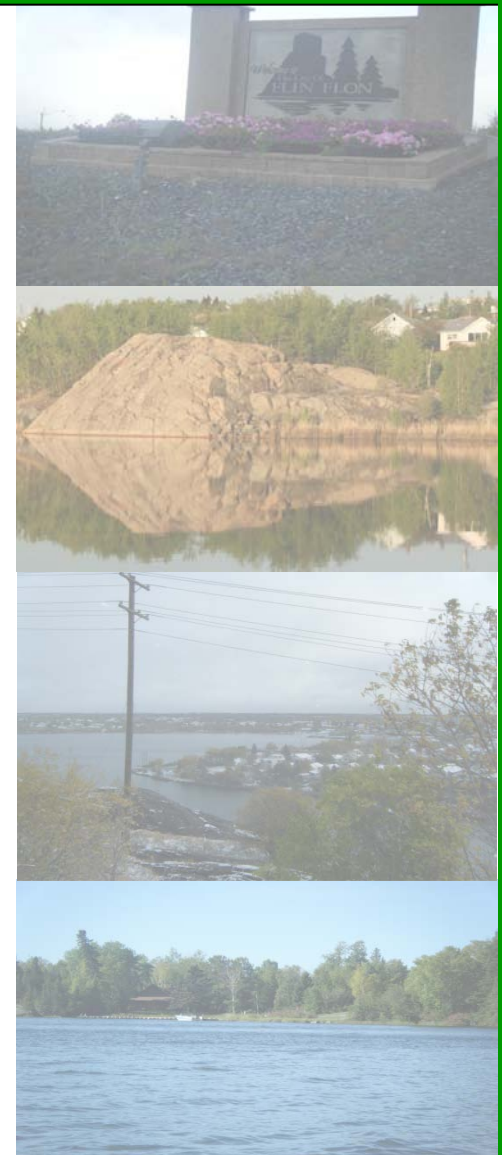


CHAPTER 5
RESULTS AND DISCUSSION



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RESULTS AND DISCUSSION

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5.0 RESULTS AND DISCUSSION

This chapter provides an overview of the results of the HHRA, as well as a discussion of the implications of these results for residents of the Flin Flon-Creighton area. The HHRA considered a variety of data, chemicals, communities, individuals, exposure pathways, exposure durations, and assumptions including:

- Six chemicals of concern (COC) (arsenic, cadmium, copper, lead, mercury, and selenium);
- Four communities of interest (COI) (East Flin Flon, West Flin Flon, Creighton, and Channing) as well as a Typical Background scenario;
- Five receptor age classes (*i.e.*, infant, toddler, child, teen and adult) and a composite lifetime receptor;
- Receptor characteristics characterized by Central Tendency Estimates (CTE);
- Inhalation, oral and dermal exposure pathways;
- Short-term (acute) and long-term (chronic) residential exposure scenarios, and long-term outdoor worker scenario; and,
- A large database of site-specific media concentrations characterized by the 95% upper confidence limit on the mean (95% UCLM).

Separate assessments were completed for short-term (acute) and long-term (chronic) durations because the health outcomes produced by some COC depend on the duration of exposure. It is important to distinguish between the health outcomes that might result from acute exposures *versus* effects that may occur following chronic exposures. In the chronic assessment, further distinction was made between inhalation and multiple pathway exposures since the pathway of exposure could also influence the potential health outcomes associated with each of the COC.

In recognition of the influence of these exposure variables, risk estimates were segregated into:

- Acute inhalation (24 hour durations);
- Acute oral (short-term soil and snow exposure events);
- Residential chronic multiple pathways (*i.e.*, inhalation, oral and dermal exposures); and,
- Commercial/industrial (outdoor worker) chronic multiple pathways (*i.e.*, inhalation, oral and dermal exposures).

Short-term or acute inhalation risk estimates are based on exposure periods that last from a few minutes to a few days and is characterized through consideration of 1-hour and 24-hour maximum air concentrations. For soil and snow pathways, acute exposure estimates are based on short-term transient exposure levels related to extreme activities and upper bound levels of COC in soil and/or snow. Short-term exposures are those defined as occurring over a less than lifetime duration or on an intermittent basis, and are typically characterized by a single pathway exposure only. For example, short-term airborne exposures occur as a result of weather or facility anomalies resulting in short-term deviations from typical concentrations. Short-term soil and snow exposures are those expected to occur only rarely. For example, some children will ingest larger than normal quantities of soil on rare occasions. Similarly, children are assumed to consume snow in relatively large quantities over a short time-frame. Children displaying “pica” behaviour (the intentional ingestion of soil) are discussed further in Chapter 6.

It should be noted that acute Toxicity Reference Value (TRVs) are only available for certain COC and certain time durations and routes of exposure. Due to the significant uncertainties associated with the derivation of acute TRVs, no attempt has been made to characterize risks for those COC that lack an appropriate TRV for the scenario under consideration. In addition, 1-hour maximum air concentrations were not available, as such this time frame was not evaluated.

Long-term or chronic risks (>1 year to a lifetime) are characterized by comparing predicted exposures from all pathways with the exposure limits or toxicity reference values. Chronic health risks were estimated based on the assumption that an individual is continuously exposed to multimedia concentrations. The chronic risk estimates were based on an exposure duration of one year to an assumed lifespan of 80 years (Health Canada, 2006). For non-carcinogenic COC, this comparison is typically referred to as the *Hazard Quotient* (HQ) and is calculated by dividing the predicted exposure level by the exposure limit. If the total chemical exposure from all pathways is equal to or less than the exposure limit, then the HQ would be 1.0 or less, and no adverse health effects would be expected (refer to Chapter 4 for a more detailed discussion of this topic).

For chemicals with non-threshold-type dose responses (*i.e.*, carcinogens), the comparison is referred to as the Incremental Lifetime Cancer Risk Level (ILCR) or more simply a Cancer Risk Level (CRL). The ILCR represents an upper bound estimate of the additional incidence of cancer (*i.e.*, occurrence of cancer that would not be expected in the absence of the exposure) in a population of people exposed every day over their entire lifetime. The ILCR is calculated by multiplying the predicted exposure by the slope factor or unit risk value. The ILCR is expressed as the prediction that one person per *n* people would develop cancer, where the magnitude of *n* reflects the risks to that population. In the case of carcinogens, the acceptable risk level in Manitoba is considered to be an incremental increase in cancer risk of one-in-one hundred thousand (*i.e.*, one additional cancer per one hundred thousand people). Incremental lifetime cancer risks are calculated by multiplying a chemical- and route-specific cancer slope factor by facility related exposures.

Exceedance of the acceptable non-cancer and cancer risk levels is used as a general indicator of unacceptable or elevated risk; however, elevated risk may occur to varying degrees. Recognized experts in the field of risk communication have suggested the use of qualitative terminology to express the varying levels of numerical risk (Calman, 1996; Paling, 2003). A carcinogenic risk level of less than 1-in-1 million is considered *negligible*; between 1-in-1 million and 1-in-100,000 is considered *minimal*, and for the purposes of this risk assessment, acceptable; between 1-in-100,000 and 1-in-10,000 is considered *very low*; between 1-in-10,000 and 1-in-1,000 is considered *low*; between 1-in-1,000 and 1-in-100 is considered *moderate*; and greater than 1-in-100 is considered *high* (Calman, 1996; Paling, 2003). These definitions may be useful in understanding the relative risks expressed below.

As discussed previously, risk assessments typically employ the 95% UCLM to characterize the exposure point concentration (EPC) of a given exposure unit (U.S. EPA, 2001a). The sample mean is based on a collection of samples from the exposure unit and therefore, uncertainty exists as to whether the sample mean is a true reflection of the population mean. As a result, the 95% UCLM can be thought of as an estimate of the true population mean for a given exposure unit. In this case, the exposure units were defined as the communities under assessment in the HHRA. The underlying assumption used when developing the chronic residential exposure scenarios was that individuals would move randomly within each community and, therefore, over time, come into contact with the average soil concentration within a given community (or exposure unit). In reality, individuals do not move in a random

fashion within their community, but rather exhibit predictable spatial patterns in their movements. For example, many individuals will tend to spend the majority of their time between home and work or school. Therefore, the evaluation of risks on the basis of average EPCs (assuming random movement) in an area-wide risk assessment may underestimate risks for some receptors. As a result, in addition to predicting risks using the community-based EPCs, soil provisional trigger concentrations (PTCs) were derived for each COC to be protective of residential receptors. These PTCs can then be used to determine on a property-by-property basis, which properties contain concentrations that have the potential to cause unacceptable risks.

A PTC can be defined as the average COC soil concentration within an exposure unit (EU) that corresponds to an acceptable level of risk (U.S. EPA, 2001a). In other words, the PTC is the EPC in soil within a given EU (*i.e.*, a residential property) which would yield an acceptable level of risk. Exceedances of the PTC do not necessarily indicate that conditions exist in which unacceptable health risks will occur, but rather that there is less certainty regarding the related risk level.

The U.S. EPA (2001a) recommends the use of iterative forward calculation methods when generating PTCs with non-linear parameters. The current assessment included the estimation of indoor dust concentrations based on a potentially non-linear relationship with concentrations in outdoor soil. Therefore, the iterative forward calculation method outlined by the U.S. EPA (2001a) was used to generate PTCs for this study. This method involves collecting data from multiple model runs. Each run uses a different EPC in soil. The calculation is conducted until the EPC corresponding to an HQ value of 1.0 is determined. This EPC corresponds to the PTC as it indicates the soil level corresponds to an acceptable level of risk.

The PTCs do not represent soil remediation objectives but instead are derived to determine if further consideration is required, and if warranted, to help focus the efforts of a biomonitoring program on those areas or properties that may be of the greatest concern. Should a biomonitoring program be completed, the results will be used to further evaluate risk levels.

A detailed discussion of the exposure and risk calculations for the acute, chronic residential, chronic outdoor worker, and recreational exposure scenarios are provided in Sections 5.1 to 5.4.

5.1 Detailed Discussion of Short-Term Results

Concentration Ratio (CR) values were used to evaluate the acute health risk from exposure to chemicals in air. CR values were calculated by dividing the predicted ground-level air concentration (24 hour) by the appropriate toxicity reference value, according to the following example equation:

$$CR_{duration} = \frac{[Air]_{duration}}{TRV_{duration}}$$

where:

- $CR_{duration}$ = The duration-specific CR (unitless), calculated for 24 hour durations
- $[Air]_{duration}$ = The predicted ground-level air concentration ($\mu\text{g}/\text{m}^3$) for the specific time duration
- $TRV_{duration}$ = The toxicity reference value ($\mu\text{g}/\text{m}^3$) for the specific time duration

Acute CR values at or less than 1.0 indicate that estimated COC concentrations in air are at or less than the applicable air TRV, and thus, adverse health outcomes would not be expected to occur. As this is usually a straight comparison between predicted short-term air concentrations (*i.e.*, 24 hour) and the selected TRV, the resulting CR value is receptor-independent (*i.e.*, the same value is calculated for all receptor types).

In general, interpretation of the CR values proceeded as follows:

CR ≤ 1.0

Signifies that the air concentration is less than or equal to the TRV (*i.e.*, the assumed safe level of exposure). This shows that negligible health risks are predicted. Added assurance of protection is provided by the high degree of conservatism (protection) incorporated in the derivation of the TRV.

CR > 1.0

Signifies the air concentration exceeds the regulatory TRV. This suggests that the potential for an elevated level of risk may be present for some COC. The significance of which must be balanced against the high degree of conservatism incorporated in the risk assessment (*i.e.*, the margin of safety is reduced but not removed entirely).

Hazard Quotients (HQ_{acute})

Acute HQ values were used to express risk resulting from acute exposures from soil and snow pathways. Acute exposure estimates were based on short-term transient exposure levels related to extreme activities and extreme levels of COC in soil and/or snow. HQ_{acute} values were calculated by dividing the predicted exposure by the appropriate toxicity reference value (RfD), according to the following example equation:

$$HQ_{acute} = \frac{Exp_{acute}}{TRV_{acute}}$$

where:

- HQ_{acute} = The Hazard Quotient (unitless), calculated for exposures resulting from acute exposure scenarios
- Exp_{acute} = The exposure estimate resulting from acute exposure scenarios ($\mu\text{g}/\text{kg}$ bodyweight/day)
- TRV_{acute} = The acute toxicity reference value (TRV) ($\mu\text{g}/\text{kg}$ bodyweight/day)

HQ_{acute} values less than 1.0 indicate that estimated chemical exposures are less than the applicable TRV, and thus, adverse health outcomes would not be expected to occur. As this usually involves an estimate of exposure, the resulting HQ_{acute} value is receptor-dependent. Since the toddler age group is the most likely age group to ingest soil and snow in elevated amounts, and toddlers are typical the most sensitive receptor group, the acute assessment focuses on toddlers.

