temporary increased health risk for toddlers living in pre-1926 housing (soil concentration >901 ppm). When backyard produce ingestion is included temporary health risks may occur for infants and toddlers on all properties (soil concentration >38 ppm).

children on properties developed before 1977 (soil concentration >95), and teenagers and adults on pre-1961 properties (soil concentration >191 ppm).

The blood lead levels predicted by the IEUBK model using the average risk scenario show a larger risk for children seven years and under than suggested by the PQRA risk assessment. While there is moderate risk if garden produce is not consumed (41% of children living in pre-1926 housing may have a blood lead concentration higher than 10 µg/dL), the consumption of garden produce causes an elevated risk to all children living in pre-1993 or pre-1977 housing (soil concentration >49/95 ppm) depending on the garden model used. The percentage of children with potentially elevated blood lead levels remains high for all homes in St. John's regardless of whether the predicted geometric mean blood lead concentration exceeds 10 µg/dL. Because of the IEUBK model's tendency to overestimate blood lead levels under extremely high soil lead concentrations these results should be interpreted cautiously, especially the predicted concentrations that indicate acute lead poisoning. Comparisons with other data from St. John's and across Canada indicate that the predicted blood lead concentrations for the scenario without garden produce may be the most realistic.

Overall the results of the average risk assessments for both the PQRA and the IEUBK model suggest that cumulative lifetime risk of negative health risks occurring in St. John's may be a problem for those individuals living on the oldest properties with the highest soil lead concentration (>901 ppm). Toddlers living on these properties may also

have a temporary increase in health risk even if no garden produce is eaten. It appears that migitation measures for these properties should be undertaken at the very least.

There is a large amount of uncertainty associated with the estimation of lead concentrations in garden produce, which makes interpreting the risks associated with garden produce exposure difficult to determine. Both models generate high lead exposures, which over the short harvest season appears to greatly increase the health risk for all receptors living in pre-1961 housing (soil concentration >191 ppm) and for infants and toddlers in new homes built after 1993 (soil concentration >38 ppm). Because of the large amount of uncertainty the risk analysis would greatly benefit from a survey of garden produce to determine actual lead concentrations for St. John's. Until that is done the most conservative recommendation would be to avoid planting any vegetables in local earth and to use raised beds or containers filled with store-bought soil.

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Chapter 4 Summary

Environmental Sampling of Soil and Dust

Approximately half of the surface soil samples collected in St. John's exceeded CCME guidelines resulting in a geometric mean soil lead concentration of 162 ppm for all samples. This is higher than some Canadian cities, notably Ottawa and Vancouver, but lower than sites with a point source of lead pollution such as Sydney, N.S., and Trail, B.C. However, when the soil samples were categorized according to property age striking patterns emerged. While suburban properties developed after 1960 and urban properties developed between 1926 and 1960 had minimal to moderate soil lead geometric means (a range from 38 ppm to 334 ppm) properties in downtown St. John's had much higher levels (901 ppm). This level of soil lead concentration is higher than Sydney or Trail and puts central St. John's on par with studies done in larger American inner-cities (Mielke, 1994). If increases of 1000 ppm soil lead are related to a 2 to 7 µg/dL increase in blood lead (Lanphear *et al.*, 1998) and average blood lead levels are around 2µg/dL (Schemberger *et al.*, 2005) then it is possible for children living in downtown St. John's to be at risk for elevated blood lead, especially since there is recent evidence that negative health effects can occur at less than 5 µg/dL (Lanphear *et al.*, 2000).

The data for the indoor dust lead survey in St. John's was highly skewed, with most samples containing very little lead. As such geometric mean dust lead loadings did not exceed US EPA standards for any sample location (window sill, entrance or kitchen

floor) even when the data was stratified by housing age. However 11 houses sampled did exceed those standards. Those houses with elevated samples were generally built before 1950 and had dripline soil lead concentration above 900 ppm. Recent research has indicated blood lead levels increase dramatically at floor dust loadings of 5-10 $\mu g/ft^2$ (Lanphear *et al.*, 1998). Forty-four percent of entrance and kitchen floor samples were above 5 $\mu g/ft^2$ and 24% were above 10 $\mu g/ft^2$, mainly in houses built before 1950.

In St. John's there is reason to be concerned about soil and dust lead exposure for individuals, particularly children, living in houses built in the first half of the twentieth century as a proportion of those houses have soil and dust lead levels above guidelines.

An Exercise in Risk Assessment for Lead Exposure

Cumulative lifetime effects may be a problem for those individuals living on pre-1926 properties (soil lead concentration >901 ppm), but only when garden produce is being consumed. Health risk may be temporarily increased for toddlers living on properties developed before 1926, even if they are not consuming garden produce. For all other receptors increased risk occurs only when backyard produce is eaten. This occurs for infants and toddlers on all properties (soil lead concentration >38 ppm), children on properties developed before 1977 (soil lead concentration >95 ppm), and teenagers and adults on properties developed before 1961 (soil lead concentration >191 ppm).

The blood lead levels predicted by the IEUBK model show a larger risk for children seven years and under than suggested by the PQRA risk assessment. While there is moderate risk if garden produce is not consumed (41% of children living in pre-1926 housing may have a blood lead concentration higher than 10 µg/dL), when garden produce is included the risk jumps sharply, extending to all children living in pre-1993 or pre-1977 housing (soil concentration >49/95 ppm) depending on the garden model used. When backyard produce is consumed the percentage of children with potentially elevated blood lead levels remains high for all homes in St. John's regardless of whether the predicted geometric mean blood lead concentration exceeds 10 µg/dL. Because of the IEUBK model's tendency to over estimate blood lead levels under extremely high soil lead levels these results should be interpreted cautiously, especially the predicted concentrations that indicate acute lead poisoning. Comparisons with other studies conducted in St. John's and across Canada indicate that the predicted blood lead concentrations for the scenario without garden produce may be the most realistic.

Overall the results for both the PQRA and the IEUBK models suggest that cumulative lifetime risk of negative health risks occurring in St. John's may be a problem for those individuals living on the oldest properties with the highest soil lead concentration (>901 ppm). Toddlers living on these properties may also have a temporary increase in health risk even if no garden produce is eaten. It appears that mitigation measures for these properties should be undertaken at the very least. Trying to interpret the risk assessments including garden produce is difficult because there exists a large amount of uncertainty.

Without actual produce sampling it is difficult to predict the lead concentrations in backyard produce, as so many factors are influenced by site-specific conditions and modelling results are so heavily influenced by the choice of BCF.

Even without being totally sure of the garden risk assessment results it is still safe to say that the main source of lead intake for St. John's residents is garden produce, and when that was not consumed the ingestion of indoor dust, supermarket food, soil, and drinking water contributed the most to the total daily lead intake. Reducing exposure to these pathways would be the most beneficial and methods are discussed in the Recommendations section.

Combined Outcome of Environmental Sampling and Risk Assessment

The CCME recommends that soil lead concentrations be kept below 140 ppm on residential properties. This is a conservative estimate based on year-round exposure for the highest susceptible receptors, children. Currently there are residents in St. John's who may be at risk at or below this soil lead level. In the average scenario infants, toddlers, and children who consume garden produce may be at risk below the CCME guideline (>38 ppm and >95 ppm, respectively). This range is consistent with the beginning of elevated blood lead concentrations in the average IEUBK model. This expands to include teens in the conservative risk assessment (>117 ppm). If garden produce is not eaten then the safe soil lead concentration rises to over 900 ppm according to the average risk assessment and 420 ppm in the conservative risk assessment. If all residents were advised

not to grow vegetables or fruit in their soil then the number of hazardous samples would decrease from 51% to 25% based on a conservative safe level of 420 ppm, or from 51% to 12% based on an average safe level of 900 ppm.

Despite predictions that many residents may be at increased health risk for lead exposure, medical studies in St. John's have not shown generally elevated blood lead results. A chart review of blood records indicated a geometric mean of 3.13 µg/dL for children under 12 (O'Brien, 2006) and a pilot blood lead study found a geometric mean of 2.04 µg/dL for children under six (Allison, 2006). No association was found between blood lead levels and soil lead concentrations for those blood samples that could be matched to a specific St. John's neighbourhood. These studies did not target children with high environmental lead levels; instead they were an opportunistic sample which may explain the low blood lead findings. Alternatively it is possible that the risk assessments conducted in this study overestimated the transfer of lead in the environment to human receptors.

Recommendations

Without the medical evidence to suggest serious health risks to children it is difficult to suggest extreme remediation measures such as soil and paint removal, especially since these methods can actually increase health risks if not done properly. Instead, to reduce the risk it is obvious that the consumption of garden produce on local soil should be

eliminated and exposure to high soil lead limited for those houses with soil lead above 900 ppm.

There are several suggestions in the literature for urban gardeners. For highly contaminated soil it is best to plant vegetables in containers or raised beds using new topsoil and to make sure there is a semi-permeable barrier between the new and old soil to allow water flow but prevent mixing (Finster *et al.*, 2004). If this is not feasible there are several other steps gardeners can take.

First, it is important to avoid planting any produce close to the foundation of buildings, especially those with visible paint deterioration, as these locations often have the highest soil lead concentrations. Second, fruiting plants such as tomatoes, cucumbers, and strawberries should be preferentially planted over root and leafy vegetables as lead tends to accumulate in the lower regions of the plant, in roots, stems, and leaves and also because soil adheres more easily to root and leafy parts of the plant (Samsoe-Petersen *et al.*, 2002; Finster *et al.*, 2004). Third, remove outer layers of leafy vegetables, peel vegetables when possible, and rinse produce before bringing it into the house to remove adhered contaminated soil and to prevent contaminated soil from being tracked into the home (Finster *et al.*, 2004). Fourth, soil can be amended with organic compost rich in phosphate and an alkaline pH can be maintained to reduce the mobility of lead in the soil and also to dilute the concentration (Sterrett *et al.*, 2000 in Finster *et al.*, 2004). Of course any compost added to the garden should not contain plant material previously grown in

contaminated soil. Using a mulch or weed taro can reduce the transfer of soil dust and prevent splash up on plants during watering (Finster *et al.*, 2004), additional keeping the soil moist will also control dust.

Gardening as an activity can also increase the amount of soil lead consumed by a receptor and simple things like rinsing garden equipment and exposed body parts after gardening, as well as avoiding eating, drinking, and smoking while gardening will reduce exposure.

Residents need to keep in mind that the contamination may come not only from their own property but from neighbours as well, especially if exterior renovations are being conducted (Shinn *et al.*, 2000; Gulson *et al.*, 1995, in Finster *et al.*, 2004). Monitoring soil and making improvements during these times is also important.

Soil and dust ingestion may contribute up to 14% of the total daily lead intake for toddlers and exposure to this pathway may also be reduced in several ways. Contaminated soil may be removed or replaced, enclosed under a permanent structure or surface, or covered with grass, mulch, or gravel (US EPA, 1998). The EMPACT Lead-Safe Yard Project developed by the US EPA provides a guideline for low-cost mitigative measures based on soil lead concentrations (Table 4.1).

According to this system soil lead levels of 981 ppm would justify enclosing or covering procedures but not costly soil removal. In addition there are behavioural changes that can

be made to decrease the ingestion of outdoor soil including frequent hand and toy washing.

Table 4.1. EMPACT Lead-Safe Yard Project Strategies (US EPA, 1998).