In general, interpretation of the HQ_{acute} values proceeded as follows:

$$HQ_{acute} \leq 1.0$$

Signifies that the estimated exposure is less than or equal to the TRV (*i.e.*, the assumed safe level of exposure). This shows that negligible health risks are predicted. Added assurance of protection is provided by the high degree of conservatism (protection) incorporated in the derivation of the TRV.

$$HQ_{acute} > 1.0$$

Signifies the exposure estimate exceeds the regulatory TRV. This suggests that the potential for an elevated level of risk may be present for some COC. The significance of which must be balanced against the high degree of conservatism incorporated in the risk assessment (*i.e.*, the margin of safety is reduced but not removed entirely).

5.1.1 Acute Inhalation Health Risks

The following sections provide a discussion of the predicted acute exposure and risks for receptors in the Flin Flon-Creighton area (Table 5-1). For inhalation related risks, the risk evaluation is a simple comparison of measured air concentrations and appropriate TRVs.

COI and COC	Exposure Duration	Air Concentration ($\mu\text{g}/\text{m}^3$)	TRV ($\mu\text{g}/\text{m}^3$)	CR_{acute}
West Flin Flon				
Arsenic	24 hrs	0.74	0.3	2.5
Cadmium	24 hrs	0.66	2.0	0.33
Copper	24 hrs	4.2	50	0.084
Lead	24 hrs	2.4	2.0	1.2
Mercury (inorganic)	24 hrs	0.056 ^a	2.0	0.028
Selenium	24 hrs	0.27 ^a	10	0.027
East Flin Flon and Channing				
Arsenic	24 hrs	0.22	0.3	0.73
Cadmium	24 hrs	0.12	2.0	0.060
Copper	24 hrs	0.82	50	0.016
Lead	24 hrs	0.39	2.0	0.20
Mercury (inorganic)	24 hrs	0.00032	2.0	0.00016
Selenium	24 hrs	0.089	10	0.0089
Creighton				
Arsenic	24 hrs	0.03	0.3	0.10
Cadmium	24 hrs	0.018	2.0	0.0090
Copper	24 hrs	0.28	50	0.0056
Lead	24 hrs	0.14	2.0	0.070
Mercury (inorganic)	24 hrs	0.0042	2	0.0021
Selenium	24 hrs	0.02	10	0.0020

Bolded values highlighted in grey exceed a CR of 1.0.

^a Concentration is based on the maximum concentration measured for Creighton adjusted according to the relationship between maximums for arsenic, cadmium, copper, and lead for West Flin Flon and Creighton (*i.e.*, an adjustment factor of 13.4 was applied to the maximums for Creighton).

Elevated risks are predicted for arsenic and lead in the West Flin Flon area only. By definition, acute exposure are short-term and transient in nature, typically occurring as a result of a unique or extreme weather condition or facility anomaly resulting in short-term concentration excursions from normal average concentrations. The results of this evaluation indicated that some people may experience short-term and reversible health effects at intermittent times during facility

operations, potentially occurring at times when air concentrations exceed the TRVs. These occurrences are somewhat rare (see Table 5-2), the magnitude of exceedances are less than an order of magnitude and the margins of safety inherent in the acute TRVs are large, indicating that the occurrence of acute health effects is unlikely. Table 5-2 indicates the number of samples collected for one year from September 2007 to August 2008 that are in excess of the acute TRV.

COI	Arsenic	Cadmium	Copper	Lead	Mercury	Selenium
West Flin Flon	9 of 210	0 of 210	0 of 210	2 of 210	ND	ND
East Flin Flon and Channing	0 of 57	0 of 57	0 of 57	0 of 57	0 of 57	0 of 57
Creighton	0 of 59	0 of 59	0 of 59	0 of 59	0 of 59	0 of 59

^a Air data considered in the calculation of CRs for East Flin Flon, Channing, and Creighton were collected from September 2007 to August 2008, for West Flin Flon from June 2007 to June 2008.

ND No data (short term air concentration predicted).

Of note, the concentrations of cadmium in ambient air does not result in an elevated risk. Manitoba Conservation has selected the OMOE 24 hour air standard of 2 µg/m³ for comparison to concentrations measured in TSP in Flin Flon and Creighton. From the year spanning from June 2007 to July 2008, all samples measured at the Provincial Building (n=210), Ruth Betts (n=129), and Creighton School (n=192) were below this standard. Although this indicates that concentrations of cadmium in ambient air measured at these stations are not anticipated to result in adverse health effects, it should be noted that the standard of 2 µg/m³ has been updated by the OMOE within Regulation 419 (February, 2008). A standard of 0.025 µg/m³ has been derived by the OMOE to be protective of health effects including lung cancer, however, this standard does not come into effect until February, 2013. The OMOE has indicated that until this date, a standard of 0.25 µg/m³, which represents an upper risk threshold, should be used. Following the approach used by Manitoba Conservation which utilized TSP air data, comparison of the concentrations of cadmium measured at each of the sampling stations with the updated standards protective of lung cancer indicates several exceedances (Table 5-3).

Sampling Location	Number of Samples in Excess of Air Standard		
	2 µg/m³	0.25 µg/m³	0.025 µg/m³
Provincial Building (n=210)	0	8	64
Ruth Betts (n=129)	0	1	16
Creighton School (n=192)	0	0	2

This comparison indicates that there were a number of days in which concentrations of cadmium were above the concentrations associated with potential health effects. It should be noted that the endpoint of concern for the 0.025 µg/m³ criterion is cancer, and that cancer is not typically considered in the evaluation of short-term exposures. Cadmium related cancer risks evaluated utilizing the Health Canada Inhalation Unit Risk value are discussed in Section 5.2.2.

5.1.2 Acute Ingestion Health Risks

Acute Ingestion of Soil

For soil ingestion-related risks, the risk evaluation requires a simple exposure calculation as indicated in the following equation:

$$EXP_{acute} = \frac{[soil]_{max} \times IR_{acute}}{BW}$$

where:

EXP _{acute}	=	Acute exposure estimate (µg/kg/day)
[soil] _{max}	=	Maximum measured soil concentration (µg/g)
IR _{acute}	=	Acute soil ingestion rate (assumed to be 400 mg/day or 0.4 g/day; defined by U.S. EPA (2002) as the upper bound estimate of soil ingestion in normal children)
BW	=	Body weight (kg)
HQ _{acute}	=	Estimates were then calculated as indicated in Section 5.1 (Table 5-4)

<i>COI and COC</i>	<i>Max Soil Concentration (µg/g)</i>	<i>Estimated Exposure (µg/kg-day)</i>	<i>TRV (µg/kg-day) (oral exposure limit)</i>	<i>HQ</i>
West Flin Flon				
Arsenic	237	5.8	5.0	1.2
Cadmium	71	1.7	4.1	0.42
Copper	7,810	190	10	NA
Lead	820	20	NA	NA
Mercury	971	24	7.0 (inorganic)	3.4
Selenium	286	6.9	NA	NA
East Flin Flon				
Arsenic	33	0.80	5.0	0.16
Cadmium	27	0.65	4.1	0.16
Copper	2,050	50	10	NA
Lead	333	8.1	NA	NA
Mercury	18	0.44	7.0 (inorganic)	0.062
Selenium	12	0.29	NA	NA
Channing				
Arsenic	36	0.87	5.0	0.17
Cadmium	21	0.51	4.1	0.12
Copper	700	17	10	NA
Lead	266	6.4	NA	NA
Mercury	7.0	0.17	7.0 (inorganic)	0.024
Selenium	4.0	0.10	NA	NA
Creighton				
Arsenic	300	7.3	5.0	1.4
Cadmium	32	0.78	4.1	0.19
Copper	1,800	44	10	NA
Lead	456	11	NA	NA
Mercury	24	0.58	7.0 (inorganic)	0.083
Selenium	18	0.44	NA	NA

Bolded values highlighted in grey exceed an HQ of 1.0.

For acute soil, marginal exceedances were noted for arsenic in West Flin Flon and Creighton, and for mercury in West Flin Flon. By definition, acute exposures are short-term and transient in nature. The results of this evaluation indicated that some people may experience short-term and reversible health effects (such as irritation and gastrointestinal upset). However, it must be noted that the likelihood is rare, the magnitude of exceedances are small and the margins of safety inherent in the acute TRVs are large, indicating that the occurrence of acute health effects is unlikely.

It is noted that reliable short-term TRVs were not available for several COCs which results in problems quantifying acute risks. Inclusion of TRVs with large uncertainties may result in the inappropriate interpretation of acute risks causing concern when none exists. The characterization of chronic risk will protect for acute exposures since most of the pathways (*i.e.*, soil, drinking water, food) are chronic exposure situations, with the exception of the pica child behavior. Pica is a unique condition and is discussed more fully in Chapter 6, Section 6.5.

Acute Ingestion of Snow

For snow ingestion related risks, the risk evaluation requires a simple exposure calculation as indicated in the following equation:

$$EXP_{acute} = \frac{[snow] \times IR_{acute}}{BW}$$

where:

EXP _{acute}	=	Acute exposure estimate (µg/kg/day)
[snow] _{max}	=	Measured snow concentration (µg/L)
IR _{acute}	=	Estimated snow ingestion rate [it was assumed that a toddler would consumer 50 mL of snow (approximately a mouthful) per day and that the snow contained a 3 to 1 volume to volume ratio of snow to water resulting in a daily ingestion of 16.7 mL snow water per day]
BW	=	Body weight (kg)
HQ _{acute}	=	Estimates were then calculated as indicated in Section 5.1 (Table 5-5).

COC	Snow Concentration (µg/L)		Estimated Exposure (µg/kg-day)		TRV (µg/kg-day) (oral exposure limit)	HQ _{acute}	
	MAX	95% UCLM	MAX	95% UCLM		MAX	95% UCLM
Arsenic	147	96	0.15	0.10	5	0.03	0.02
Cadmium	183	113	0.18	0.11	4.1	0.05	0.03
Copper	4940	3000	5.0	3.0	10	0.50	0.30
Lead ^a	732	483	0.74	0.49	3.6	0.21	0.14
Mercury	1.8	1	0.0018	0.0010	7	0.0003	0.0001
Selenium ^a	2.8	2	0.0028	0.0020	6.2	0.0005	0.0003

^a Short-term TRVs were not available for lead or selenium, as a result chronic TRVs were conservatively utilized in this calculation. Chronic TRVs were used in lieu of acute TRVs for the evaluation of snow since snow was not considered in the chronic evaluation.

No acute HQ exceedances were noted for the snow pathway indicating that infrequent consumption of snow within the Flin Flon-Creighton area is not anticipated to result in adverse health effects.

5.2 Detailed Discussion of Long-Term Residential Results

The following sections provide a discussion of the predicted exposure, risks, and soil PTCs derived for residential receptors in the Flin Flon-Creighton area. Note that the results presented for non-carcinogenic risks are generally for the toddler and for carcinogenic risks for the lifetime

composite receptor. Detailed results for all COI, COC, and receptors are provided in Appendix M.

5.2.1 Arsenic

Estimated Exposure to Arsenic

The single largest source of exposure to arsenic for residents of each COI was through the consumption of market basket (*i.e.*, grocery store) food items, representing 55, 44, 49, and 54% of the total daily exposure for residents of East Flin Flon, West Flin Flon, Creighton and Channing, respectively (Figure 5-1). Although market basket exposure was assumed to be the same for residents in each of the four COI, the percent contribution of market basket foods to total exposure differed as a result of differences from exposure to community-specific environmental media concentrations (*i.e.*, soil, dust, air, and drinking water). Arsenic content in market basket food items is unrelated to environmental contamination in the Flin Flon area and is reflective of foods consumed throughout Canada and North America. The next largest sources of exposure for each COI are through direct contact (*i.e.*, incidental ingestion and dermal contact) with impacted outdoor soil and indoor dust (10, 23, 28 and 11% for East Flin Flon, West Flin Flon, Creighton and Channing, respectively) and the consumption of drinking water (24, 19, 16 and 24%). Contributions from the consumption of local fish, wild game, and blueberries were very minor, each representing less than 2% of the total exposure for toddlers in each COI. The exposure of arsenic *via* the inhalation pathway was greatest for residents of West Flin Flon (representing 8% of the total exposure for the toddler), but was much lower for each of the other COI (5, 1 and 5% for East Flin Flon, Creighton, and Channing, respectively) which experience lower outdoor air concentrations.