Soil Lead Level (ppm)	Recommended Action
> 5000 (very high)	Soil removal, semi-permanent barrier (gravel mulch), relocate garden
2000 - 5000 (high)	Seed grassy areas, cover with mulch woodchips, install stone paths for high traffic areas, relocate garden, relocate play area, pet area, and picnic area if possible or cover these areas with a wooden platform or woodchips
400 - 2000 (moderately high)	Seed or cover with mulch, install stone paths for high traffic areas, used raised bed garden with clean topsoil, install wooden platform or woodchip area for play and picnic areas
< 400 (urban background)	Requires no action

Indoor dust lead can be mitigated by wet mopping or vacuuming regularly, although removal of the source of dust lead (soil, paint) would be a more permanent measure (US EPA, 1998). For areas with lead based paint it is possible to use a heat gun and hand-scrape the paint from the surface. Chemical removal, replacement of components painted with lead based paint, enclosure of the surface with a rigid, solid material, or encapsulation of the effected area with durable coatings are other solutions (US, EPA 1998).

It is important that this information be disclosed to the residents of St. John's. If homeowners are made aware of the environmental lead levels in the city as well as the preventative measures that can be taken they will be better able to reduce lead exposure for their children.

Future Research

This assessment would be greatly enhanced by collecting more site-specific information, in particular house dust lead concentrations instead of loadings, plant uptake factors for garden produce, drinking water concentrations, and the bioavailability of lead in all exposure pathways. Using site-specific data decreases uncertainty and increases the reliability of hazard quotients and predicted blood lead levels.

In addition to the deterministic risk model used in this thesis it may be useful to conduct a probabilistic risk assessment to thoroughly understand how variations in the data itself affect the predicted health risk. In probabilistic risk methodology probability distributions are assigned to each of the parameters used to determine the estimated dose and a range of risks are then generated.

The potential health effects of other metals in soil and house dust in St. John's should also be examined using spatial analysis and risk assessment methods. In particular those metals with CCME guidelines should be evaluated; arsenic, barium, beryllium, cadmium, chromium, cobalt, copper, nickel, and zinc.

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Appendix A

Quality Control of Soil and Dust Samples

In order to assess the quality of the data it was important to look at the accuracy of laboratory and field methods, including the consistency of field and lab duplicate concentrations for both soil and dust samples.

Quality Control of Soil Samples

In the geochemistry laboratory every 20th soil sample was split and both parts analyzed to evaluate the reproducibility of the laboratory results. The laboratory duplicates were highly correlated indicating that laboratory methods were precise (n=75, r = 0.999, p = 0.000) (Figure A1.1).

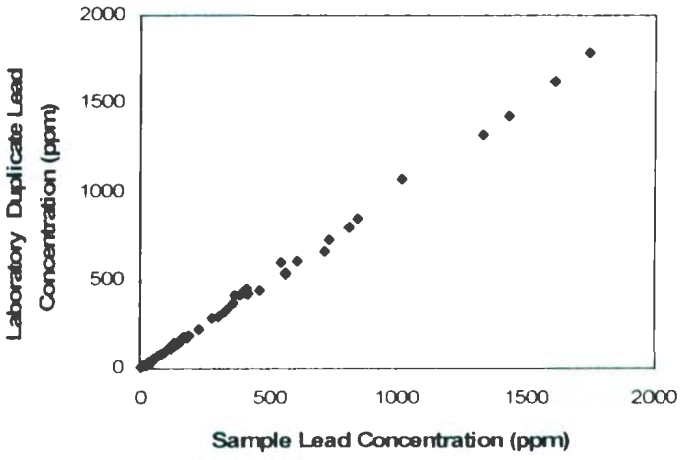

Figure A.1. The relationship between the lead concentrations of soil samples and the corresponding lab duplicates.

Field duplicates were collected beside approximately every 10th sample. Field duplicates were also highly correlated to the original sample, but to a lesser extent than laboratory duplicates (n = 104, r = 0.934, p = 0.000) (Figure A1.2). The test results indicate that

individual samples are largely representative of local soil conditions. Some samples with high lead concentrations did not correlate well with their duplicates. These samples came from along the foundation of houses and the large variability may be due to the presence or absence of paint chips in the duplicate sample that would dramatically affect the overall lead concentration. In order to better view the relationship between field samples and duplicates both were plotted on log-log graph paper.

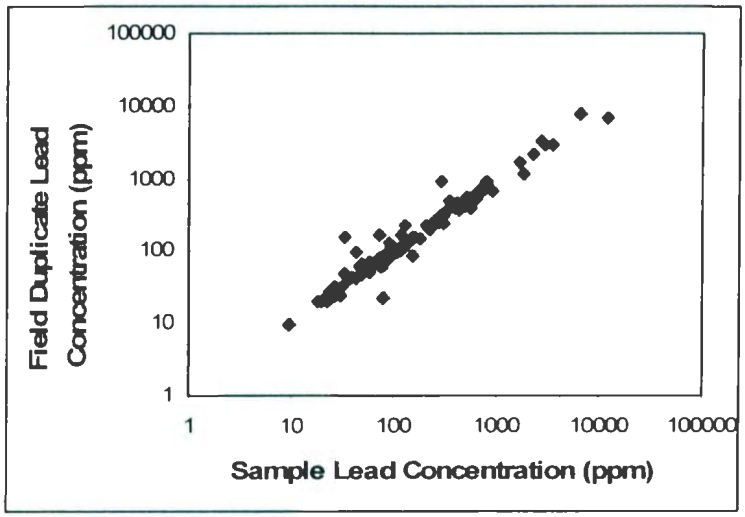

Figure A.2. The relationship between the lead concentrations of soil samples and the corresponding field duplicates.

Quality Control of Dust Wipe Samples

Laboratory blanks were made of de-ionized water from a NANOpure system. All samples were below detection limits indicating minimal machine contamination. Analysis of pure reagents indicated that during laboratory procedure a very small amount of lead was introduced to samples, roughly 4 ppm (Table A1.1). Un-used Ghost brand dust wipes

were also analyzed for lead content. In the laboratory, blank wipes contained on average roughly 5 ppb more lead than reagents alone. In the field blank wipes had comparable lead concentrations to laboratory blank wipes with the exception of one contaminated field blank that was discovered upon analysis (55.75 ppb).

Table A.1. Lead Concentrations in Laboratory and Field Blanks.

	n	*Mean (ppb)*	*SE Mean (ppb)*	*Minimum (ppb)*	*Median (ppb)*	*Maximum (ppb)*
Nanopure Water	*6*	*0.8*	*0.4*	*0.14*	*0.18*	*2.19*
Reagent Blanks	*8*	*3.6*	*0.5*	*1.97*	*3.90*	*5.66*
Lab Wipe Blanks	*4*	*8.7*	*1.2*	*5.37*	*9.44*	*10.57*
Field Wipe Blanks	*9*	*12.2*	*5.5*	*3.55*	*6.22*	*55.75*

Nineteen laboratory duplicates were run to see how variable the digestates were and how precise the ICP-MS analysis was. The duplicates were taken from all sample types including entrances, kitchens, window sills, and field blanks. Lab duplicates were highly correlated ($n = 19$, $r^2 = 1.000$, $p=0.000$) indicating high analytical precision (Figure A1.3).

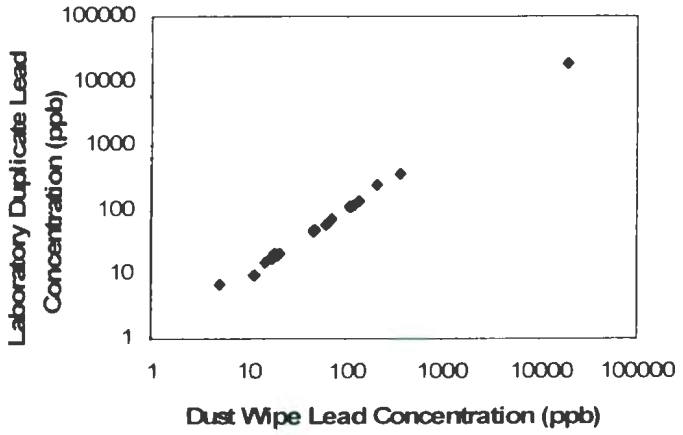

Figure A.3. The relationship between the lead concentrations of dust wipe samples and the corresponding lab duplicates.

Field duplicates were not so highly correlated (n=9, r^2 = 0.947, p=0.000), but still indicate that indoor dust lead values are reproducible in the field (Figure A1.4).

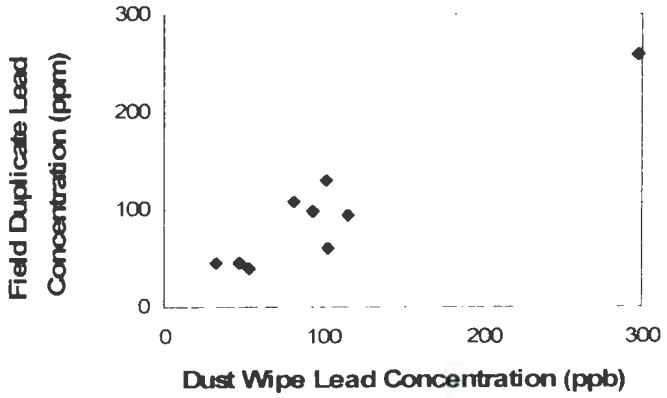

Figure A.4. The relationship between the lead concentrations of dust wipe samples and the corresponding field duplicates.

Appendix B

Detailed Description of the Methods for the Preliminary Quantitative Risk Assessments

Ingestion of Outdoor Soil and Indoor Dust

The total daily intake of lead from the ingestion of soil was calculated using Equation 1 (Health Canada, 2006) and data from Table B.1.

$$ING_{Soil\ /\ Dust}(mg\ /\ kg \cdot day) = \frac{C_{Soil\ /\ Dust} \times IR_{Soil\ /\ Dust} \times F_{Soil\ /\ Dust} \times RAF_{GI} \times D_1 \times D_2}{BW} \quad (1)$$

Table B.1. Model parameters used for the ingestion of soil and dust exposure pathways in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Soil Lead Concentration	C_{Soil}	mg/kg	Geometric mean soil concentration derived from all soil samples (ambient, dripline, roadside)	Health Canada, 2004a
Dust Lead Concentration	C_{Dust}	mg/kg	Two times the geometric mean soil lead concentration	Healy, 2007
Soil Ingestion Rate	IR_{Soil}	kg/day	45% of PQRA soil ingestion rates	Walker and Griffin, 1998; OME, 2002; US EPA, 2005
Dust Ingestion Rate	IR_{Dust}	kg/day	55% of PQRA soil ingestion rates	Walker and Griffin, 1998, OME, 2002, US EPA, 2005
Relative Gastrointestinal Absorption for Lead in Soil and Dust	RAF_{GI}	unitless	0.8	Health Canada, 2006
Exposure Duration	D_1	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004a
	D_2	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004a
Body Weight	BW	kg	Standard PQRA values (Table 3.1)	Richardson, 1997

In PQRA protocol the maximum concentration of soil contaminant is usually used in order to be conservative and this protocol was also followed in the risk assessment for Port Colborne (OME, 2002); however for a more likely estimate of risk researchers in Belledune and Trail have used the average concentration (Government of New Brunswick, 2006; Hilts et al., 2001). The St. John's soil lead survey was quite extensive; therefore it may be more reasonable to use the geometric mean soil lead concentration for the average risk estimate. Soil samples were collected from three different areas of the yard: beside the front road, beside the foundation, and in a part of the yard away from both the road and the foundation. The geometric mean soil concentration used for the risk assessment considered all three sample types as a receptor could be exposed to soil in any location of the yard.