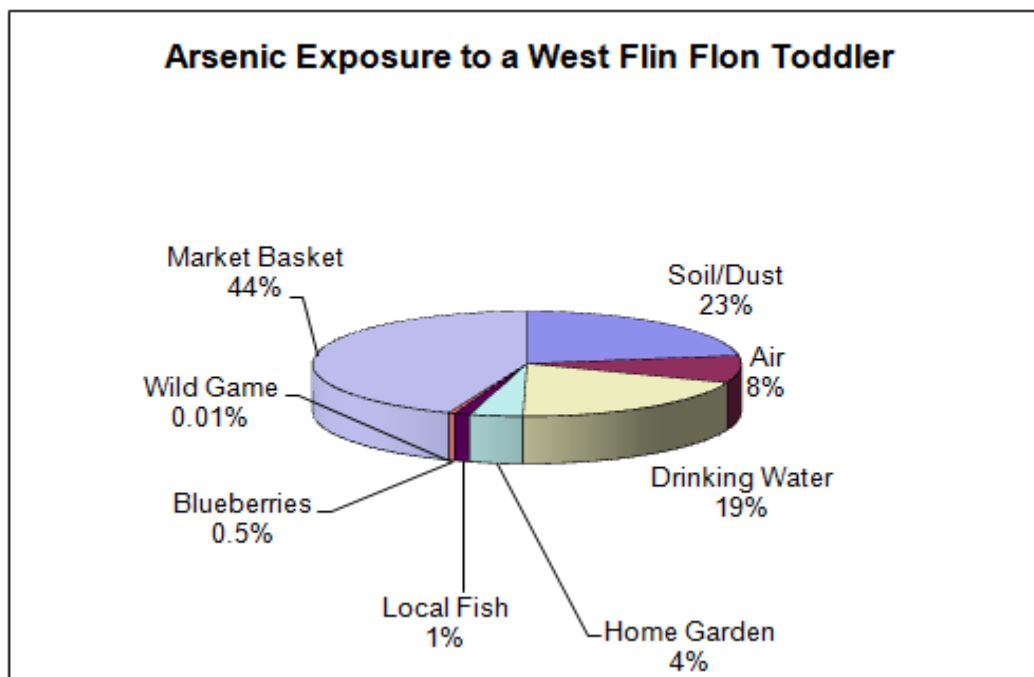


Figure 5-1 Contributions of Media to Inorganic Arsenic Exposure for a Toddler Living in West Flin Flon

Soil/dust related exposure in Creighton were elevated relative to West Flin Flon as a result of higher concentrations of arsenic in outdoor soil. Exposure related to the inhalation of arsenic in air was lower in Creighton as a result of lower ambient air concentrations (Figure 5-2).

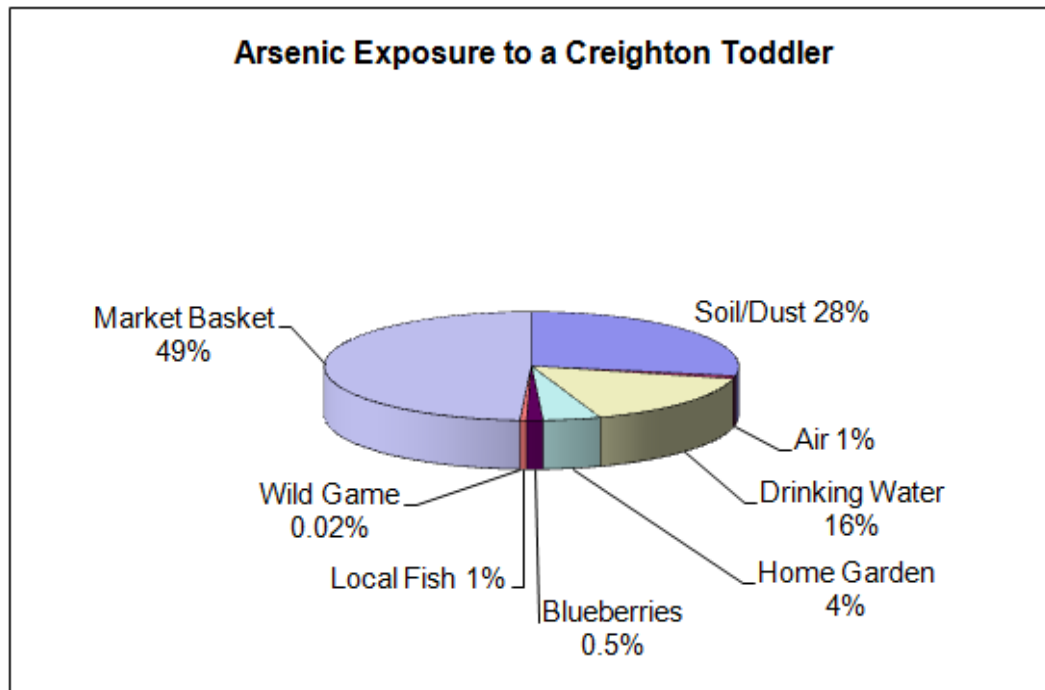


Figure 5-2 Contributions of Media to Inorganic Arsenic Exposure for a Toddler Living in Creighton

Although risks associated with exposure to arsenic *via* the inhalation pathway were assessed using inhalation-specific exposure limits, the contribution of arsenic absorbed through this route to the total daily dose was also considered.

A detailed breakdown of the pathway-specific exposures for residents of West Flin Flon in each of the 5 age classes is provided in Table 5-6. Details for receptors in other communities are provided in Appendix M.

Exposure Pathway	Environmental Media Concentrations		Percent of Lifetime Total EDI	Estimated Daily Intakes (EDI) (µg/kg bw/day)					
	Value	Units		Infant	Toddler	Child	Teen	Adult	Lifetime
Inhalation of Fine Particulate	0.084	µg/m ³	10%	0.022	0.047	0.037	0.022	0.019	0.022
Dermal Contact – Outdoors	77	µg/g	1.8%	0.0087	0.0064	0.0049	0.0039	0.0037	0.0040
Dermal Contact – Indoors	68	µg/g	0.56%	0.0031	0.0021	0.0015	0.0012	0.0011	0.0012
Outdoor Soil Ingestion	77	µg/g	4.6%	0.041	0.082	0.010	0.0057	0.0048	0.0099
Indoor dust Ingestion	68	µg/g	2.0%	0.018	0.036	0.0046	0.0025	0.0021	0.0044
Home Garden Root Vegetables	0.012	µg/g ww	0.25%	0.00023	0.0010	0.00087	0.00067	0.00047	0.00055
Home Garden Other Vegetables	0.12	µg/g ww	5.9%	0.020	0.022	0.018	0.012	0.011	0.013
Local Wild Blue Berries	0.035	µg/g ww	1.2%	0.0026	0.0025	0.0026	0.0026	0.0026	0.0026
Local Wild Game	0.00017	µg/g ww	0.035%	0	0.000078	0.000078	0.000077	0.000077	0.000077
Local Fish	0.0097	µg/g ww	3.0%	0	0.0065	0.0065	0.0065	0.0065	0.0065
Drinking Water	3	µg/L	30%	0.11	0.11	0.073	0.050	0.064	0.066
Market Basket Contribution	NA	µg/g	40%	0.28	0.25	0.16	0.096	0.063	0.087
Summary									
Estimated Daily Intake (µg/kg/day)			--	0.52	0.58	0.34	0.22	0.19	0.23
Inhalation Route Only			10%	0.022	0.047	0.037	0.022	0.019	0.022
Direct Soil/Dust Contact			9.0%	0.071	0.13	0.021	0.013	0.012	0.020
Market Basket Foods			40%	0.28	0.25	0.16	0.096	0.063	0.087
Drinking Water			30%	0.11	0.11	0.073	0.050	0.064	0.066
Local Foods (Home Garden, Blueberries, Wild Game and Fish)			10%	0.023	0.032	0.028	0.022	0.021	0.022

Hazard Quotients for Non-Cancer Risks

Although lifetime cancer risk is typically the end-point of interest when assessing the human health implications of inorganic arsenic exposure, non-cancer end-points have also been established to be protective of these effects. The U.S. EPA has recommended an oral TDI for arsenic of 0.3 µg/kg/day to be protective of non-cancer effects such as hyperpigmentation, keratosis, and possible vascular complications. Exposure of arsenic *via* inhalation may result in distinct non-cancer health effects. This was assessed through a comparison of the ambient air EPCs to the air standard as described below. However, since exposure resulting from inhalation can contribute to the total internal dose, this contribution was also included in the total exposure and comparison to the oral TDI.

HQ estimates for residents living in each of the COI ranged from 0.49 to 1.9, with the highest HQs predicted for residents of West Flin Flon (Table 5-7).

Table 5-7 Predicted Hazard Quotients from Exposure to Arsenic

Receptor	East Flin Flon	West Flin Flon	Creighton	Channing	Typical Background
Infant	1.5	1.7	1.5	1.5	1.3
Toddler	1.5	1.9	1.7	1.5	1.2
Child	0.96	1.1	0.91	0.97	0.80
Teen	0.61	0.68	0.57	0.61	0.49
Adult	0.54	0.60	0.49	0.54	0.43

Bolded values highlighted in grey exceed the acceptable HQ of 1.0.

HQ estimates for the Typical Background scenario were similar to those predicted for residents in the Flin Flon-Creighton area. This is a result of the significant contribution of market basket foods to total exposure and the similar concentrations of arsenic in drinking water in the Flin Flon-Creighton area relative to background drinking water concentrations (Figure 5-3).

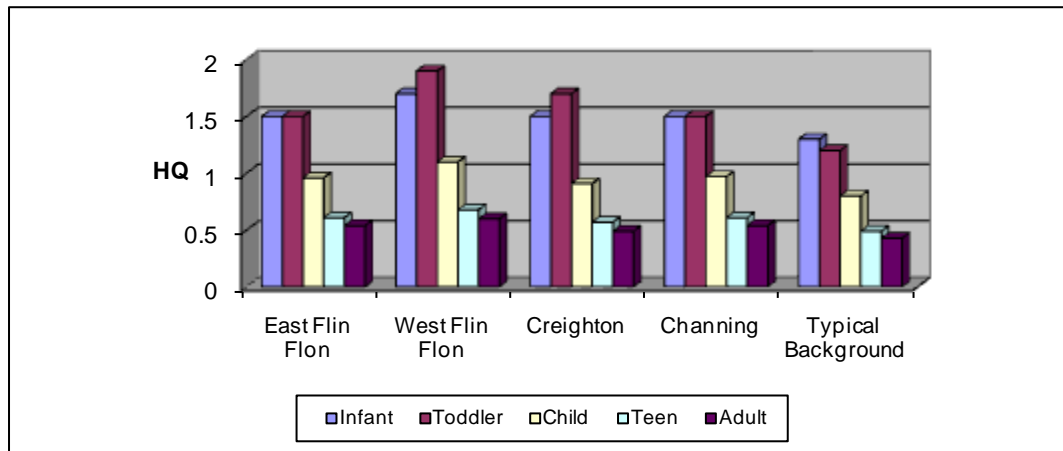


Figure 5-3 Predicted Hazard Quotients for a Toddler as a Result of Exposure to Arsenic

The difference in exposure to inorganic arsenic between residents of the COI and the Typical Background conditions can be attributed primarily to incidental soil/dust ingestion and inhalation of ambient air. This difference in exposure is a result of the elevated EPCs for soil and air in the Flin Flon-Creighton area relative to the Typical Background concentrations. Overall, the contribution of exposure *via* inhalation to the systemic risk level is relatively minor. Contribution of risk from exposure through the consumption of local foods (*i.e.*, wild game, fish, blueberries, and home garden vegetables) is also minor relative to the total risk level (Figure 5-4). As

previously mentioned, the total local foods exposure pathway applies only to the Flin Flon-Creighton COI. The market basket risks for a Typical Background scenario are slightly higher than those of the COI because a proportion of an individual’s diet (living within a COI) has been apportioned to locally derived foods.