The concentration of lead in house dust was not measured because of the cost of the equipment required. Instead Ghost Wipes were used to determine dust lead loadings, a metric that can still be compared to American health guidelines but cannot be included in a risk assessment for total daily intake of lead. However it is still important to include the ingestion of contaminated dust in the risk assessment as some researchers claim that this is a significant source of lead exposure for urban children (Lanphear *et al.*, 1996; Rasmussen *et al.*, 2001). In lieu of direct dust lead measurements, the concentration was estimated from the lead concentration in the soil. For both the conservative and average risk assessments indoor dust concentrations were calculated as twice the soil lead concentration. This was recommended by Health Canada based on studies done in British

Columbia, New Brunswick and Ottawa. At British Columbian Coast Guard light stations indoor dust was found to be roughly 1.6 times higher than soil lead concentrations (Healey, 2007). In St. John, New Brunswick the ratio ranged from 0.5:1 in entrances to 0.9:1 in living areas and 4:1 in bedrooms (CMHC, 1995), while Ottawa total lead in indoor dust was found to be 5.5 times higher than soil (Rasmussen, 2004). These ratios are higher than those traditionally used in risk assessment. For example, the IEUBK model uses a dust to soil ratio of 0.7 (US EPA, 2005), while a review of ratios in the literature by the Ontario Ministry of the Environment for the Port Colborne risk assessment found that this ratio might be too high (OME, 2002). Instead 0.4 was used based on research by the OME (1999), Hwang *et al.* (1997), Rutz *et al.* (1997), PTI (1994), and Calabrese and Stanek (1992) which all indicated that the ratio was between 0.2 and 0.5. The reason for the lower ratio might be that other sources may contribute to indoor dust lead, including deteriorated interior leaded paint.

Standard PQRA soil ingestion rates were used in the calculation (Table 3.1). Review of ingestion rates in the literature shows that PQRA values of 0.02 to 0.08 g/day are modest. Many studies have used rates over 0.100 g/day (Ferreira-Baptista and De Miguel, 2005; Nathanail *et al.*, 2005; Albering *et al.*, 1999; Boyd *et al.*, 1999). Even the US EPA IEUBK model uses default values above this level for children between 1 and 5 years of age (US EPA, 2005), but Calabrese and Stanek (1991; in Sheppard, 1995) argue that intake has been previously overestimated due to flawed methodologies and instead propose childhood ingestion rates between 0.009 and 0.040 g/day of soil. This range is

more in line with PQRA rates and those used in other studies (Sheppard, 1995; Meek and Huges, 1995; Hassanien and Horvath, 1999).

Only 45% of soil ingestion was attributed to outdoor soil. The other 55% was allocated to the ingestion of indoor dust. The PQRA model does not discuss the ingestion of household dust, but it is reasonable to assume that as receptors spend more time indoors they would be exposed to a higher percentage of soil/dust ingestion from inside their homes. This ratio was used in the Port Colborne risk assessment (OME, 2002) based on the research of Walker and Griffin for arsenic (1997) and is also used by the USEPA in the IEUBK blood lead prediction model (US EPA, 2005). Recent research suggests that dust ingestion rates might be double that of soil, 127 mg/day for dust versus 65 mg/day for soil (Calabrese *et al.*, 1997). Recently the authors reduced the soil intake for toddlers to 30-40 mg/day (Stanek and Calabrese, 2000; Stanek *et al.*, 2001). Because rates were only available for toddlers the other method was kept.

The relative gastrointestinal absorption of soil lead is typically assumed to be 100%, in PQRA methods. This assumes that the amount of lead absorbed from soil is equivalent to the amount of lead absorbed in the media used for the critical toxicological study from which the tolerable daily intake was derived, in this case breast milk (Equation 2).

$$\text{Re}\,lativeAbsorptionFactor_{soil} = \frac{AbsoluteAbsorption_{soil}}{AbsoluteAbsorption_{milk}} \qquad (2)$$

A review of the literature indicated that the bioavailability of lead in soil is typically less than the absolute bioavailability of milk (50%). Rural Ontario topsoil was found to have a relative bioavailability factor of 29% for soil particles between 100 and 400 µm, roughly 14% for those smaller than 100 µm and between 13 and 15% for smaller particle sizes (Rasmussen, 2004). A review of *in vivo* and *in vitro* studies undertaken for Health Canada showed a range of relative bioavailabilities from 0.5 to 87% with an average of 46% ± 27% and 51% ± 26% respectively (Health Canada, 2006). The report recommended that 80% be used as a reasonable maximum for screening level risk assessments as this was the 95th percentile of the reported values and this value will be used in both the conservative and average risk scenarios (Health Canada, 2006).

Estimates of the absolute bioavailability of lead in soil also differ between studies as as differences in methodologies as well as physical and chemical soil properties can dramatically alter the outcome. In Port Colborne soil lead was found to be on average 76% bioaccessible, with some samples as low as 61% and some as high as 90% (OME, 2002). In the supporting document for drinking water standards for lead, Health Canada reviewed the literature and suggested that children absorb 30% of ingested lead in soil and dust while adults may absorb as low as 10% (1992).The bioavailability suggested for children is similar to the default IEUBK bioavailability of 30% used in Trail, British Columbia (Hilts, 1995). The US EPA has extensively studied the bioavailability of lead in soil and they base their estimate on juvenile swine research done by Weiss *et al.* (1994) (US EPA, 1999).

Because soil and dust are often grouped together the bioavailabilities are often left the same (US EPA, 2005; OME, 2002); however, bioavailability is most likely higher in house dust because it has a higher organic content and a smaller particle size (Rasmussen, 2004). In Ottawa suburban house dust had a relative bioavailability of 60% compared to roughly 14% for rural topsoil (Rasmussen, 2004). In Port Pirie, Australia, the dissolution of indoor dust lead as a percentage of the total lead ranged from 26 to 46% (Oliver et al, 1999). While there is extensive research on the bioavailability of lead in soil, research is sparse for house dust making it hard to generalize. Because of this, bioavailabilities will be assumed to be the same for dust as for soil.

Soil ingestion was assumed to be constant, occurring daily throughout the year and was assumed to be independent of time spent outdoors as per PQRA protocol. Research conducted by Walker and Griffin (1997) confirms that soil and dust ingestion is not proportional to the number of hours spent outdoors or indoors and this principle has been used in other risk assessments (OME, 2002). It is unlikely that during the winter months when the ground is covered by snow that a receptor would be exposed to outdoor soil; however, the PQRA intake rates are averaged over the year with more soil potentially being ingested in the summer and less in the winter, but the average daily rate remains the same .

Outdoor Soil and Indoor Dust Inhalation

There exists a potential for soil with high lead concentrations to create outdoor dust which could be inhaled by receptors. Indoor dust may also be re-suspended into the air and subsequently inhaled. PQRA generally assumes the inhalation of contaminated soil and dust to be minimal compared to the ingestion of soil, dust, and water and the intake due to dermal contact with soil and dust; however it is additional pathway of exposure and should be considered, at least in the initial stages, to see if it has an impact on the overall lead intake. The daily intake of lead from the inhalation of outdoor soil particles and indoor dust was calculated using Equation 3 and data from Table B.2 (Health Canada, 2004a):

$$INH_{Soil\ Dust}(mg/kg \cdot day) = \frac{C_{Soil\ /\ Dust} \times P_{Air} \times IR_{Air} \times RAF_{Inh} \times D_1 \times D_2 \times D_3}{BW} \qquad (3)$$

Standard PQRA inhalation rates were used for soil and dust inhalation (Table 3.1). In general inhalation rates are dependent on activity and the level of exertion, and examination of the data source indicates that this was considered in the determination of standard values (Allan, 1995 in Richardson, 1997). Meek and Hughes (1995) used similar rates in their risk assessment of metals under the Canadian Environmental Protection Act, although the source of the intake rates was also Health Canada. Air intake rates used for children in the IEUBK model are slightly lower for children ages one to seven than those

recommended for PQRA, but still close enough to reinforce the validity of the standard rates (US EPA, 2005).

Table B.2. Model parameters used for the inhalation of soil and dust exposure pathways in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Soil Lead Concentration	C_{Soil}	mg/kg	Geometric mean soil concentration derived from all soil samples (ambient, dripline, roadside)	Health Canada, 2004a
Dust Lead Concentration	C_{Dust}	mg kg	Two times the geometric mean soil lead concentration	Healy, 2007
Particulate Concentration in air	P_{Air}	µg/m3	0.76	US EPA, 1992
Receptor Air Intake Rate	IR_{Air}	m^3/hour	Standard PQRA intake rates (Table 3.1)	Health Canada, 2004a
Relative Inhalation Absorption for Lead in Soil and Dust	RAF_{Inh}	unitless	1	Health Canada, 2004a
Soil Exposure Duration	D_1	hours/day	1.5	Modification of Richardson, 1996
	D_2	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004a
	D_3	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004a
Dust Exposure Duration	D_1	hours/day	22.5	Modification of Richardson, 1997
	D_2	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004a
	D_3	weeks/52 weeks	52 weeks per year exposed 52 weeks	Health Canada, 2004a
Body Weight	BW	kg	Standard PQRA values (Table 3.1)	Richardson, 1997

Unlike soil ingestion, inhalation exposures are dependent on the amount of time spent outdoors. PQRA methods suggest Canadian adults spend on average 1.5 hours per day outside, and make the assumption that if children are accompanied by adults they would also spend at least 1.5 hours per day outside. The US EPA (2004) references the summary of the National Human Activity Pattern Survey by Tsang and Klepeis (1996)

for time spent inside and outside the residence. The PQRA recommended 1.5 hours spent outdoors are similar to the 50th percentile values, but are much lower than the 95th percentile values (which are roughly 7 hours/day). The warmer climate in the United States might also have contributed to slightly higher outdoor times.

In New Jersey Yiin *et al.* (2000) found that in the spring and fall 66% of families allow their children to play outside for more than an hour and in the summer, from June to August, 48% do. The authors noted that playing outside from two to five hours was common in the summer. However from October to March the percentage of children playing outside for more than an hour dropped to 20%. Of course with St. John's having a cooler climate than New Jersey these percentages would probably be even lower. In fact in the preliminary dust lead study in St. John's all respondents reported not spending any appreciable time outdoors in the winter and the question was then dropped from the final questionnaire. During the warm months of the year (roughly June to September) residents of St. John's spent just a bit longer outside than the PQRA daily average of 1.5 hours, with the exception of teenagers who were found to spend less than half and hour per day outside (Table B.3). The number of people surveyed was quite small so the averages need to be interpreted with caution, however the results indicate that while individuals most likely spend more time outdoors during the summer this is countered by a decreased time outside during the winter making the annual average time spent outdoors of 1.5 hours per day reasonable.

Table B.3. Average Time Spent Outdoors by St. John's Residents during the Warm Months

Age Category	N	Time Spent Outside During the Warm Months (hours/day)
Infant	0	ND
Toddler	11	2.4
Child	10	2.9
Teen	12	0.4
Adult	60	1.8

ND – no data

Conversely since 1.5 hours per day were said to be spent outdoors a maximum of 22.5 hours could be spent inside the house. This could be particularly pertinent for infants, toddlers, parents who work at home, and retirees. It is more likely that school age children and most employed adults spend less time indoors at their residence because they spend time during the day at school or work.. Because 1.5 hours was selected as the annual average daily time spent outside for both the conservative and average risk scenarios, 22.5 hours will be selected for both scenarios in order to keep the methodology consistent.

Dermal Contact with Soil and Indoor Dust

Direct contact of contaminated soil or dust with skin can cause a very small transfer of lead into the blood stream. The PQRA and US EPA risk assessment protocols assume one dermal contact event per day; however PQRA does not account for exposure to contaminated indoor dust. The risk assessment conducted in Port Colborne (OME, 2002) incorporated dust exposure by weighting the one daily dermal event by the time spent

outdoors versus indoors. This method was used for the St. John's risk assessment. This method was used for the St. John's risk assessment. The intake of lead from dermal contact was calculated using Equation 4 and data from Table B.4 (Health Canada, 2004a).

$$DERM_{Soil\ \ Dust}(mg/kg \cdot day) = \frac{C_{Soil\ \ Dust} \times ASoil\ \ Dst \times F_{Out\ \ In} \times RAF_{Derm} \times EF \times D_1 \times D_2}{BW} \quad (4)$$

The US EPA (2004) recommends using the surface area for face, hands, forearms, lower legs, and feet for calculating a reasonable maximum dermal exposure which was justified by the warm climate experienced in the southern states, and because some studies suggest that exposure to chemicals can occur under clothing (Maddy *et al.*, 1983). In order to compensate for a more temperate climate the Port Colborne risk assessment used hands, arms, legs, and feet only in July and August, and reduced the exposed body parts to hands, arms, and legs in June and September, and then only hands and arms for the rest of the year (OME, 2002). Because of the mild climate in St. John's exposed skin was reduced to hands, forearms, lower legs, and feet from June to September and only hands for the rest of the year. Richardson (1997) gives the full surface area associated with arms and legs so the data were multiplied by 0.45 and 0.4 respectively to give the area only for forearms and lower legs (US EPA, 2004). See Table 3.1 for skin surface areas associated with these body parts. Surface areas for each body part were multiplied by the corresponding soil or dust loading factor (discussed below), weighted by the number of months exposed (four for the warm months and eight for the cool months) and then divided by 12 to give the annual average amount of soil or dust in contact with the skin.

Table B.4. Model parameters used for the dermal contact soil and dust exposure pathways in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Soil Lead Concentration	C_{Soil}	mg/kg	Geometric mean soil concentration derived from all soil samples (ambient, dripline, roadside)	Health Canada, 2004a
Dust Lead Concentration	C_{Dust}	mg/kg	Two times the geometric mean soil lead concentration	Healy, 2007
Annual Average Amount of Soil or Dust Dermal Contact	$A_{Soil\ Dust}$	kg	Surface areas for each body part were multiplied by the corresponding soil or dust loading factor, weighted by the number of months exposed (four for the warm months and eight for the cool months) and then divided by 12	Modification of OME, 2002
Exposed Skin Surface Area		cm2	Standard surface areas for hands, forearms, lower legs, and feet assumed to be exposed from June to September. Only hands assumed to be exposed for the rest of the year.	Health Canada, 2004a; and a modification of OME, 2002
Soil Loading Factor		mg/cm2	Geometric mean soil loadings for children in dry soil were used for infants, toddlers, and children for all body parts except feet for which no loading factor was available. Instead the soil loading for feet for daycare children with both outdoor and indoor exposures was used. Soil loading factors for gardeners were applied to teenagers and adults for all body parts.	OME, 2002;US EPA, 2004
Dust Loading Factor		mg/cm2	Geometric mean dust loading factors for children indoors were used for all age groups because of the lack of comparable data for teenagers and adults	OME, 2002;US EPA, 2004
Fraction of Time Spent Outdoors	F_{Out}	unitless	1.5 hours/ 24 hours (0.0625)	
Fraction of Time Spent Indoors	F_{In}	unitless	22.5 hours/ 24 hours (0.9375)	
Relative Lead Dermal Absorption Factor	RAF_{Derm}	unitless	0.006	Health Canada, 2004a
Exposure Frequency	EF	events/day	1	
Dermal Exposure	D_1	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004a
	D_2	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004a
Body Weight	BW	kg	Standard PQRA values (Table 3.1)	Richardson, 1997

Soil or dust loading is dependent on the body part, soil characteristics, and the type of activity being undertaken (US EPA, 2004). The standard PQRA soil to skin loading factor for hands (0.0001 g/cm^2*event) is an order of magnitude higher than for other body parts (0.00001 g/cm^2*event). The US EPA (2004) provides more detailed loading factors based on body part and activity which were also used in the Port Colborne risk assessment (OME, 2002). See Table B.5 below for values.

Table B.5. Soil loading factors for specific situations and body parts (US EPA, 2004).

Soil and Dust Exposure Situation	Soil Loading (mg/cm^2)			
	Hands	Arms	Legs	Feet
Indoor Children and Day Care				
Geometric Mean	0.020	0.007	0.005	0.017
95th Percentile	0.136	0.042	0.019	0.071
Children Dry Soil				
GM	0.097	0.014	0.042	ND
95th Percentile	0.632	0.281	0.608	ND
Daycare Children (indoors and outdoors)				
GM	0.093	0.023	0.023	0.049
95th Percentile	0.394	0.071	0.071	0.853
Gardeners				
GM	0.190	0.052	0.033	0.197
95th Percentile	0.958	0.240	0.166	3.473

US EPA soil loading factors for children in dry soil were used for infants, toddlers, and children for all body parts except feet for which no loading factor was available. Instead the soil loading for feet for daycare children with both outdoor and indoor exposures was used. Soil loading factors for gardeners were applied to teenagers and adults for all body parts. This may overestimate the risk as not all individuals have as much contact with soil as gardeners. US EPA dust loading factors for children indoors were used for all age groups because of the lack of comparable data for teenagers and adults. This may

overestimate the risk for these two age groups as they do not spend as much time on the floor and do not have as many hand to mouth habits. For both soil and dust loadings the conservative risk assessment used the 95th percentile, while geometric means were used for the average risk assessment.

Ingestion of Home Grown Produce

While the soil survey did not set out to quantify the number of houses with gardens, 31 homeowners requested that soil samples be taken from their garden out of a total of 305. If it is assumed that our sample is random and representative of the entire city then at the very least ten percent of the homes in St. John's have gardens where edible produce is grown. Therefore the ingestion of produce grown in soil with high lead concentrations is an important pathway to consider for a small percentage of the population.

In order to learn more about the gardening practices in St. John's a questionnaire was given to homeowners participating in the indoor dust survey which included questions on the type of produce grown and the frequency of consumption. Houses with gardens were preferentially chosen in order to collect the most information on home grown produce and thus 15 of the 32 houses tested for indoor dust lead also had gardens, a higher proportion than for the city as a whole. According to our sample of 15 gardens herbs, strawberries, raspberries and tomatoes were the most commonly grown while fewer households grew root vegetables (Table B.6). Since root vegetables have been shown to

have the highest transfer of lead from the soil to edible plant tissue (Finster *et al.*, 1994) fewer people in St. John's might be at risk than initially predicted.

Table B.6. The types and frequencies of produce grown in a small sample of St. John's households (n=15).

Root Vegetables	Number of Houses	Leafy Vegetables	Number of Houses	Other Vegetables	Number of Houses	Fruit	Number of Houses
Onions	4	Herbs	10	Tomatoes	7	Strawberries	9
Carrots	3	Lettuce	3	Beans	5	Raspberries	7
Potatoes	2	Kale	2	Peas	3	Rhubarb	3
				Brussel			
Garlic	2	Bok Choy	1	Sprouts	2	Blackberries	1
Beets	1	Rape Seed	1	Cucumber	2	Cherries	1
		Turnip				Black	
		Tops	1	Zucchini	1	Currents	1
		Spinach	1	Broccoli	1		
				Artichokes	1		
				Asparagus	1		
				Squash	1		

Participants also indicated that they consumed garden produce most commonly for one to three months of the year, with a third of the houses reporting that they froze or dried produce for later consumption through out the rest of the year (Table B.7). One house with a greenhouse recorded that garden produce was eaten for six months of the year. During these months participants ate on average four meals per week that contained garden produce (Table B.7). These frequencies were hard to quantify as often only small amounts of herbs or berries were consumed.

Table B.7. The number of months per year and meals per week during those months that households consume garden produce.

Months Per Year	Number of Houses	Meals Per Week	Number of Houses
1	4	<1	2
2	5	1	2
3	4	2	1
6	1	3	3
12	5	4	3
		7	4
		11	1

Richardson (1997) has calculated the average daily vegetable and fruit intake for Canadians (Table 3.1). It was assumed that this intake could be evenly distributed over three meals per day, and that according to Table B.7 four meals a week were assumed to consist completely of garden produce over a three month growing season.

Chemicals can enter plants via several pathways. They can be taken up directly by the foliage from the air or indirectly from the deposition of contaminated particulate matter. Contaminants can also be absorbed from the soil by the roots (Giordano *et al.*, 1994; Constantinou and Seigneur, 1993). For this risk assessment only root uptake will be considered because lead does not occur in gaseous form which eliminates direct uptake from the air, and because the deposition of contaminated particulate matter in St. John's is unknown. One of the simplest ways to estimate the uptake of lead in soil to home grown produce is to use a bioconcentration factor (BCF), a ratio of the concentration of lead measured in the plant and the concentration of lead measured in the soil (Giordano *et al.*, 1994). There are many factors which influence the uptake of lead including the type

of plant, crop density, soil pH and organic content, and environmental and climatic conditions (Giordano *et al.*, 1994).

Two sets of BCFs were used to show how influential the choice of factor is on lead intake. A review for Health Canada indicated that the best models for estimating heavy metal concentrations in backyard produce are the Multimedia, Multipathway, and Multireceptor Risk Assessment (3MRA) Modeling System (US EPA, 2003a) and the Guidance for Developing Ecological Soil Screening Levels (US EPA, 2003b) (Health Canada 2005a). One set of BCFs for root vegetables, other vegetables, and fruit were taken from the 3MRA model (US EPA, 2003a). The other set of BCFs were taken from Boyd *et al.* (1999) because the BCFs used in that study were highest in the root of plants and decreased with distance from the soil, a pattern seen in other studies (Finster *et al.*, 2004), but not the 3MRA model. BCFs that matched those fruits and vegetables found in St. John's gardens were taken from Boyd *et al.* (1999) and averaged for each of the three produce categories.

The final amount of lead in home grown produce ingested by receptors was calculated using Equations 5 and 6 and parameter values from Table B.8.

$$ING_{Produce} = ING_{Root} + ING_{Other} + ING_{Fruit} \quad (5)$$

Where:
$ING_{Produce}$ daily intake of ingested lead from home grown produce (mg/kg*day)
ING_{Root} daily intake of ingested lead from home grown root vegetables (mg/kg*day)
ING_{Other} = daily intake of ingested lead from home grown other vegetables (mg/kg*day)
ING_{Fruit} daily intake of ingested lead from home grown fruit (mg/kg*day)

$$ING_{Root\ \ Other\ \ Fruit} = \frac{C_{Soil} \times BCF_{Root\ \ Other\ \ Fruit} \times IR_{Root\ \ Other\ \ Fruit} \times AF_{GI} \times D_1 \times D_2}{BW} \quad (6)$$

Table B.8. Model parameters used for the ingestion of garden produce exposure pathway in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Soil Lead Concentration	C_{Soil}	mg/kg	Geometric mean soil concentration derived from all soil samples (ambient, dripline, roadside)	Health Canada, 2004a
Plant Lead Bioconcentration Factor	$BCF_{Root\ Other\ Fruit}$	unitless	0.033 for root vegetables, 0.010 for other vegetables, and 0.00004 for fruit	Boyd *et al.*, 1999
Garden Produce Consumption Rates	$IR_{Root\ Other\ Fruit}$	kg/day	Assumed that one entire meal of garden produce was consumed (one third of daily root, other vegetable, or fruit intake as reported by Richardson, 1997) for four meals a week over three months of the year. This amount was averaged over 365 to provide a daily intake.	Modification of Richardson, 1997 using data from St. John's questionnaire
Relative Gastrointestinal Absorption for Lead in Food	RAF_{GI}	unitless	1	Health Canada, 2004a
Exposure Duration	D_1	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004a
	D_2	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004a
Body Weight	BW	kg	Standard PQRA values (Table 3.1)	Richardson, 1997

Ingestion of Store-Bought Food

PQRA methods provide an equation for determining the amount of lead consumed in an average receptor's diet based on adding the amount of lead present in specific foods consumed; however information gathered from The Canadian Total Diet Study Report provided an overall total daily dietary lead intake based on the collection and preparation

of supermarket food from eight Canadian cities (Health Canada, 2005b). Intakes for sixteen age groups were presented in the report and had to be combined according to the five PQRA age categories using age-weighted averages in order to be used in both the conservative and best estimate risk scenarios (Table B.9).