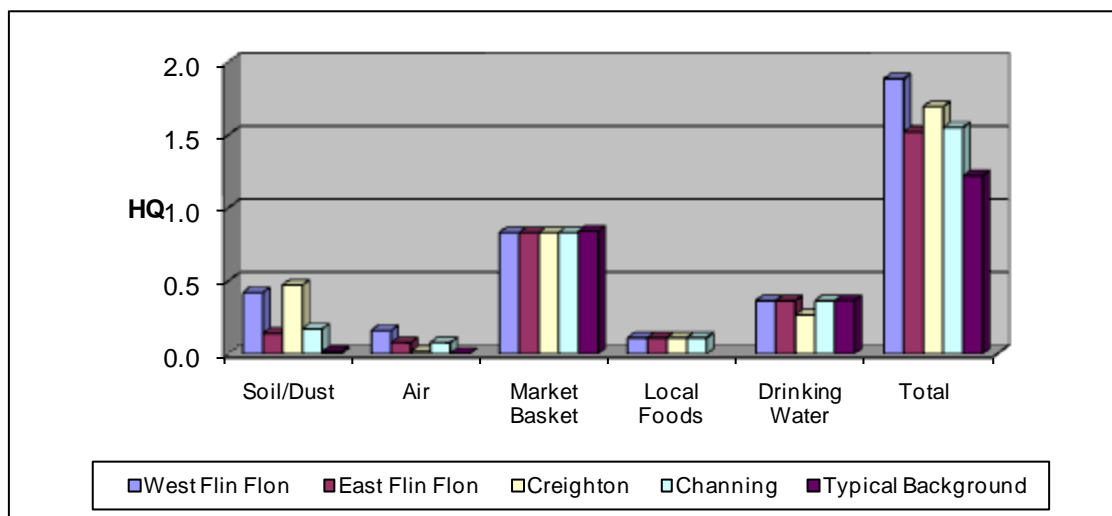


Figure 5-4 Pathway-Specific Hazard Quotients for a Toddler in each COI as a Result of Exposure to Arsenic

Therefore, although the consumption of drinking water and market basket foods are two of the largest sources of overall arsenic risk to residents of the COI, contributions from these sources are essentially equivalent to those experienced by receptors living in communities that are not impacted by a point source of arsenic emissions. The incremental increase in risks for Flin Flon area residents above background risks are primarily attributed to the incidental ingestion of soil and dust with elevated concentrations of arsenic.

Exposure to arsenic *via* inhalation may elicit adverse effects that differ from those described in oral exposure studies. Although there is no indication that inhalation exposure will result in adverse effects within sensitive respiratory tissues, additional systemic effects have been noted in inhalation studies. As a result, the contribution of exposure *via* inhalation was included in the total multimedia assessment using the oral TDI, as well as through a comparison of the ambient air EPC to the air standard (0.03 µg/m³) protective of non-cancer adverse effects *via* the inhalation route during chronic exposure. This comparison produces a concentration ratio (CR). Concentration ratios that are at or below 1.0 are considered to represent acceptable exposure levels. This assessment indicated that the EPCs for arsenic in ambient air in East Flin Flon, West Flin Flon, and Channing are above the air standard protective of adverse non-cancer effects as a result of chronic exposure to arsenic in ambient air (Table 5-8).

	<i>East Flin Flon</i>	<i>West Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>
EPC (µg/m ³)	0.040	0.084	0.0085	0.040
CR	1.3	2.8	0.28	1.3

Given the minor exceedance of the acceptable CR of 1.0 in East Flin Flon and Channing, and that the EPC for these communities is based on data collected from Ruth Betts which is likely to over predict air concentrations in these areas, it is not anticipated that there are significant non-cancer risks associated with exposure to arsenic *via* the inhalation pathway alone. Higher

concentrations of arsenic in ambient air in West Flin Flon create a greater potential for the occurrence of adverse effects, however, given the conservatism associated with the derivation of the air standard, the CR of 2.8 is still considered to be a relatively minor exceedance. Future reductions in smelter-related emissions containing arsenic would have a direct and immediate effect on reducing inhalation-related exposure and risks.

ILCR Estimates for Arsenic

ILCR estimates for inorganic arsenic represent the additional (or incremental) lifetime cancer risk resulting from the predicted lifetime average daily exposure to inorganic arsenic from all site-specific exposure pathways. It should be recognized that only those exposure pathways specific to the COI were included in the derivation of ILCR estimates. ILCR estimates for the lifetime composite receptor were considered to be low, ranging from 3.0×10^{-4} in East Flin Flon to 5.0×10^{-4} in West Flin Flon (Table 5-9). The Health Canada acceptable ILCR is 1.0×10^{-5} , or one-in-one hundred thousand. Since the ILCR is an estimate of the cancer risk associated with exposure to COC in the Flin Flon-Creighton area only, exposure and risks resulting from the consumption of market basket food items was not included in the calculation of the ILCR. For comparative purposes, the total CRL for a Typical Background scenario is presented in Figure 5-5, which includes risks associated with exposure to background levels of arsenic in soil/dust, air, and drinking water.

	<i>East Flin Flon</i>	<i>West Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>	<i>Typical Background</i>
ILCR	3.0E-04	5.0E-04	1.7E-04	3.1E-04	1.0E-04

Bolded values highlighted in grey are in excess of the acceptable ILCR of 1.0×10^{-5} .

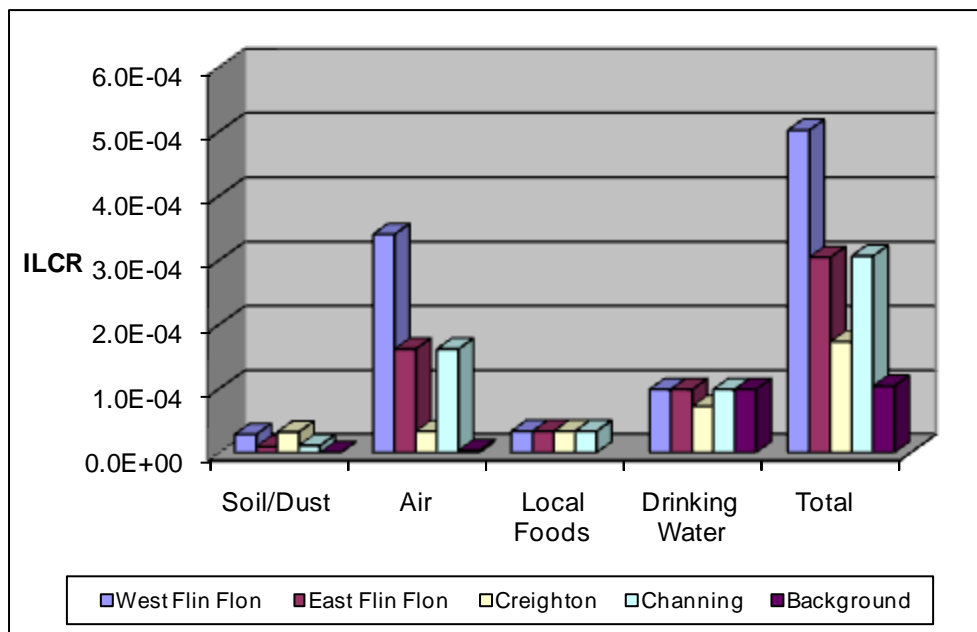


Figure 5-5 Pathway-Specific ILCRs for a Lifetime Composite Receptor in each COI as a Result of Exposure to Arsenic

As shown in Figure 5-5, the most significant source of cancer risk for East and West Flin Flon and Channing is the inhalation of arsenic in air. Since the EPCs of arsenic in ambient air in

Creighton are lower than in other COIs, this pathway is less significant in Creighton. As discussed above for non-carcinogenic effects, future reductions in smelter-related emissions containing arsenic would have a direct and immediate effect on reducing cancer-related risks. Concentrations of arsenic in drinking water in Flin Flon and Creighton are similar to those measured in drinking water throughout Manitoba and Saskatchewan. Therefore, the cancer risk associated with the consumption of drinking water for Flin Flon area residents does not represent an incremental increase in cancer risks experienced under the Typical Background scenario *via* this pathway.

To help put the predicted ILCRs into perspective, it is useful to consider the cancer risks related to the consumption of market basket food items. The ILCRs resulting from Flin Flon/Creighton site-related contributions represent 33 to 64% of the total CRL, with market basket foods representing 20 to 43% of the total CRL. When the CRLs associated with market basket foods and drinking water are combined, these background sources represent 36 to 67% of the total CRL for Flin Flon/Creighton area residents (Figure 5-6).

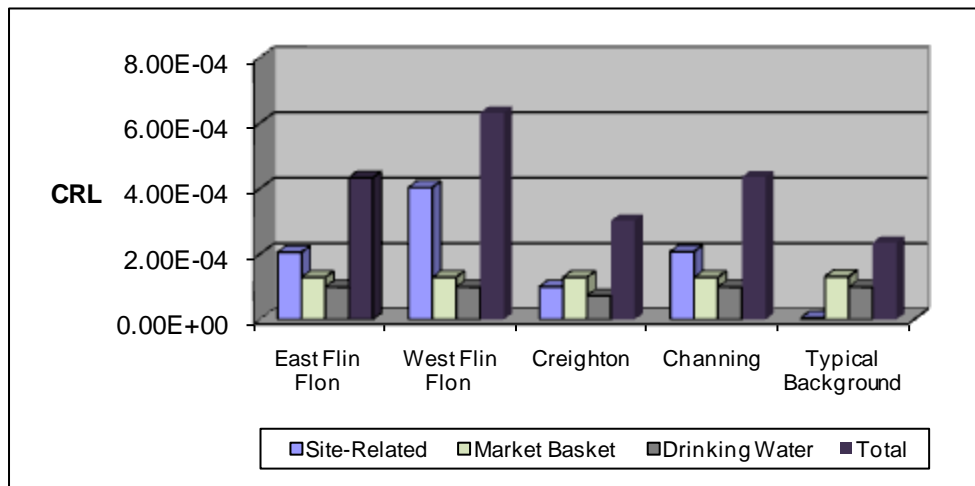


Figure 5-6 Comparison of the Cancer Risk Levels Associated with Site-Related Environmental Exposure and Exposure from Consumption of Market Basket Foods and Drinking Water

Since there are a number of unique issues associated with the assessment of risk from exposure to arsenic, a weight-of-evidence discussion is provided to provide further context on the results of the HHRA.

Weight-of-Evidence Discussion for Arsenic

Risk assessment of arsenic-contaminated sites is a complex and problematic exercise, the interpretation of which has been a source of controversy and complication when managing these sites. The issue of the cancer potency of arsenic, and the interpretation of and response to predicted risks in excess of the traditional de minimis or negligible risk levels of one-in-one-hundred thousand, has complicated issues surrounding the risk assessment and management of arsenic-contaminated sites. The issue is complicated by the fact that its cancer potency, as assessed by all major regulatory agencies, including Health Canada, WHO, Cal EPA and U.S. EPA, is based upon Taiwanese epidemiological studies of drinking water exposure in the 1970's (*i.e.*, Tseng *et al.*, 1968; Tseng, 1977) in which populations were exposed to inorganic arsenic primarily in their drinking water.

Several weaknesses of these studies have been cited in the scientific literature, including poor nutrition in the study population, possible genetic predisposition to adverse effects, unquantified exposures to arsenic from other sources, and possible bias in examiners (U.S. EPA, 1998). Despite these shortcomings, regulatory agencies continue to rely on these studies as the basis for oral slope factors since evidence of carcinogenicity in humans is considered to be superior to animal studies when classifying carcinogens. Among the concerns raised in relation to the Taiwanese studies are the following:

- Adequacy of the model used by U.S. EPA to derive the slope factor;
- Accuracy and reliability of the exposure data (Brown *et al.*, 1997a,b). The selection of exposure groups based on average concentrations of inorganic arsenic in Taiwanese village wells is a key source of uncertainty in these studies (Brown *et al.*, 1997a; Chappell *et al.*, 1997). Villages were represented by average well water concentration, although there were large variations in arsenic concentrations across individual wells. This precludes any ability to assess individual rates of exposure to those who developed cancers because of the lack of linkage with well water concentrations of arsenic, and well usage rates. The considerable variation in inorganic arsenic concentrations within the wells was recognized by Tseng *et al.* (1968), but was not addressed in the exposure estimation. Furthermore, concentrations of arsenic in the wells were measured in the early 1960s, and for many villages, only 2 to 5 analyses were conducted, and for other villages, only one analysis was performed. Also, tap water was supplied to many areas after 1966, and the arsenic-containing wells were only used in dry periods, which would greatly limit arsenic exposures. This was not accounted for. In addition, due to study design, particular wells used by subjects with skin cancer could not be identified and arsenic intake could only be assigned at the village level (U.S. EPA, 1998);
- Unique host and environmental factors among Taiwanese populations that are not applicable elsewhere (Carlson-Lynch *et al.*, 1994);
- A possible threshold for arsenic carcinogenicity and nonlinearities in the dose-response curve (Abernathy *et al.*, 1996; Slayton *et al.*, 1996);
- Differences in health and nutrition between Taiwan and other countries that might lead to higher cancer risks in Taiwan for the same level of exposure (Beck *et al.*, 1995). The importance of nutritional status in arsenic toxicology, as well as the actual nutritional status of the study population, has been debated in the literature. In general, poor nutritional status can lead to increased susceptibility to the toxic action of many chemicals. In the case of arsenic, there may be an additional role of nutrition in relation to carcinogenicity. The biomethylation of arsenic has been postulated to play a role in its genotoxicity and carcinogenicity. The status of the methyl donor pool, which is dependent on dietary intake of proteins and amino acids such as cysteine and methionine, may play a significant role in susceptibility to arsenic carcinogenicity. Indeed, Hsueh *et al.* (1995) found that malnutrition, indexed by a high consumption of dried sweet potato as a staple food, was a risk factor for skin cancer. However, Smith *et al.* (1995) reviewed the Taiwanese intake of protein, and found it adequate by current standards. Beck *et al.* (1995), in rebuttal, pointed out that current standards dictate intakes required for normal bodily processes, and may not be adequate to methylate an excessive and sustained intake of arsenic;
- The possibility that arsenic is an essential nutrient at lower doses (NRC, 1999); and,
- Uncertainty regarding the amount of water consumed daily by Taiwanese males (U.S. EPA, 1998).