Table B.9. Total dietary lead intake for specific age groups of Canadians modified from the Canadian Total Diet Study Report (Health Canada, 2005b)

Age (years)	Dietary Lead Intake (mg/kg*day)
0 - <0.5	0.000479
0.5 - <5	0.000487
5 - <12	0.000368
12 - <20	0.000224
20+	0.000187

Ingestion of Drinking Water

The ingestion of lead in drinking water was calculated using the following equation and data from Table B.10 (Health Canada, 2004a):

$$ING_{Water}(mg/kg \cdot day) = \frac{C_{Water} \times IR_{Water} \times RAF_{oral} \times D_1 \times D_2}{BW} \quad (7)$$

The drinking water supply for St. John's must meet the maximum acceptable concentration (MAC) of lead in Canadian drinking water, 0.010 mg/L (Health Canada, 1992). It was reasonable to assume that this was the maximum concentration of lead in the drinking water leaving the plant. However the concentration may change as the water

flows through municipal and residential plumbing due to the potential existence of lead pipes and leaded soldering.

Table B.10. Model parameters used for the ingestion of drinking water exposure pathway in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Drinking Water Lead Concentration	C_{Water}	mg/l	0.0048	Graham, 1988; Health Canada, 1992
Water Ingestion Rate	IR_{Water}	L/day	Standard PQRA values	Richardson, 1997
Relative Gastrointestinal Absorption for Lead in Drinking Water	RAF_{GI}		1	Health Canada, 2004a; US EPA, 2005
Exposure Duration	D_1	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004a
	D_2	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004a
Body Weight	BW	kg	Standard PQRA values (Table 3.1)	Richardson, 1997

Almost all of the homeowners surveyed during indoor dust collection did not have lead pipes, although several homeowners were not sure and could not recall when the plumbing was last replaced. Lead service connecters were common in well-built homes before 1920, and 50 percent lead solder was used until 1990 when the National Plumbing Code of Canada drastically reduced the lead content (Canada Mortgage and Housing Corporation, 2007; Health Canada, 2004b). It is still possible for homes built before 1950 to contain leaded distribution lines and service connections if they have not undergone renovations (Health Canada, 2004b). A lot of research on leaded drinking water has been done in the city of Edinburgh, Scotland. In this city lead piping was used until 1955 and

lead solder was not banned until 1982 (Macintyre *et al.*, 1998), a situation similar to Canada. In an initial study done in an area of Edinburgh with a high proportion of houses with lead plumbing and a water supply that readily dissolves lead the mean water lead concentration was 0.037 mg/L (Raab *et al.*, 1987). This concentration was assumed for houses built before 1948 and the MAC of 0.010 mg/L was assumed for houses built after 1948 for the conservative risk assessment.

Several studies of drinking water quality have been conducted in Canada. One such study was done by Graham (1988) on 40 houses in Ontario. The lead concentrations of the water samples ranged from 0.0011 to 0.0307 mg/L and had a median value of 0.0048 mg/L. Health Canada (1992) suggests that this median lead concentration is the most realistic estimate from all water quality studies reviewed, and it was used for all houses in the average risk scenario.

Dermal Contact with Water during Bathing

The following equation was used to estimate the daily intake of lead that passes through the skin while bathing (US EPA, 2004a; US EPA, 1997):

$$DERM_{water}(mg \cdot kg \cdot day) \quad \frac{C_{water} \times CF \times PC \times SA_{total} \times EF \times D_1 \times D_2}{BW} \qquad (8)$$

Table B.11. Model parameters used for the dermal contact with bathing water exposure pathway in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Drinking Water Lead Concentration	C_{Water}	mg L	0.0048	Graham, 1988; Health Canada, 1992
Volumetric Conversion of Water	CF	L/cm^3	0.001	
Dermal Permeability Coefficient for Lead	PC	cm/hour	0.0001	US EPA, 2004
Surface Area	SA_{Total}	cm2	Total body surface area (Table 3.1)	Richardson, 1997
Length of Bathing Time		hours/day	0.33 (50^{th} percentile)	US EPA, 1997
Dermal Exposure	D_1	days/7days	7 days per week exposed/ 7 days	Health Canada, 2004a
	D_2	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada, 2004a
Body Weight	BW	kg	Standard PQRA values (Table 3.1)	Richardson, 1997

Inorganic compounds transfer very slowly through the skin. The US EPA lists a dermal permeability coefficient of 0.00005 cm/hr for $Pb(CH_3CO_2)_2$, 0.00013 for $Pb(NO_3)_2$, and recommends 0.0001 in general for lead (US EPA, 2004). Since the dominant form of lead that potentially exists in the water supply was not known the general coefficient was used for both risk scenarios.

Inhalation of Urban Air

Equation 11 was used to calculate the daily intake of lead from inhaled outdoor and indoor urban air (Health Canada, 2004a).

$$INH_{AirOut\ \ In} = \frac{C_{AirOut\ \ In} \times IR_{Air} \times RAF_{Inh} \times D \times D_2 \times D_3}{BW} \quad (9)$$

Table B.12. Model parameters used for the inhalation of outdoor and indoor air exposure pathway in the average risk scenario.

Parameter	Symbol	Units	Best Estimate	Source
Outdoor Air Lead Concentration	C_{AirOut}	µg/m3	0.06	Health Canada, 1992
Indoor Air Lead Concentration	C_{AirIn}	µg/m3	0.045 (75% of the concentration of outdoor air)	OME, 2002 (based on Roberts *et al.*, 1974)
Receptor Air Intake Rate	IR_{Air}	m^3/hour	Standard PQRA intake rates (Table 3.1)	Health Canada, 2004a
Relative Inhalation Absorption for Lead in Air	AF_{Inh}		0.64 (absolute AF of 32%)	Health Canada, 2004a; US EPA, 2005
Outdoor Air Exposure Duration	D_1	hours/day	1.5	Modification of Richardson, 1996
	D_2	days/7days	7 days per week exposed/ 7 days	Health Canada 2004a
	D_3	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada 2004a
Indoor Air Exposure Duration	D_1	hours/day	22.5	Modification of Richardson, 1997
	D_2	days/7days	7 days per week exposed/ 7 days	Health Canada 2004a
	D_3	weeks/52 weeks	52 weeks per year exposed/ 52 weeks	Health Canada 2004a
Body Weight	BW	kg	Standard PQRA values (Table 3.1)	Richardson, 1997

According to data from the National Air Pollution Surveillance stations the annual geometric mean of lead in Canadian air has decreased from 0.74 µg/m^3 in 1973 to less then the detection limit of 0.1 µg/m^3 in 1991 (Health Canada, 1992). Major cities have measurable concentrations, but they still do not exceed 0.1 µg/m^3. Health Canada (1992) has estimated the concentration of lead in the air to be 0.06 µg/m^3 by assuming the

concentration to be one third of the detection limit and by multiplying the concentration by two to account for the difference between the measured concentration taken on rooftops and the concentration experienced at street level. This concentration was used for both risk scenarios for outdoor air, while indoor air was set for 0.045 $\mu g/m^3$ which is 75% of the concentration of outdoor air (Roberts *et al.*, 1974 in OME, 2002).

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Appendix C

Results for the Worst-Case Preliminary Quantitative Risk Assessment

Worst-Case PQRA Exposure Assessment and Risk Characterization

According to the worst-case risk assessment, all residents of St. John's who consume garden produce may have an unacceptable risk of health effects (HQ>1), except for adults living on properties developed after 1993 (soil lead concentration <117 ppm) according to the 3MRA model and adults living on properties developed after 1977 (soil lead concentration < 149 ppm) according to the model based on Boyd *et al.* (1999) (Table C.1). Hazard quotients are close to unity for post-1993 properties, but reach up to 126 times the acceptable daily lead intake for infants in pre-1926 housing.

When the consumption of garden produce was not considered in the worst-case risk assessment hazard quotients dropped, and only exceeded unity for infants and toddlers living on properties developed before 1961 (soil lead concentration >1011 ppm) and children on properties developed before 1926 (soil lead concentration >3722 ppm (Table C.2). Hazard quotients ranged from 0.13 for adults living on new properties to 6.44 for toddlers on the oldest properties. Unlike the scenario incorporating the ingestion of garden produce, risk was higher for toddlers than infants.

Table C.1. Hazard quotients for the worst-case risk assessment when receptors are consuming garden produce.

	0 - < 5 months		5 months - < 5 years		5 years - <12 years		12 years - <20 years		20+ years	
Age of Property	Hazard Quotient (3MRA BCFs)	Hazard Quotient (Boyd *et al.* BCFs)	Hazard Quotient (3MRA BCFs)	Hazard Quotient (Boyd *et al.* BCFs)	Hazard Quotient (3MRA BCFs)	Hazard Quotient (Boyd *et al.* BCFs)	Hazard Quotient (3MRA BCFs)	Hazard Quotient (Boyd *et al.* BCFs)	Hazard Quotient (3MRA BCFs)	Hazard Quotient (Boyd *et al.* BCFs)
Pre-1926	**125.59**	**84.94**	**103.21**	**54.79**	**58.97**	**38.02**	**33.41**	**28.73**	**26.93**	21.31
1926 - 1948	**91.62**	**62.01**	**75.32**	**40.05**	**43.05**	**27.29**	**24.40**	**20.99**	**19.69**	15.60
1949 - 1960	**34.20**	**23.16**	**28.13**	**14.98**	**16.09**	**10.40**	**9.12**	**7.85**	**7.35**	5.83
1961 - 1976	**14.36**	**9.77**	**11.84**	**6.37**	**6.79**	**4.43**	**3.86**	**3.33**	**3.12**	2.49
1977 - 1992	**5.23**	**3.61**	**4.34**	**2.41**	**2.51**	**1.68**	**1.44**	**1.25**	**1.18**	0.95
1993 - Present	**4.16**	**2.88**	**3.46**	**1.94**	**2.01**	**1.36**	**1.15**	**1.00**	0.95	0.77

Note: all bolded HQs are greater than 1.00 and therefore represent elevated health risk.

Table C.2. Hazard quotients for the worst-case risk assessment when receptors are not consuming garden produce.

		0 - < 6 months	5 months - < 5 years	5 years - <12 years	12 years - <20 years	20+ years
Age of Property	95th Percentile Soil Lead Concentration (ppm)	Hazard Quotient	Hazard Quotient	Hazard Quotient	Hazard Quotient	Hazard Quotient
Pre-1926	3722	**3.55**	**6.44**	**1.14**	0.69	0.66
1926 - 1948	2711	**2.73**	**4.83**	0.93	0.56	0.55
1949 - 1960	1011	**1.06**	**1.85**	0.39	0.23	0.22
1961 - 1976	420	0.58	0.92	0.26	0.16	0.16
1977 - 1992	149	0.36	0.48	0.21	0.13	0.13
1993 - Present	117	0.33	0.43	0.20	0.13	0.13

Note: all bolded HQs are greater than 1.00 and therefore represent elevated health risk.

The percentage of lead intake from parameters associated with soil lead (ingestion of soil, dust, and garden produce) is highest on the oldest properties and decreases on progressively newer properties because the associated soil lead concentration also diminishes (Table C.3). It is important to note that the upper percentages for soil and dust ingestion represent toddlers who consume more soil and dust than other receptors. All other parameters remain constant and thus their relative contribution to the total daily lead intake increases as property age decreases because soil related parameters are contributing less. Dermal contact and inhalation pathways contribute very little to the total daily lead intake and will not be discussed further (Table C.3).

Garden produce consumption is the largest contributor to daily lead intake on older properties with high soil lead concentrations; however the 3MRA model predicts that fruit is the largest contributor to daily lead intake, followed by root vegetables, and then other vegetables, while the model based on Boyd *et al.* (1999) predicts that root vegetables will contribute the most, then other vegetables, and finally fruit will contribute very little (Table C.3). The ingestion of indoor dust is the next highest contributor for both models, followed by the ingestion of soil, water, and supermarket food.

On newer properties with lower soil lead concentrations the influence of soil related parameters on the daily lead intake drops. Garden produce still contributes the most, but the ingestion of supermarket food and water have a similar impact as the ingestion of dust and soil (Table C.3).

Table C.3. The contribution of each worst-case risk parameter to the percent of total daily lead intake. The percentage range represents the array of values for different ages of receptors.

	Including Garden Produce				Not Including Garden Produce	
Risk Parameter	Pre-1926 Housing (3MRA)	Pre-1926 Housing (Boyd *et al.*)	Post 1993 Housing (3MRA)	Post 1993 Housing (Boyd *et al.*)	Pre-1926 Housing	Post 1993 Housing
Ingestion of Fruit	81 - 75%	<1%	75- 69%	<1%	-	-
Ingestion of Root Vegetables	13 - 7%	84 – 73%	12 - 7%	75 – 65%	-	-
Ingestion of Other Vegetables	11 - 6%	20 – 14%	10 - 5%	19 – 12%	-	-
Ingestion of Dust	4 - 1%	8 - 1%	4 - 1%	7 - 1%	69 - 39%	32 - 6%
Ingestion of Soil	2 - <1%	3 - <1%	2 - <1%	2 - <1%	22 – 13%	11 - 2%
Ingestion of Water	<1%	1 - <1%	6 - 2%	8 - 4%	33 - 6%	47 - 24%
Ingestion of Supermarket Food	<1%	1%	5 - 3%	8 - 4%	9 - 2%	51 - 32%
Dermal Contact with Dust	<1%	<1%	<1%	<1%	5 - 1%	1 - <1%
Dermal Contact with Soil	<0.1%	<0.1%	<0.1%	<0.1%	2 - <1%	<1%
Inhalation of Dust	<0.01%	<0.01%	<0.01%	<0.01%	<0.1%	<0.1%
Inhalation of Outdoor Air	<0.001%	<0.01%	<0.1%	<0.1%	<0.1%	<1%
Dermal Contact with Water	<0.001%	<0.001%	<0.01%	<0.01%	<0.1%	<0.1%
Inhalation of Indoor Air	<0.0001%	<0.0001%	<0.01%	<0.01%	<0.1%	3 - 1%
Inhalation of Soil	<0.0001%	<0.001%	<0.0001%	<0.001%	<0.01%	<0.001%

– parameter not included for this risk assessment

When garden produce is not eaten the ingestion of indoor dust is the most important pathway of lead exposure, followed by the ingestion of water, soil, and supermarket food, for older properties with high soil lead concentrations (Table C.3). Dermal exposure to dust and soil also has a small effect on lead intake. On newer properties supermarket food and drinking water contribute the most to daily lead intake, while dust and soil ingestion contribute less (Table C.3). The inhalation of indoor air and to a lesser extent the dermal contact with dust also have a small impact on the daily lead intake.

When the estimated daily intake of lead is averaged over a lifetime, hazard quotients are below unity for all individuals who do not eat garden produce, except those who live on pre-1926 properties (Table C.4). Hazard quotients also exceed unity for those receptors who consume garden produce and live on pre-1976 properties according to the 3MRA produce model and for those receptors on pre-1961 properties according to the model based on Boyd *et al.* (1999) (Table C.4).

Table C.4. The cumulative daily intake of lead averaged over a lifetime of exposure for residents living on properties of different ages for the worst-case risk scenario.

	Consuming Garden Produce for Three Months of the Year		Not Consuming Garden Produce
Age of Property	Hazard Quotient (3MRA)	Hazard Quotient (Boyd *et. al.*)	Hazard Quotient
Pre-1926	**9.98**	**7.43**	1.10
1926 - 1948	**7.35**	**5.49**	0.88
1949 - 1960	**2.76**	**2.07**	0.35
1961 - 1976	**1.22**	0.94	0.22
1977 - 1992	0.52	0.41	0.16
1993 - Present	0.43	0.35	0.15

Note: all bolded HQs are greater than 1.00 and therefore represent elevated health risk.

Neighbourhood Housing Distribution in St. John's in Relation to Health Risk

Knowing that the risk of increased blood lead concentration is related to the soil lead concentrations and thus indirectly to the age of property, a map of housing age for neighbourhoods in St. John's may be a useful tool for evaluating risk.

Figure C.1 shows the percent of the housing stock for each neighbourhood that was built before 1946. High values represent an increased lifetime CDI risk even if no garden produce is consumed. They also represent an increased EDI risk for children who do not consumer garden produce. Once again this map may overestimate the spatial extent of the previously described risks as they refer to only pre-1926 properties which could not be specifically mapped due to data limitations.

Figure C.2 shows the percentage of houses built before 1960 in individual neighbourhoods in St. John's. It may be associated with an increase in lifetime CDI risk for those individuals consuming garden produce (Boyd et al.) as well as an increase in EDI risk for infants and toddlers who do not consume garden produce.

Figure C.3 shows the percentage of houses built before 1980. In the worst-case risk assessment these areas illustrate an increased CDI risk for receptors consuming garden produce (3MRA).

Figure C.4 illustrates that percentage of houses built before 1990 which may be associated with and increased EDI risk for adults consuming garden produce (3MRA and Boyd et al.).

All houses may produce an increased EDI health risk for infants, toddlers, children, and teenagers (3MRA and Boyd *et al.*) and may be associated with elevated blood lead concentrations in children under seven who consume garden produce according to both 3MRA and Boyd *et al.* data. This was not mapped as it includes the entire city.

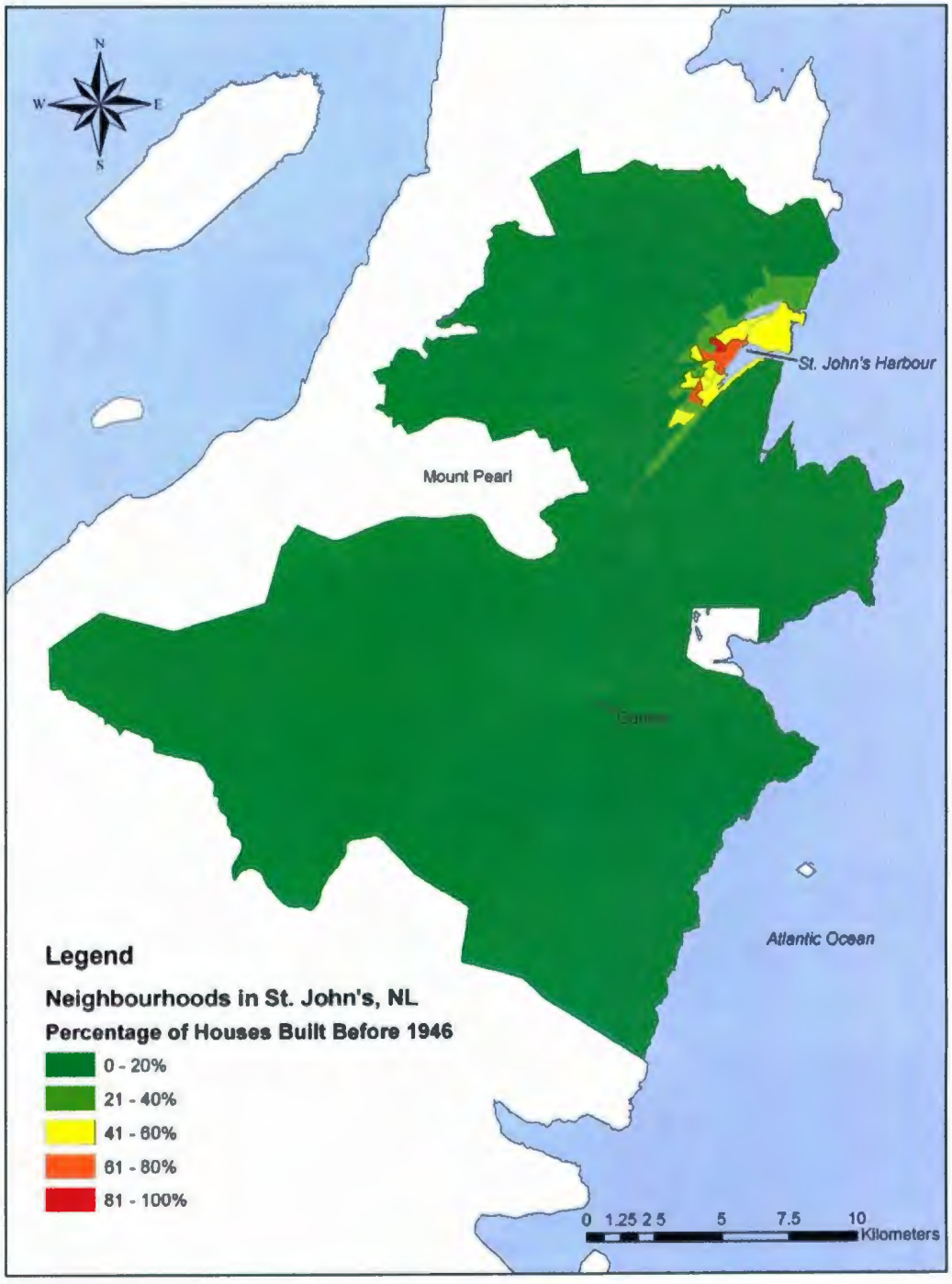

Figure C.1. The percentage of neighbourhood houses built before 1946 (Community Accounts, 2007).