The possibility of unquantified significant exposure of the Taiwanese subjects to inorganic arsenic from sources other than the well water was not accounted for (Chappell *et al.*, 1997). The Tseng *et al.* (1968) and Tseng (1977) studies assumed all exposure to inorganic arsenic

was derived from drinking water, when in fact, diet and other environmental sources may have provided significant additional exposure (Brown and Abernathy, 1997; Chappell *et al.*, 1997). Brown and Abernathy (1997) concluded that the inclusion of dietary intake of arsenic in the cancer risk estimate would have significantly reduced the Maximum Likelihood Estimate. Similarly, Schoof *et al.* (1998) note that dietary intake of inorganic arsenic in the Taiwanese study population appears to be considerably higher than was assumed by the U.S. EPA in derivation of the oral cancer slope factor, and that consideration of a more realistic dietary inorganic arsenic intake may result in a substantial reduction in cancer potency estimates for ingested inorganic arsenic. Mushak and Crocetti (1995) noted that proportions of inorganic arsenic species in vegetables, and specifically potatoes, are less than 10%, meaning that dietary exposures to inorganic arsenic would likely be insignificant. However, herbal medicines and teas are widely consumed in Taiwan, and may represent a significant, albeit poorly characterized, source of exposure to inorganic arsenic (Espinoza *et al.*, 1995; Ernst, 1998). In addition, Schoof *et al.* (1999) reported that rice contains the highest inorganic arsenic content of a large number of foods tested for total, inorganic, and organic arsenic content (including fats, oils, sweets, dairy products, meats, eggs, fish, nuts, vegetables, fruits, breads, cereals, and pastas). Rice is a staple food in Taiwan, and likely contributes significantly to total inorganic arsenic exposure. The Tseng *et al.* (1968) and Tseng (1977) studies did not account for any dietary sources of inorganic arsenic.

Concomitant exposures to other chemicals are also key confounding factors. Byrd *et al.* (1996) suggest humic acids, for which elevated concentrations in Taiwanese drinking water are correlated to that of arsenic, and may lead to erroneously high estimates of the cancer risk being attributable to arsenic. Chan and Huff (1997) also considered humic acids to be a potentially confounding factor in these studies, observing that these substances are mutagenic and may produce symptoms similar to those of Blackfoot disease if elevated in drinking water. Brown *et al.* (1997a) considered the above limitations, together with the sensitivity of the cancer risk estimate to the model used, and concluded that the Tseng studies are not suitable for dose-response assessment, and for extrapolating to lower exposure levels.

From the concerns and limitations noted above in relation to the Tseng studies, it appears a number of potential confounding factors were not considered in the attribution of elevated skin cancer prevalence rates to inorganic arsenic in drinking water. This results in what is widely considered to be an overly conservative estimate of the carcinogenic potency of inorganic arsenic. Thus, use of the current regulatory agency oral slope factors routinely results in significant risk values in virtually every HHRA of arsenic (including scenarios focused on background concentrations), and even at laboratory detection limits for arsenic in various media.

Background soil concentrations of inorganic arsenic are frequently concluded to have the potential to elevate cancer risks greater than one-in-one-hundred thousand for typical Canadian residents living in an area with no local industrial source of arsenic. When other media that also commonly contain low natural levels of arsenic are included, such as food and drinking water, risk estimates for background exposures can often be in the one- in-one thousand to one-in-ten thousand range. These background cancer risks for arsenic are commonly similar to, and can sometimes be greater than, the arsenic cancer risks associated with an identified source, such as an industrial facility or contaminated site.

A similar situation also exists in the interpretation of non-carcinogenic risks from oral arsenic exposure, as the U.S. EPA oral RfD (used in this HHRA) is also based on health outcomes reported in the Tseng *et al.* (1968) and Tseng (1977) studies. Thus, any TRV based on the data from these studies is likely to lead to overestimates of human health risks that must be put into

perspective. The U.S. EPA and Health Canada are currently reviewing the toxicological limits established for arsenic based upon many of the issues discussed above.

In summary, inorganic arsenic risk estimates that are generated through use of the currently available oral TRVs are believed to be overestimates of the actual level of risk. While regulatory agency oral TRVs based on the Taiwanese studies are the only TRVs currently available to use in HHRA (and were used in the current HHRA), arsenic risk estimates should be interpreted with great caution. A failure to account for the limitations of these TRVs could lead to an inappropriate conclusion of significant risks from inorganic arsenic exposure even when the concentrations are at background levels or below analytical detection limits. Alternatively, a weight-of-evidence approach has been successfully used at several other sites across Canada and the U.S. [Port Hope (OMOE, 1991); Deloro (OMOE, 1999); Wawa (OMOE, 2001a); Anaconda, Montana (Calabrese, unpublished; Hwang *et al.*, 1997a,b; Walker and Griffin, 1998); Balmerton (Gradient, 1985); Sudbury (SARA, 2008)]. Risk assessments involving multi-pathway exposure assessment (air, water, soil, backyard produce, fish and market basket foods) and use of the Health Canada and/or U.S. EPA slope factors revealed risk levels in the one-in-one thousand range for many of these sites. In fact, the results predicted for arsenic as part of the Flin Flon-Creighton HHRA are consistent with those obtained at other similar sites.

The unsatisfactory nature of these arguments carries directly across to the discussion of potential health outcomes related to arsenic entering the environment from various human activities. To the risk assessor, the concern is not necessarily focussed on what risks are predicted for the specific population of study, but the risks relative to background or typical populations. In the case of arsenic, risks well above the *de minimis* level are routinely predicted for exposures associated with typical North American diets, and high-quality, regulated North American drinking water supplies.

Further investigation into the risk assessment results for communities within the Flin Flon-Creighton area revealed the following:

- (i) Market basket foods and drinking water were significant contributors to arsenic related risks;
- (ii) Generic CCME criteria (12 µg/g) result in elevated risk levels (greater than one-in-one hundred thousand);
- (iii) The contribution of soil to overall arsenic-related risks was small relative to air and drinking water;
- (iv) Health-based PTCs, as determined by the risk assessment, are typically economically and technologically impossible to achieve; and,
- (v) Removal of all soil above the generic criteria would only result in a small overall risk reduction, with the assessment still predicting risks at the generic criteria level. It should be noted that the current Canadian drinking water standard for arsenic is based on treatment technology constraints, and this standard is currently under review and will likely be revised shortly in both the federal guideline and provincial standards. Once water treatment facilities are required to treat water to a lower standard, a significant source of arsenic exposure will be reduced, thus reducing the contribution of risk from arsenic exposure from non-soil related pathways.

It is clear that additional information, beyond that typically contained within a risk assessment, is needed to complete the decision making process, incorporating more of a weight-of-evidence approach. When considering potential exposures to arsenic from soils, the ionic species of arsenic (typically found in soils) forms insoluble salts with a number of cations and is adsorbed by organic matter, iron and aluminum oxides within the soil. Arsenic thus becomes tightly

bound to the soil and very difficult to liberate for biological uptake. Therefore, relatively high levels of arsenic in soil may pose little risk if they are indeed highly insoluble; and therefore, not available for absorption if ingested. In fact, the measured bioaccessibility of arsenic in the Flin Flon area soils was an average of 28% (or a 95% UCLM of 33%).

Studies in other regions of Canada and the U.S. where a weight of evidence approach was used to interpret the human health risks of environmental inorganic arsenic exposure have frequently reached the overall conclusions that soil remediation would not substantially reduce overall risks to local communities, and therefore soil remediation was concluded to be unwarranted. A selection of these studies include Port Hope (OMOE, 1991); Deloro (OMOE, 1999); Wawa (OMOE, 2001a); Balmertown (Gradient, 1995); and, Sudbury (SARA Group, 2008)]. Other studies, such as those conducted at the Anaconda, Montana site (Calabrese, unpublished; Hwang *et al.*, 1997a,b; Walker and Griffin, 1998), further support these conclusions, and recent regulatory announcements at this site have indicated soil remedial work will be undertaken only in areas where residential soils are greater than 250 µg/g arsenic (ATSDR, 2008a). Chronic exposure to soils less than this level were concluded to not be expected to result in adverse health effects (ATSDR, 2008a).

A comparison to a selection of these studies is instructive, in that arsenic soil concentrations and bioaccessibility data within the Flin Flon area are similar to, or less than those found in several of these studies. Table 5-10 provides summary information from a selection of previous Canadian studies for comparative purposes. For example, in the Town of Falconbridge, Ontario, mean and maximum soil arsenic concentrations of 79 µg/g and 400 µg/g were reported; N=118 (SARA Group, 2008). By comparison, the community of Creighton had the highest arsenic concentrations within the current assessment, with a mean soil inorganic arsenic concentration (N=183) of 67 µg/g and a maximum of 300 µg/g. Arsenic soil bioaccessibility for Falconbridge soils was higher than that determined for the soils in the current HHRA (see Table 5-10), and therefore, the arsenic in soils in Falconbridge is more readily taken into the body. The Sudbury Soils Study HHRA conducted with these data concluded no significant health risks exist beyond those predicted for a background area, and that remediation of arsenic in soil in Falconbridge would yield no significant reduction in risk levels (SARA Group, 2008). This conclusion was based on a weight of evidence, which included a comprehensive urinary arsenic monitoring program (see discussion below) and an epidemiological review of health statistics in the town and surrounding area. Urinary arsenic is a well-validated indicator of recent arsenic exposure. It was found that urinary arsenic levels in town residents were not significantly different from those in a control population with typical Ontario background levels of arsenic in soil (SARA Group, 2008).

Similar conclusions were also reached in a 1999 study of a town impacted by elevated levels of arsenic in soil due to former gold mining activity (*i.e.*, Deloro, Ontario). In Deloro, the mean and maximum soil inorganic arsenic concentrations were 111 µg/g and 605 µg/g; N=147 (OMOE, 1999). Bioaccessibility testing was not a common practice in 1999 and was not conducted; rather, a literature-based value of 14% for similar mine site soils was used. The HHRA conducted with these data concluded no significant health risks exist beyond those predicted for a background area, and that remediation of arsenic in soil in Deloro would yield no significant reduction in risk levels. In fact, it was concluded that even if all contaminated soils in Deloro were replaced with soils containing typical Ontario background concentrations, the overall human health risks would be reduced by only 2 to 4%. The Deloro study conclusions were based on a weight of evidence of the HHRA results, a comprehensive urinary arsenic monitoring program, and an epidemiological evaluation. It was found that urinary arsenic levels in town residents were not significantly different from those in a control population with typical Ontario background levels of arsenic in soil (see discussion below). The epidemiological review

of cancer incidence and mortality data in Deloro and surrounding areas concluded that for the cancers of interest, there was no incidence or mortality statistics that warranted further investigation or study.