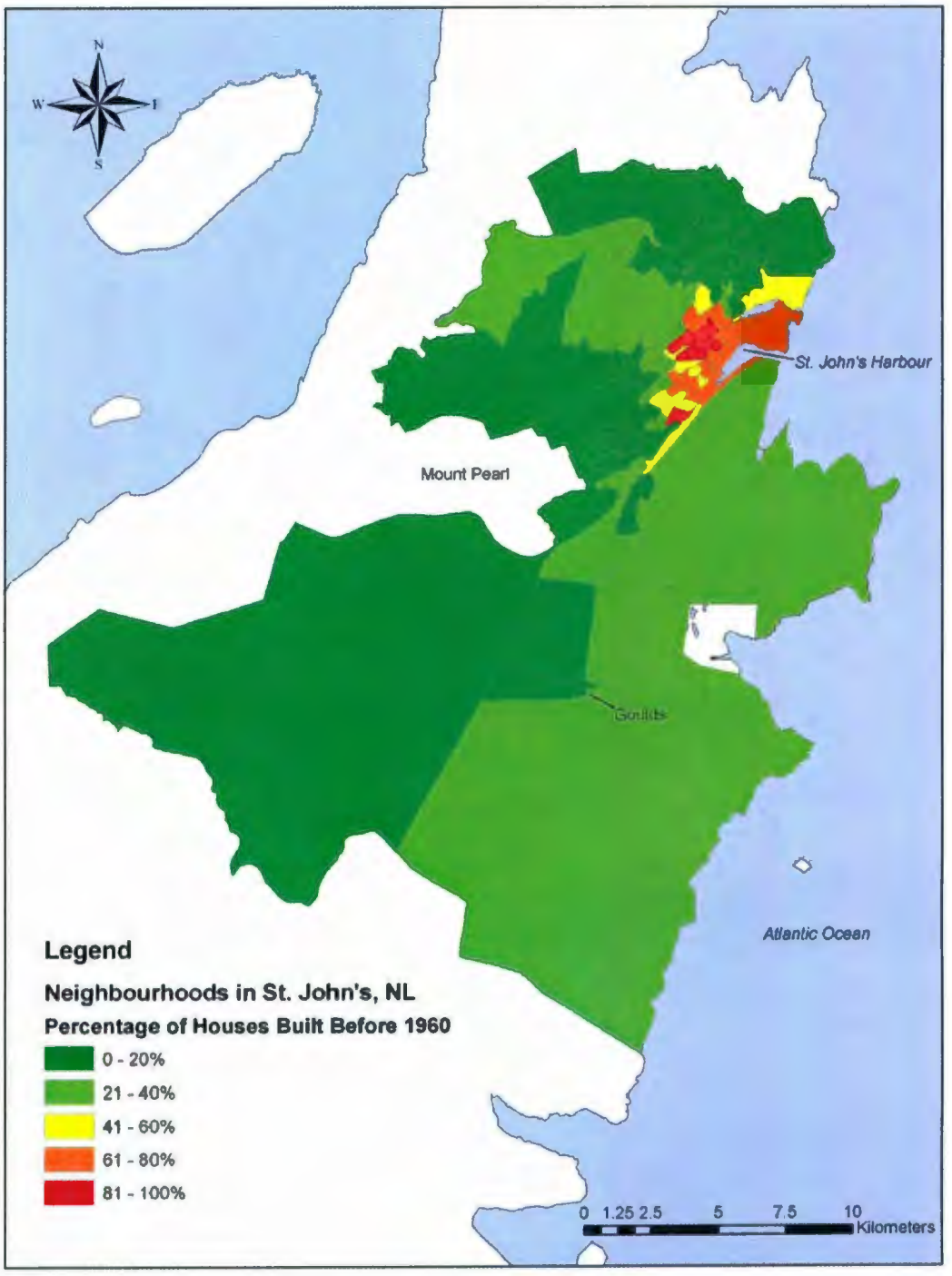

Figure C.2. The percentage of neighbourhood houses built before 1960 (Community Accounts, 2007).

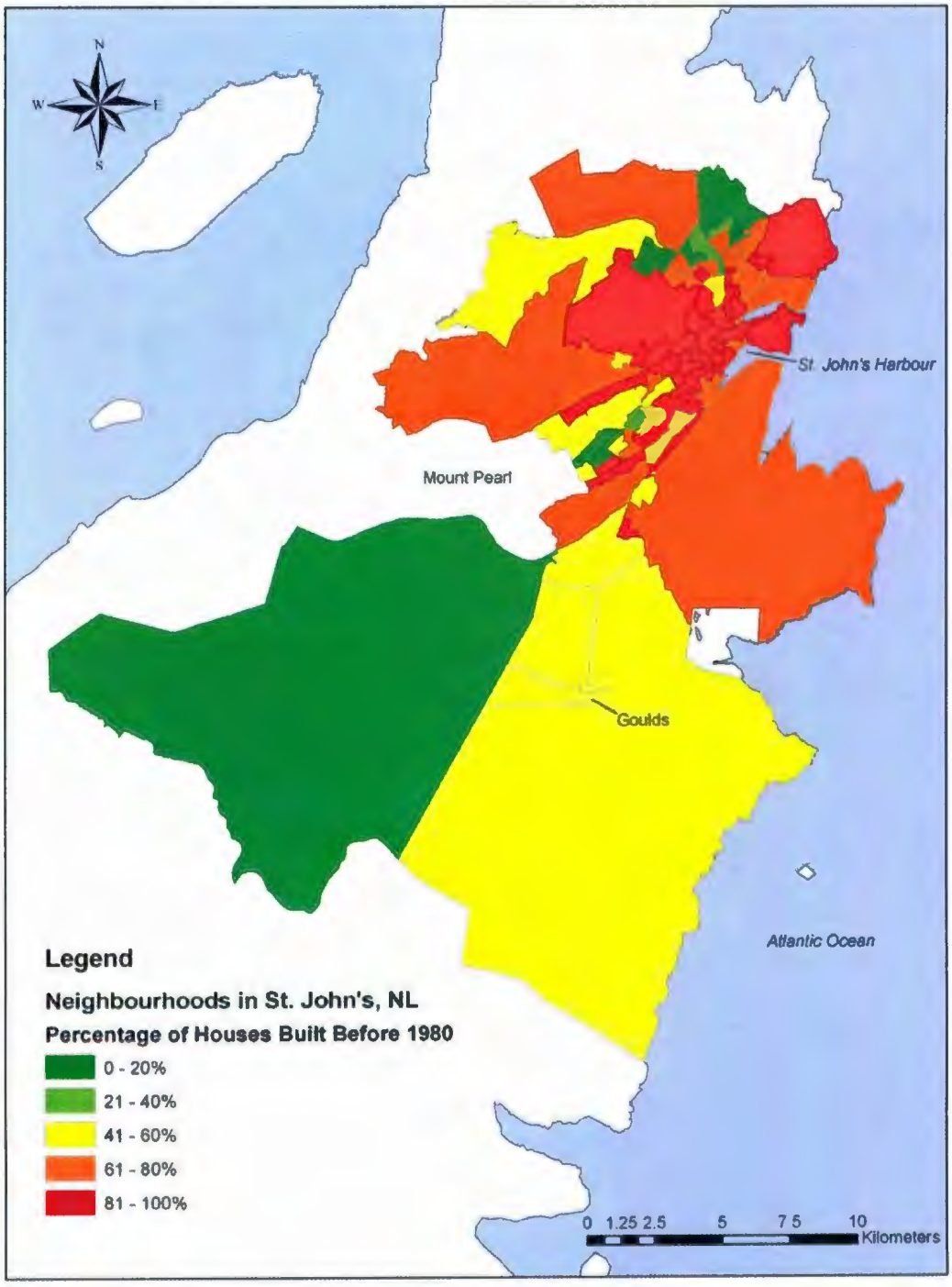

Figure C.3. The percentage of neighbourhood houses built before 1980 (Community Accounts, 2007).

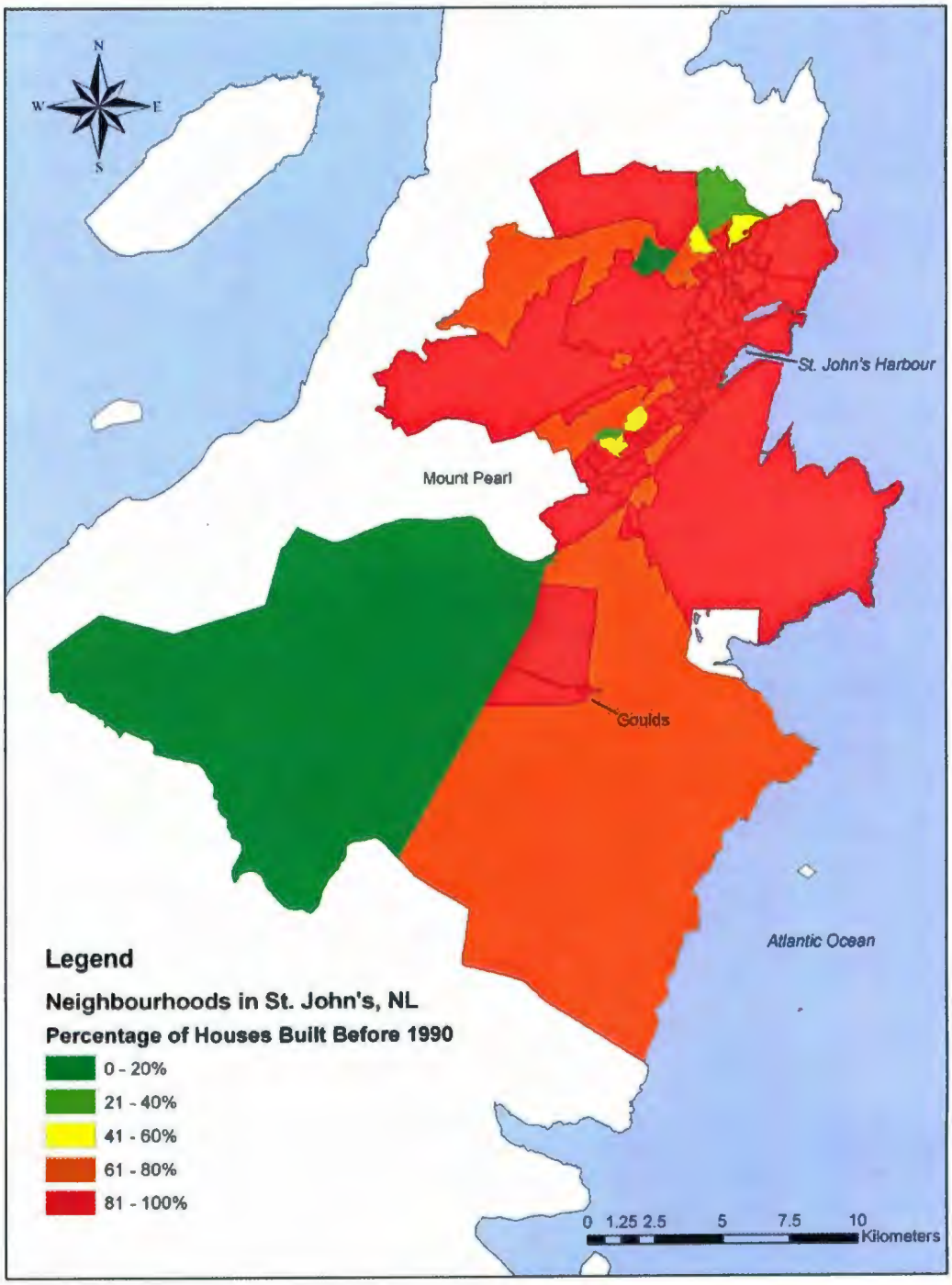

Figure C.4. The percentage of neighbourhood houses built before 1990 (Community Accounts, 2007).

Appendix D

Uncertainty Analysis for the Preliminary Quantitative Risk Assessment

Table D.1. The effect of parameter assumptions on the predicted PQRA risk estimates.

Parameter	Assumption	Under Estimates Risk	No Change in Risk	Over Estimates Risk	Unknown Effect on Risk
Soil Ingestion					
Soil Concentration	The 95th or 50th percentile soil lead concentrations for all samples taken on properties of a certain age was used in the conservative and average risk assessment respectively. It is most likely that a constant exposure to the 95th percentile of soil lead is an overestimate of risk, but it does provide an upper bound to compare to the risk calculated by the more realistic 50th percentile concentration.		50th Percentile *	95th Percentile *	
Soil Concentration	Soil lead concentration was derived from an average of all three types of samples, those taken by roads, dripline samples taken by the foundation of houses, and ambient samples taken in open areas. In general dripline and roadside samples had the highest concentrations but occurred over smaller areas of the property; however they were weighted the same as ambient samples which generally had lower concentrations and were representative of a larger area of the property. This may have overestimated the overall soil lead concentration.			*	
Soil Ingestion Rate	Soil ingestion rates may be considered low by some standards, but they are perhaps more realistic and are also endorsed by the Federal PQRA guidelines.	*			
Soil Ingestion Rate	The outdoor soil ingestion rate was estimated as only 45% of total soil ingestion in order to account for indoor dust ingestion. Splitting soil ingestion into outdoor and indoor components may underestimate risk; however it is reasonable to assume that individuals are less exposed to outdoor soil than indoor dust because they spend less time outside. This percentage was generated in a study on arsenic exposure (Walker and Griffin, 1998).	*			