Studies conducted in Balmertown, Ontario (Gradient, 1995) and Wawa Ontario (OMOE, 2001a) yielded comparable conclusions. In the Balmertown, Ontario study, mine tailings related to past gold mining activities present within parts of the town resulted in residential arsenic soil concentrations ranging up to 300 µg/g (Table 5-10). This study included soil, dust (limited sample numbers), home garden produce sampling, soil and dust bioaccessibility testing, as well as urinary arsenic analysis. The study concluded that exposure to inorganic arsenic in Balmertown was similar to exposure in reference communities unaffected by elevated levels of arsenic in soil and dust. They concluded that if soils were removed from the town, the total lifetime exposure reduction would be 3%. Therefore, the need for soil removal was not indicated (Gradient, 1995). Concentrations of arsenic in soils in Wawa, Ontario ranged from 21 to 175 µg/g within the town, with bioaccessibility of soils assumed to be near 50%. The urinary arsenic study (see below) provided evidence that arsenic exposures were not elevated in the community, and soil remedial work was not recommended.

In all of these studies, cancer risk estimates related to soil/dust exposure pathways were elevated when compared to regulatory benchmarks of 1: 100,000 or 1: 1,000,000 (depending upon the jurisdiction). These ILCRs from different studies are difficult to compare with confidence, due to possible differences in exposure assumptions, exposure pathways and even TRVs. Therefore, these were only considered qualitatively. Generally, the ILCRs between the sites were within the same order of magnitude for all communities (Table 5-10).

One of the most persuasive pieces of evidence associated with these other sites was the urinary arsenic biomonitoring studies. Normal total urinary arsenic levels are less than 50 µg/L in the absence of recent consumption of seafood that contains organic forms of arsenic (*i.e.*, fish arsenic) (ATSDR, 2008a). In Falconbridge, urinary arsenic testing was conducted in both the town of Falconbridge, and a reference community (Hanmer, Ontario). Urine was analyzed for total and inorganic arsenic, and its major metabolites [monomethylarsenic acid (MMA) and dimethylarsinic acid (DMA)]. A total of 369 urine samples were collected in Falconbridge, and 321 samples in Hanmer. An environmental risk questionnaire was also given, to provide insight into the possible roles of diet, occupational exposure, drinking water, *etc.*, to the urinalysis results. The mean inorganic arsenic concentrations in urine were nearly identical in these communities (7.1 µg/L in Falconbridge, and 7.2 µg/L in Hanmer). The distribution of urine concentrations within each of these communities was very similar; 80% of tested individuals in both communities were less than 10 µg/L inorganic arsenic in urine, with 3% in both of the background and Study area communities being above 20 µg/L. The average urinary arsenic levels in Falconbridge were very similar to those in the comparison community, both on an overall basis, and within the various age groups tested. It was concluded that Falconbridge residents were not incurring increased arsenic exposure, when compared to typical Canadians. As indicated in Table 5-10, soil concentrations in Falconbridge were, on average, similar to those measured in the Flin Flon study area, but the upper end of the concentration range was higher in Falconbridge. The measured bioaccessibility of arsenic in soils was higher in the Sudbury area, relative to that measured in Flin Flon soils (see Table 5-10).

Table 5-10 Weight of Evidence Information for Other Studies with Elevated Soil Arsenic Concentrations								
Study Location	Balmertown	Deloro	Wawa ^a	Sudbury Soils Study				Flin Flon/Creighton
				Coniston	Copper Cliff	Falconbridge	Sudbury Centre	
# Soil Samples (residential areas)	27	147	69	203	197	188	597	183
Arsenic Soil Concentration Range (µg/g)	13-544 (garden soil) 93-622 (yard soil) 52-483 (play areas)	2.4-605	21-175 (across both medium and high zones)	2.5-56	2.5-72	2.5-400	2.2-59	(maximum) West Flin Flon - 237 East Flin Flon - 33 Creighton - 300 Channing - 36
95 th %ile Arsenic Soil Concentration (µg/g)	NC	308	NC	29.3	41.5	205	17.4	NC
95% UCLM Arsenic Soil Concentration (µg/g)	NC	NC	NC	12.2	19	79	7.2	33-88
Mean Arsenic Soil Concentration (µg/g)	58 (garden soil) 214 (yard soil) 239 (play areas)	111	51 (medium zone) 133 (high zone)	9.5	17	69	6	16-67
Outdoor Soil Bioaccessibility (%)	15 (measured)	14 (assumed based on literature)	51 (assumed)	39 (measured)				33 (measured)
Indoor Dust Bioaccessibility (%)	14 (measured)	100 (assumed)	Not assessed	45 (measured)				33 (assumed)
Soil/Dust Ingestion and Dermal Contact	Risk Estimates not calculated in	2.9E-4 (max scenario) 3.3E-5	1.4E-5 (medium zone)	1.4E-5	1.7E-5	4.1E-5	1.2E-5	9.8E-6 – 3.3E-5

Table 5-10 Weight of Evidence Information for Other Studies with Elevated Soil Arsenic Concentrations								
Study Location	Balmertown	Deloro	Wawa ^a	Sudbury Soils Study				Flin Flon/Creighton
				Coniston	Copper Cliff	Falconbridge	Sudbury Centre	
ILCR	Study	(mean scenario)	3.6E-5 (high zone)					
Health / Epidemiology Review Conducted?	No	Yes	No	Yes				Yes
Urinary Arsenic Study?	Yes	Yes	Yes	No	No	Yes	No	On-going
Recommendation / Conclusion for arsenic in soil	No soil corrective action	No soil corrective action	No soil corrective action	No soil corrective action				To be determined

NC Not calculated by the investigators for the study, or not presented in available study documentation.
 a The information presented for Wawa is for medium and high impact zones that were investigated. Risk estimates presented are for the soil ingestion pathway only, which always greatly exceeds estimated exposures and risks from dermal contact. ILCRs are estimated from exposure estimates provided in the study report.

With respect to the Deloro urinary arsenic study, 80% of residents participated in the survey, and there was similar participation rate from the comparison community of Havelock, Ontario. As per the Falconbridge Study, an environmental risk questionnaire was also given, to provide insight into the possible roles of diet, occupational exposure, drinking water, etc., to the urinalysis results. With the high participation rates, the results are considered quite reliable for a study of this nature. Both total and speciated arsenic levels for Deloro and Havelock were well below those normally associated with adverse health effects. There were a handful of people (four in Deloro and one in Havelock) whose results for urinary arsenic were slightly above the normal range. Detailed analysis of the information provided by these residents showed no adverse health effects or unusual exposures. The levels of arsenic in urine (total and speciated) in Deloro residents were very similar to those in the comparison (non-exposed) community (4.36 µg/L in Deloro, versus 4.57 µg/L in Havelock). The distribution of urinary arsenic levels in Deloro residents was also very similar to the distribution in Havelock residents and there was no statistical difference in mean values for all the arsenic results between the two communities. The Ontario Ministry of Environment (OMOE, 1999) concluded that there was nothing unusual about any of the urinary arsenic findings, and, therefore arsenic exposure in Deloro was concluded to be similar to that of a community where no environmental arsenic exposure was expected. The data also indicated that there was no demonstrable relationship between arsenic levels in residential yards and garden soil and arsenic levels in people's urine (*i.e.*, higher arsenic concentrations in soil on a property did not lead to a higher urinary arsenic level in the resident(s) of that property). As noted in Table 5-10, soil concentrations in Deloro were notably higher than those found in the Flin Flon study area, albeit, the bioaccessibility of arsenic in the soils was assumed to be lower than that measured in Flin Flon.

In the Wawa, Ontario urinary arsenic study, 184 samples were taken. The mean value (5.62 µg/L) was similar to those reported in the reference community of Havelock (4.57 µg/L) and Deloro (4.36 µg/L). As indicated in Table 5-10, soil concentrations bracketed those measured in Flin Flon, with the high exposure area in Wawa being substantially higher than that measured in the Flin Flon Study area. Wawa bioaccessibility data were not measured, but was assumed to be higher than that reported in the Flin Flon study.

There are other studies published in the peer-reviewed scientific literature, which further illustrate that soil arsenic concentrations within the concentration range measured in Flin Flon do not have a significant impact on total exposure to arsenic, based on measured urinary arsenic concentrations (*e.g.*, Hwang *et al.*, 1997a,b; soils averaging 150 µg/g arsenic resulted in urinary arsenic concentrations averaging 7.5 µg/L; soils averaging 90 µg/g arsenic resulted in urinary arsenic concentrations averaging 7.1 µg/L).

The soil studies that were conducted in other regions of Canada used a weight of evidence approach to interpret the human health risks of environmental inorganic arsenic exposure. All studies determined that soil remediation was unnecessary. Consideration of all pieces of evidence (*i.e.*, the risk assessment, a review of the scientific literature and other similar studies, community health status, urinary arsenic study), and the relative strength of evidence associated with each of these elements, resulted in conclusions that there were no unsafe exposures or increased health effects associated with the observed arsenic levels in soil in these communities. Soil concentrations of inorganic arsenic reported in these other studies were frequently higher than those reported in the Flin Flon area, and soil bioaccessibility of arsenic within the soils in these studies was generally similar to, or higher than arsenic soil bioaccessibility in Flin Flon area soils.

The soil studies that were conducted in other regions of Canada used a weight of evidence approach to interpret the human health risks of environmental inorganic arsenic exposure. All

studies determined that soil remediation was unnecessary. Consideration of all pieces of evidence (*i.e.*, the risk assessment, a review of the scientific literature and other similar studies, community health status, urinary arsenic study), and the relative strength of evidence associated with each of these elements, resulted in conclusions that there were no unsafe exposures or increased health effects associated with the observed arsenic levels in soil in these communities. Each element of the weight of evidence provides strong, complementary lines of evidence to assist in the realistic evaluation of health risks associated with exposures to arsenic. Only after consideration of all pieces of evidence (*i.e.*, the risk assessment, a review of the scientific literature, community health status, urinary arsenic study), and the relative strength-of-evidence associated with each of these elements, was it possible to conclude that there were no unsafe exposures or increased health effects associated with the observed arsenic levels in soil in these communities.

The Community Health Status Assessment of Flin Flon and Creighton, completed by public health officials from Manitoba Health and Healthy Living and the Saskatchewan Ministry of Health found that the overall health status of the Flin Flon area population is as good if not better than the provincial averages for most of the indicators studied. It should be noted however, that non-melanoma skin cancers (NMSCs) had been excluded from the Community Health Status Assessment. This is generally consistent with international cancer statistics protocols and results from a lack of consistent reporting of these types of cancers. Given that one of the most common forms of cancer associated with arsenic exposure is skin cancer, the results of the Community Health Status Assessment may not have captured an increased incidence in all cancers that are possibly related to arsenic exposures in the Flin Flon area population relative to the Provincial averages.

Although the Community Health Status Assessment is an important component of a weight-of-evidence approach, the most powerful and persuasive piece of evidence in other studies was the urinary arsenic study results, which compare urinary arsenic levels of an impacted community with those of a control community. It is recommended that a similar study be undertaken for the Flin Flon area, focusing on homes in West Flin Flon and Creighton in which a significant number of homes included within the residential soil sampling program contained concentrations of arsenic in excess of the PTC as presented below.

Derivation of the PTC for Arsenic

The derivation of a health-based arsenic PTC (*i.e.*, soil concentration resulting in an ILCR of less than one-in-one hundred thousand indicting the need for further action such as a biomonitoring study or risk management plan) was completed using a modified version of the approach used by the CCME (1999) to derive the human health-based component of the Canadian Soil Quality Guideline for residential soils as follows:

$$PTC = \frac{RsD * BW}{AF * SIR} + BSC$$

where:

PTC	=	Provisional trigger concentration for arsenic (µg/g)
RsD	=	Risk specific dose (0.0067 µg/kg/day)
BW	=	Composite receptor body weight (63 kg)
AF	=	Absorption factor from gut (0.33 based on site-specific bioaccessibility study)
SIR	=	Composite receptor soil ingestion rate (0.02 g/day)
BSC	=	Background arsenic soil concentration (10 µg/g (CCME, 1996))

The risk specific dose (RsD) ($0.0067 \mu\text{g}/\text{kg}/\text{day}$) represents the dose that is associated with an ILCR of 1.0×10^{-5} and an oral slope factor of $0.0015 \mu\text{g}/\text{kg}/\text{day}^{-1}$ (i.e., $1.0 \times 10^{-5} \div 0.0015 \mu\text{g}/\text{kg}/\text{day}^{-1}$). Since the PTC is derived to be protective of an incremental cancer risk associated with soil exposure above the risks associated with background soil concentrations, the background soil concentration is an additive component of this calculation. Due to a paucity of information available to derive an arsenic soil concentration reflective of regional background levels in the Flin Flon area, the CCME (1996) Canadian background soil concentration was selected. Since the protection of an ILCR of 1.0×10^{-5} is based on a lifetime exposure, the body weight and soil ingestion rate for the lifetime composite receptor were used to derive the PTC. A soil PTC of $74 \mu\text{g}/\text{g}$ is derived for the Flin Flon-Creighton area using this approach.

A comparison of the arsenic PTC of $74 \mu\text{g}/\text{g}$ with the results of the residential soil sampling program indicates that 40 of the 183 properties sampled contained concentrations of arsenic in excess of the PTC (Table 5-11). All of these properties were located in West Flin Flon and Creighton.

	West Flin Flon	East Flin Flon	Creighton	Channing	Total
# of Properties Sampled	77	66	30	10	183
# of Properties >74 $\mu\text{g}/\text{g}$	30 (39%)	0	10 (33%)	0	40 (22%)

Figure 5-7 illustrates the maximum concentrations of arsenic in outdoor soil on individual properties relative to the PTC of $74 \mu\text{g}/\text{g}$.

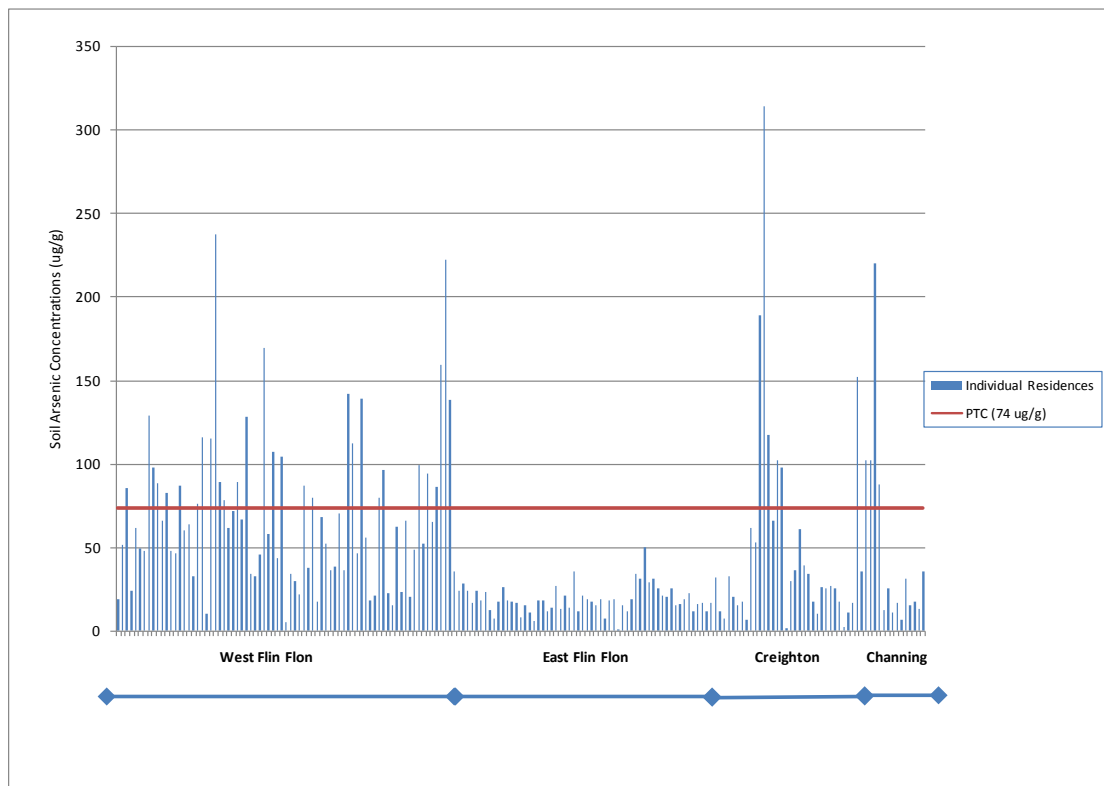


Figure 5-7 Maximum Concentrations of Arsenic on Individual Residential Properties Relative to the PTC of $74 \mu\text{g}/\text{g}$.

It is recommended that a urinary arsenic study be undertaken for the Flin Flon area, focusing on homes in West Flin Flon and Creighton in which a significant number of homes included within the residential soil sampling program contained concentrations of arsenic in excess of the PTC.

5.2.2 Cadmium

Estimated Exposure to Cadmium

The primary source of exposure to cadmium for residents of each COI was through the consumption of market basket food items. Market basket foods accounted for 66 to 74% of the total daily exposure for a toddler (Figure 5-8). Although market basket exposure was assumed to be the same for residents in each of the four COI, the percent contribution of market basket foods to total exposure differed slightly as a result of differences from exposure to community-specific environmental media concentrations (*i.e.*, soil, dust, air, and drinking water). Cadmium content in market basket food items is unrelated to environmental contamination in the Flin Flon area and is reflective of foods consumed throughout Canada and North America. Another significant source of exposure to cadmium was direct contact with soil and dust (*i.e.*, ingestion and dermal contact) representing 9 to 14% of the total exposure for toddlers varying from community to community based on the EPC soil concentrations. Drinking water, home garden produce, and wild game each contributed between 4 and 6% of the total cadmium exposure. Exposure *via* inhalation accounted for 2 to 4% in the communities of Flin Flon and Channing, and less than 1% in Creighton.

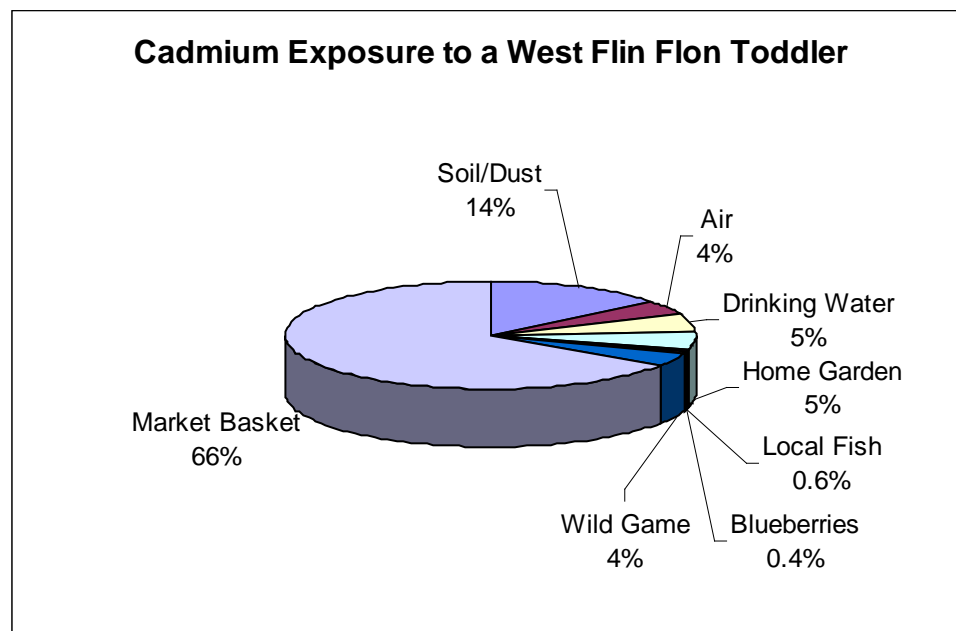


Figure 5-8 Contributions of Media to Total Cadmium Exposure for a Toddler Living in West Flin Flon

A detailed breakdown of the pathway-specific exposures for residents of West Flin Flon in each of the 5 age classes is provided in Table 5-12. Details for receptors in other communities are provided in Appendix M.

Exposure Pathway	Environmental Media Concentrations		Percent of Lifetime Total EDI	Estimated Daily Intakes (EDI) (µg/kg bw/day)					
	Value	Units		Infant	Toddler	Child	Teen	Adult	Lifetime
Inhalation of Fine Particulate	0.070	µg/m ³	5.0%	0.018	0.039	0.031	0.019	0.016	0.019
Dermal Contact – Outdoors	28	µg/g	0.013%	0.00011	0.000078	0.000059	0.000047	0.000045	0.000049
Dermal Contact – Indoors	22	µg/g	0.0035%	0.000033	0.000023	0.000016	0.000012	0.000012	0.000013
Outdoor Soil Ingestion	28	µg/g	2.9%	0.045	0.090	0.011	0.006	0.005	0.011
Indoor Dust Ingestion	22	µg/g	1.1%	0.018	0.035	0.0044	0.0024	0.0021	0.0043
Home Garden Root Vegetables	0.051	µg/g ww	0.63%	0.00099	0.0044	0.0037	0.0028	0.0020	0.0024
Home Garden Other Vegetables	0.24	µg/g ww	6.8%	0.041	0.043	0.036	0.024	0.023	0.025
Local Wild Blue Berries	0.048	µg/g ww	0.95%	0.0035	0.0035	0.0035	0.0035	0.0035	0.0035
Local Wild Game	0.079	µg/g ww	9.6%	0	0.036	0.036	0.036	0.036	0.036
Local Fish	0.0084	µg/g ww	1.5%	0	0.0056	0.0056	0.0056	0.0056	0.0056
Drinking Water	1.3	µg/L	7.7%	0.048	0.047	0.032	0.022	0.028	0.029
Market Basket Contribution	NA	µg/g	64%	0.58	0.58	0.46	0.26	0.18	0.24
Summary									
Estimated Daily Intake (µg/kg/day)			--	0.75	0.89	0.62	0.38	0.30	0.37
Inhalation Route Only			5.0%	0.018	0.039	0.031	0.019	0.016	0.019
Direct Soil/Dust Contact			4.1%	0.063	0.126	0.016	0.0087	0.007	0.015
Market Basket Foods			64%	0.58	0.58	0.46	0.26	0.18	0.24
Drinking Water			7.7%	0.048	0.047	0.032	0.022	0.028	0.029
Local Foods (Home Garden, Blueberries, Wild Game and Fish)			19%	0.045	0.093	0.085	0.072	0.070	0.073

Hazard Quotients for Cadmium

Health Canada (2008) has recommended an oral TDI of 1.0 µg/kg/day to be protective of adverse effects such as renal dysfunction. Exposure of cadmium *via* inhalation may also result in adverse effects in kidney function. Therefore, since exposure resulting from inhalation can contribute to the total internal dose, this contribution is included in the total exposure and comparison to the oral TDI. Exposure of cadmium *via* inhalation may also elicit cancer in respiratory tissues. This will be assessed through the application of a cancer unit risk value to the ambient air EPCs as presented below.

HQ estimates for residents living in each of the COI ranged from 0.28 to 0.89, with the highest HQs predicted for residents of West Flin Flon (Table 5-13). All HQs were below the acceptable HQ value of 1.0 based on a TDI of 1.0 µg/kg/day. HQs were derived using the EPC for soil, dust, drinking water and ambient air selected to be representative of typical exposure to residents in each of the COI. Receptors living at locations with concentrations of cadmium in soil and dust that are higher or lower than the EPC are subject to risks that are subsequently higher or lower than those predicted for the general community population. HQ estimates for the toddler are higher than those predicted for other receptors as a result of the elevated soil ingestion rate assumed for children of this age.

Table 5-13 Predicted Hazard Quotients from Exposure to Cadmium

Receptor	East Flin Flon	West Flin Flon	Creighton	Channing	Typical Background
Infant	0.72	0.75	0.70	0.72	0.58
Toddler	0.82	0.89	0.79	0.81	0.58
Child	0.60	0.62	0.58	0.60	0.46
Teen	0.37	0.38	0.35	0.36	0.26
Adult	0.29	0.30	0.28	0.29	0.18

Due to the significant contribution of market basket foods to the total cadmium exposure, predicted risk levels for receptors living in each of the COI are similar to those predicted under the Typical Background scenario (Figure 5-9).