Parameter	Assumption	Under Estimates Risk	No Change in Risk	Over Estimates Risk	Unknown Effect on Risk
Absorption Factor	80% relative absorption was used both the conservative and average assessments. This is based on a review of the literature done for Health Canada which stated 80% would be a reasonable maximum, thus risk may be slightly overestimated. However site specific physical and chemical conditions (pH, particle size etc.) as well as the test used to determine bioavailability all influence the outcome. This uncertainty could be reduced by testing the bioavailability of soil in St. John's.			*	
Exposure Duration	Soil ingestion was assumed to be constant over the entire year, but because the rates used were an annual average a balance was achieved between low winter and high summer intakes.		*		
Dust Ingestion					
Dust Concentration	Estimated as 200% of the concentration in outdoor soil. This relationship is controversial as traditionally dust has been estimated to be lower than soil concentration. Recent studies in Canada have found that in fact dust concentrations may be higher than soil, especially in communities where there is no industrial source of lead. 200% may be a slightly conservative estimate. This uncertainty could be eliminated by taking environmental samples from which dust lead concentrations could be determined.			*	
Dust Concentration	Because dust concentrations were derived from soil concentrations it is likely that the 95th percentile is an overestimate of risk, while the 50th percentile is more representative of actual risk.		50th Percentile *	95th Percentile *	
Dust Ingestion Rate	As previously mentioned the total soil ingestion rates may be considered low by some therefore the total dust intake may also be slightly underestimated; however this rate is endorsed by Federal PQRA guidelines.	*			

Parameter	Assumption	Under Estimates Risk	No Change in Risk	Over Estimates Risk	Unknown Effect on Risk
Dust Ingestion Rate	The indoor dust ingestion rate was estimated as 55% of total soil ingestion. Splitting soil ingestion into outdoor and indoor components may underestimate risk; however it is reasonable to assume that individuals are more exposed to indoor dust because they spend more time inside. This percentage was generated in a study on arsenic exposure (Walker and Griffin, 1998).	*			
Absorption Factor	Assumed to be the same as soil, although there is evidence that it might be higher because of the higher organic content and smaller particle size of house dust (Rasmussen, 2004).	*			
Exposure Duration	Like soil, dust ingestion was assumed to be constant over the entire year, but because the rates used were an annual average a balance was achieved between low winter and high summer intakes.		*		
Soil and Dust Inhalation					
Soil and Dust Concentration in Air	Soil/Dust concentration was multiplied by the standard particulate concentration in the air as suggested by PQRA methods and the US EPA in order to calculate the soil/dust concentration in the air. Unsure of how the standard particulate concentration was generated by the US EPA.				*
Inhalation Rate	Standard inhalation rates were used.		*		
Absorption Factor	A relative lung absorption factor of 100% was used for borh the conservative and average risk assessments based on PQRA defaults.			*	
Exposure Duration	1.5 hours spend outdoors per day (soil) and 22.5 hours spend indoors (dust). Probably a good representation of the mean for residents of St. John's based on questionnaire data and the standard PQRA values. However it may be an overestimate for school age children and adults who work outside the home as the time spent indoors at home would be much less (14.5).		*	*	

Parameter	Assumption	Under Estimates Risk	No Change in Risk	Over Estimates Risk	Unknown Effect on Risk
Soil and Dust Dermal Exposure					
Average Annual Soil Contact (Surface Area)	Included forearms, lower legs, and feet for June to September and then just hands for the rest of the year, while Port Colborne included the surface area of the entire arm and leg during the warm months and also included the entire arm for the rest of the year. Surface area might have been underestimated.	*			
Average Annual Soil Contact (Soil Loading Factor)	For the most part specific soil and dust loading factors were used for specific age groups; however for teenagers and adults factors were taken from data for gardeners and dust loading factors were taken from children because of lack of age specific data. This overestimated the risk for teenagers and adults.			*	
Average Annual Soil Contact (Soil Loading Factor)	The Conservative risk assessment used the 95th percentile loading values while the average risk assessment used the geometric mean values.		Geometric Mean *	95th Percentile *	
Dermal Absorption Factor	The standard PQRA dermal absorption factor for lead was used.		*		
Exposure Duration	One dermal event per day, year round, was assumed, which was weighted by the amount of time spent outside for soil contact and by the amount of time spent inside for dust.		*		
Ingestion of Store-Bought Food					
Daily Lead Intake	Daily lead intake from supermarket food taken from the Total Diet Study Report (Health Canada, 2005). This study was conducted in eight Canadian cities but did not include St. John's. It is possible (although unlikely) that the intake from these cities does not correspond with that of St. John's causing an unknown effect on risk.				*

Parameter	Assumption	Under Estimates Risk	No Change in Risk	Over Estimates Risk	Unknown Effect on Risk
Absorption Factor	100% relative absorption was assumed for both the conservative and average risk assessments based on PQRA defaults..			*	
Ingestion of Drinking Water					
Concentration in Water	Lead in St. John's drinking water was not measured so concentrations were estimate from the literature. Three concentration scenarios were created; houses built before 1948 with potentially leaded pipes (0.037 mg/L), all other houses in the conservative assessment (maximum allowable concentration legislated for water treatment facilities 0.010 mg/L), and houses in the average scenario (0.0048 mg/L based on a survey of 40 Ontario homes in the late eighties).		Ontario Average *	Lead Pipe Scenario * MAC Scenario *	
Concentration in Water	It may be important to note that some individuals may consume bottled water instead of water from their tap, resulting in an overestimation of exposure			*	
Water Ingestion Rate	Standard ingestion rates were taken from Richardson (1997).		*		
Absorption Factor	100% relative absorption was assumed for both the conservative and average risk assessments based on PQRA default values.			*	
Exposure Duration	Drinking water exposure was assumed to be daily.		*		

Parameter	Assumption	Under Estimates Risk	No Change in Risk	Over Estimates Risk	Unknown Effect on Risk
Dermal Contact with Water					
Concentration in Water	Lead in St. John's drinking water was not measured so concentrations were estimate from the literature. Three concentration scenarios were created; houses built before 1948 with potentially leaded pipes (0.037 mg/L), all other houses in the conservative assessment (maximum allowable concentration legislated for water treatment facilities 0.010 mg/L), and houses in the average scenario (0.0048 mg/L based on a survey of 40 Ontario homes in the late eighties).		Ontario Average *	Lead Pipe Scenario * MAC Scenario *	
Dermal Permeability Coefficient	Coefficients are different for different species of lead. Because the form of lead was not known the general coefficient of 0.0001 cm/hr was used.				*
Skin Surface Area	Total surface area was taken from Richardson (1997)		*		
Exposure Frequency	Daily bathing was assumed for 45 minutes in the conservative assessment and 20 minutes in the average assessment. This may be a slight overestimation as some individuals may not bathe daily and those that do may bathe for significantly less than the upper bound of 45 minutes.			*	
Inhalation of Outdoor/Indoor Air					
Concentration in Outdoor Air	No data were found for St. John's so the national geometric mean lead concentration was used (0.06 $\mu g/m^3$) for outdoor air. The lack of site specific data may have introduced some unknown effect on the risk estimate.				*
Concentration in Indoor Air	Indoor air was estimated to be 75% of this concentration (0.045 µg/m3). This may be an overestimation as the IEUBK model uses a default of 30% of outdoor air concentration.			*	
Air Intake Rate	Standard inhalation rates were used.		*		

Parameter	Assumption	Under Estimates Risk	No Change in Risk	Over Estimates Risk	Unknown Effect on Risk
Absorption Factor	A relative lung absorption factor of 100% was used for both the conservative and average risk assessments based on PQRA default values..			*	
Exposure Duration	1.5 hours spend outdoors per day (soil) and 22.5 hours spend indoors (dust). Probably a good representation of the mean for residents of St. John's based on questionnaire data and the standard PQRA values. However it may be an overestimate for school age children and adults who work outside the home as the time spent indoors at home would be much less (14.5).		*	*	
Garden Ingestion					
Daily Garden Intake	Garden intake was estimated from both questionnaire data and from daily fruit and vegetable intakes from Richardson (1997). Sample size for the questionnaire was small causing and unknown effect on risk prediction.				*
Plant Uptake Factors	Only the root uptake pathway was considered because there was a lack of data for the parameters needed to predict uptake from particulate deposition. It was assumed that this additional pathway would contribute a negligible amount to total uptake, but it is possible that uptake was underestimated by this omission.	*			
Plant Uptake Factors	Only the plant specific bioconcentration factors that were listed in Boyd *et al.* (1999) were used. Many plants grown in St. John's did not have values and were therefore not considered in the overall uptake factors used which had an unknown effect on risk estimate.				*
Plant Uptake Factors	Plant uptake factors are dependent not only on the type of plant, but also soil pH, organic content, crop density, and environmental and climactic conditions. The uptake factors used from Boyd *et al.* (1999) may not be appropriate for the conditions in St. John's. It is uncertain as to how this would affect the predicted risks.				*

Parameter	Assumption	Under Estimates Risk	No Change in Risk	Over Estimates Risk	Unknown Effect on Risk
Plant Uptake Factors	Plant uptake factors taken from the 3MRA model were also used that were not plant specific, but instead were for general categories (root, exposed vegetables, exposed fruit). These factors gave a higher lead intake than those used from Boyd *et* al. (1999) and also predicted that fruit would have the highest concentration, then vegetables, than roots. This contradicts other research that indicates that lead concentrations are usually highest in roots. Because there is no evidence that one model is better than the other for St. John's the effect of the assumptions from the 3MRA model are unknown.				*
Absorption Factor	100% relative absorption was assumed for both the conservative and average risk assessments based on PQRA default values..			*	
Exposure Duration	Intake was estimated for only three months of the year. This may underestimate intake for those individuals who freeze or preserve their produce.	*			
Body Weight	Canadian average body weights were used in the assessment, but Newfoundlanders are heavier than the national average. In the assessment the total exposure has been divided over a potentially too low body weight therefore over estimating daily intake and risk.			*	
Risk Characterization					
Toxicological Reference Value	Compared total daily intake to oral toxicity value because there is no dermal or inhalation value available for lead. Adding ingestion, inhalation, and dermal absorption and comparing to the oral toxicity value may overestimate risk.			*	
Cumulative Daily Intake	Intake was averaged over an entire lifetime based on the assumption that the receptor was continually exposure to the same environmental conditions. It is likely that the receptor may move over the course of a lifetime.			*	

Appendix E

Uncertainty Analysis for the IEUBK Assessment

Table E.1. The effect of parameter changes on the IEUBK predicted blood lead concentrations.

Parameter	Difference from the IEUBK Default	Under Estimates Risk	No Change in Risk	Over Estimates Risk
Outdoor Air Concentration	Reduced from 0.1 μg m^{-3} to 0.06 μg m^{-3}	*		
Indoor Air Concentration	Increased from 30% of outdoor air concentration to 75%			*
Time Spent Outdoors	Held constant at 1.5 hours day instead of increasing from 1 to 4 hours day by age group	All Others *	0 to1 -	
Ventilation Rate	No difference		-	
Lung Absorption Factor	No difference		-	
Dietary Intake	Slight under estimation for 0 to 3 year olds (-1.35 to -0.01) greater over estimation for 4 to 7 year olds (+0.91 to +2.46)	0 to 3 *		4 to 7 *
Garden Produce	Large increase in total dietary intake based on soil lead concentration so extreme increase for the conservative assessment			***
Dietary Absorption Factor	No difference		-	
Water Concentration	Increased concentration of 37 μg/L and 10 μg/L for the conservative estimate and 4.8 μg/L for the average assessment from the default of 4.0 μg/L		Average -	Conservative **
Water Intake Rate	No difference		-	
Water Absorption Factor	No difference		-	
Soil Concentration	Varies, but this parameter is supposed to vary. In general large increase from 200 μg/g for most of the conservative assessment and only for older properties in the average assessment		-	**
Dust Concentration	Increased from 70% of soil lead concentration to 200%.			***
Soil and Dust Intake Rate	Reduced for all age groups	*		
Soil and Dust Absorption Factor	No difference		-	
Maternal Blood Lead	No difference		-	

- no change in risk
* slight change in risk estimate
** moderate change in risk estimate
*** large change in risk estimate