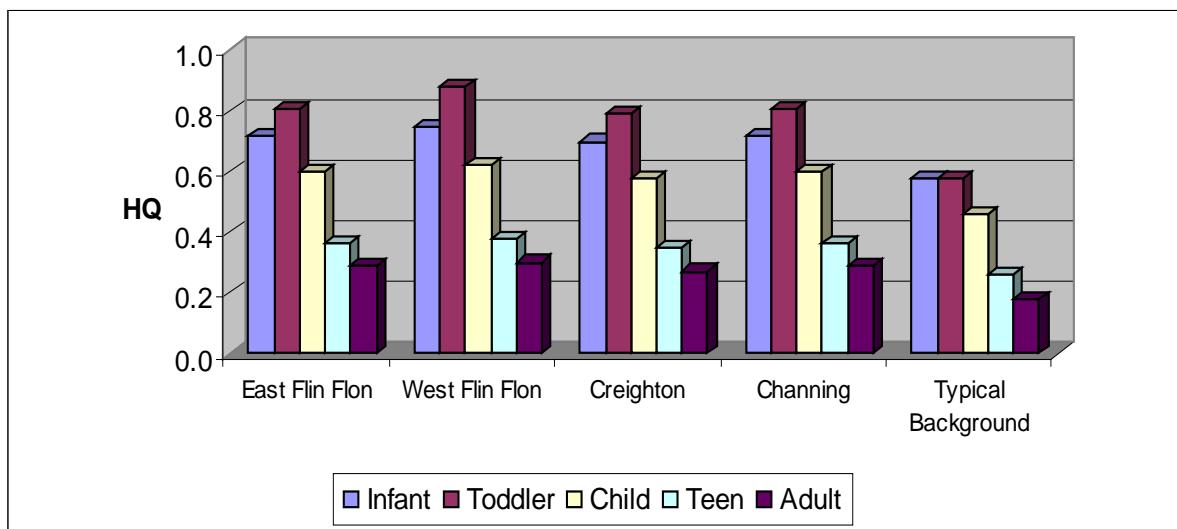


Figure 5-9 Predicted Hazard Quotients as a Result of Exposure to Cadmium

The difference in exposure to cadmium between residents of the COI and the Typical Background scenario can be attributed primarily to concentrations in soil which are notably higher in each of the COI relative to background. Accordingly, the difference in risk between residents of the COI and the Typical Background scenario can primarily be attributed to direct soil/dust pathways (Figure 5-10). Contribution of risk from exposure through the consumption of local foods (*i.e.*, wild game, fish, blueberries, and home garden vegetables) and drinking water is notable but minor relative to the total risk level.

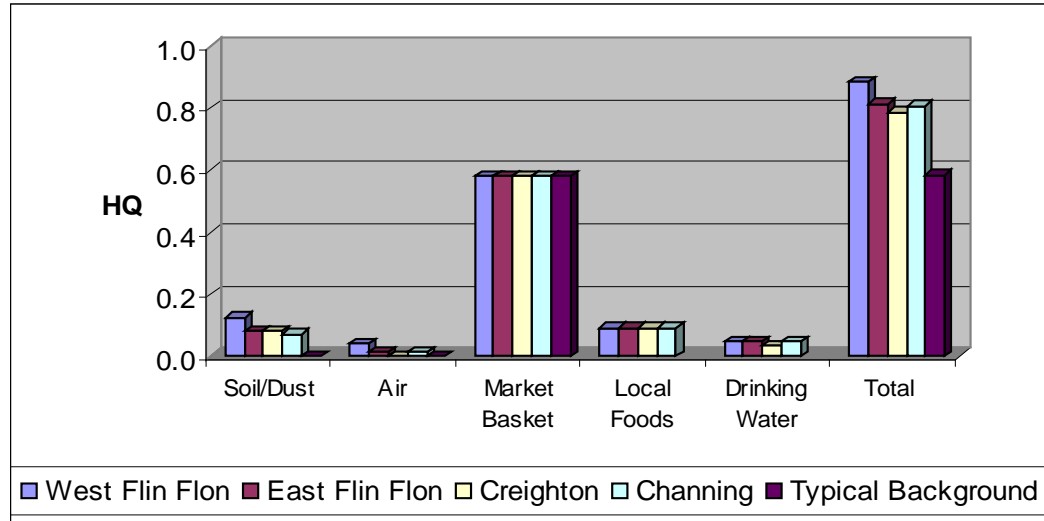


Figure 5-10 Pathway-Specific Hazard Quotients for a Toddler in each COI as a Result of Exposure to Cadmium

Therefore, using the community-based EPCs for cadmium-impacted environmental media, the predicted daily exposures for receptors living in each of the COI are not anticipated to result in the occurrence of unacceptable non-cancer health risks. However, given that concentrations of cadmium in soil are highly variable throughout each of the COI, consideration should be given on a property by property basis. This is discussed further following the derivation of the soil PTC. The cancer risk levels associated with the inhalation of cadmium are discussed below.

Incremental Lifetime Cancer Risk Estimates for Cadmium

Exposure to cadmium *via* inhalation pathways has been associated with an increased incidence of cancerous lung tumours. An inhalation unit risk value of $0.0098 (\mu\text{g}/\text{m}^3)^{-1}$ was applied to the EPC for cadmium in air in each of the COI to derive the community-specific ILCR (Table 5-14).

	<i>East Flin Flon</i>	<i>West Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>
EPC ($\mu\text{g}/\text{m}^3$)	0.026	0.070	0.0046	0.026
ILCR	2.6E-04	6.9E-04	4.5E-05	2.6E-04

Bolded values highlighted in grey exceed the acceptable ILCR of 1.0×10^{-5} .

ILCRs were above the acceptable risk level of 1.0×10^{-5} (or one-in-one hundred thousand) in each of the four COI, but are considered to represent a low carcinogenic risk level in East and West Flin Flon and Channing, and a very low risk level in Creighton. These estimates conservatively assume that concentrations of cadmium in indoor air are equal to those measured in ambient outdoor air. Although this may result in an overestimation of exposure

while spending time indoors, this assumption is necessary given the absence of indoor air measurements. Since there are no air monitoring stations located in the communities of East Flin Flon or Channing, it was assumed that receptors in these communities would be exposed to concentrations measured at Ruth Betts. Based on technical advice provided by Manitoba Conservation and HBMS regarding prevailing wind directions, it is anticipated that this assumption will result in an overestimation of risks to residents of East Flin Flon and Channing.

The HHRA used concentrations of cadmium associated with the PM₁₀ component of ambient air (which is lower than the concentration associated with TSP) to characterize exposure. The Health Canada cancer unit risk value is designed to be protective of cancer during a lifetime exposure. To be protective of an ILCR of 1.0×10^{-5} , the unit risk value of $0.0098 \text{ } (\mu\text{g}/\text{m}^3)^{-1}$ is equivalent to an air concentration of $0.001 \text{ } \mu\text{g}/\text{m}^3$ (*i.e.*, $1.0 \times 10^{-5} \div 0.0098 \text{ } (\mu\text{g}/\text{m}^3)^{-1}$). Overall, using the Health Canada cancer unit risk value indicates that concentrations of cadmium in ambient air may have the potential to result in an unacceptable increase in the risk of developing lung cancer for receptors spending a significant portion of their lifetime living in the Flin Flon area.

Due to the conservatism associated with the derivation of unit risk values and the acceptable cancer risk level of 1.0×10^{-5} , it is recommended that the results of the Community Health Assessment also be considered. It is noted that the Community Health Status Assessment would likely not be sensitive enough to detect an increase in cancer of the magnitude predicted in the HHRA given that the population of interest is only 8 to 9,000; however, it does provide some indication of current lung cancer incidence. That said, ILCR for cadmium are elevated and consideration should be given to future reductions in smelter-related emissions, which would have a direct and immediate effect on reducing inhalation-related exposure and risks.

Derivation of a Residential Cadmium Soil PTC

Although exposure to cadmium through the inhalation of particulates in air and from the consumption of drinking water may vary from community to community, these sources have a relatively minor contribution to total cadmium intake. The exposure to cadmium through the consumption of market basket foods is notably higher; however, contribution from this source is anticipated to be similar throughout each of the four COI. It can therefore be reasoned that the derivation of a single cadmium soil PTC should be applicable to all residential properties in the four COI. Since concentrations of cadmium in air are highest in West Flin Flon, environmental parameters associated with this community were used to back-calculate a soil PTC that is protective of an HQ of 1.0. An iterative process was used in which exposure to cadmium from air, drinking water, local foods, and market basket foods remained constant while the concentration in soil was adjusted to produce a total EDI of $1 \text{ } \mu\text{g}/\text{kg}/\text{day}$ and an HQ of 1.0. Since concentrations of cadmium in indoor dust are a function of the concentrations in outdoor soil, dust concentrations were automatically adjusted during this process.

Given that the TDI is $1 \text{ } \mu\text{g}/\text{kg}/\text{day}$ and the predicted exposure from market basket foods is $0.58 \text{ } \mu\text{g}/\text{kg}/\text{day}$ for the toddler, the residual TDI (RTDI) to be allocated to exposure from soil/dust, drinking water, local foods, and air is $0.42 \text{ } \mu\text{g}/\text{kg}/\text{day}$. The predicted daily exposure of a toddler living in West Flin Flon *via* drinking water ($0.047 \text{ } \mu\text{g}/\text{kg}/\text{day}$), local foods ($0.093 \text{ } \mu\text{g}/\text{kg}/\text{day}$), and air ($0.039 \text{ } \mu\text{g}/\text{kg}/\text{day}$) totals $0.18 \text{ } \mu\text{g}/\text{kg}/\text{day}$. Assuming that exposure to cadmium remains constant from all sources other than soil/dust, the RTDI to be allocated to soil/dust *via* ingestion and dermal contact is $0.24 \text{ } \mu\text{g}/\text{kg}/\text{day}$ for the toddler. Conservatively assuming 100% bioaccessibility of cadmium in soil, a soil concentration of $58 \text{ } \mu\text{g}/\text{g}$ (and an associated indoor dust concentration of $33 \text{ } \mu\text{g}/\text{g}$) produces an exposure of $1.0 \text{ } \mu\text{g}/\text{kg}/\text{day}$ and results in a total HQ of 1.0 (Table 5-15).

<i>Media</i>	<i>Exposure (µg/kg/day)</i>	<i>Media-Specific HQ</i>
Air	0.039	0.039
Drinking Water	0.047	0.047
Local Fish	0.0049	0.0049
Local Wild Game	0.036	0.036
Blue Berries	0.0038	0.0038
Home Garden Vegetables	0.048	0.048
Market Basket	0.58	0.58
EDI without Soil/Dust	0.76	0.76
TDI	1.0	--
RTDI allocated to Soil/Dust	0.24	0.24

A comparison of the cadmium soil PTC of 58 µg/g with the results of the residential soil sampling program indicates that 2 of the 183 properties sampled contained a maximum concentration of cadmium (58.1 and 70.8 µg/g) in excess of the residential soil PTC (Table 5-16). Both of these locations were located in the community of West Flin Flon. Although these concentrations are above the soil PTC, the HQs associated with these concentrations are still equal to an acceptable value of 1.0. As a result, levels of cadmium in Flin Flon area residential soils are not anticipated to pose health risks to human health within any of the COI.

	<i>West Flin Flon</i>	<i>East Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>	<i>Total</i>
# of Properties Sampled	77	66	30	10	183
# of Properties >58 µg/g	2 (2.6%)	0	0	0	2 (1.1%)

Overall, the non-cancer health risks to Flin Flon area residents associated with exposure to cadmium are within risk levels deemed to be acceptable by Health Canada and the CCME. However, concentrations of cadmium measured in ambient outdoor air may result in an increase in the risk of developing lung cancer. This assumes that residents would be exposed to the current levels of cadmium in ambient outdoor for a lifetime and that concentrations in indoor air would be similar to those measured in outdoor air. Any future increase or decrease in concentrations of cadmium released from the smelter would subsequently increase or decrease the estimated upper bound cancer risk level.

5.2.3 Copper

Estimated Exposure to Copper

The primary source of exposure to copper for residents of each COI was through the consumption of market basket food items. Market basket foods accounted for 59 to 81% of the total daily exposure for a toddler within each COI (Figure 5-11). Although market basket exposure was assumed to be the same for residents in each of the four COI, the percent contribution of market basket foods to total exposure differed slightly as a result of differences from exposure to community-specific environmental media concentrations (*i.e.*, soil, dust, air, and drinking water). Copper content in market basket food items is unrelated to environmental contamination in the Flin Flon area and is reflective of foods consumed throughout Canada and North America.

Another significant source of exposure to copper was drinking water, representing approximately 25% of the total exposure for toddlers in Flin Flon and Channing. As a result of lower concentrations of copper in Creighton drinking water, this pathway only represented approximately 7% of the total copper exposure to toddlers in Creighton. Direct exposure to soil and dust accounted for 5 to 15% of the total daily copper exposure for toddlers, varying from community to community based on the EPC soil concentrations. The contribution of copper from air, home garden vegetables, local blueberries, local fish, and wild game was minor.

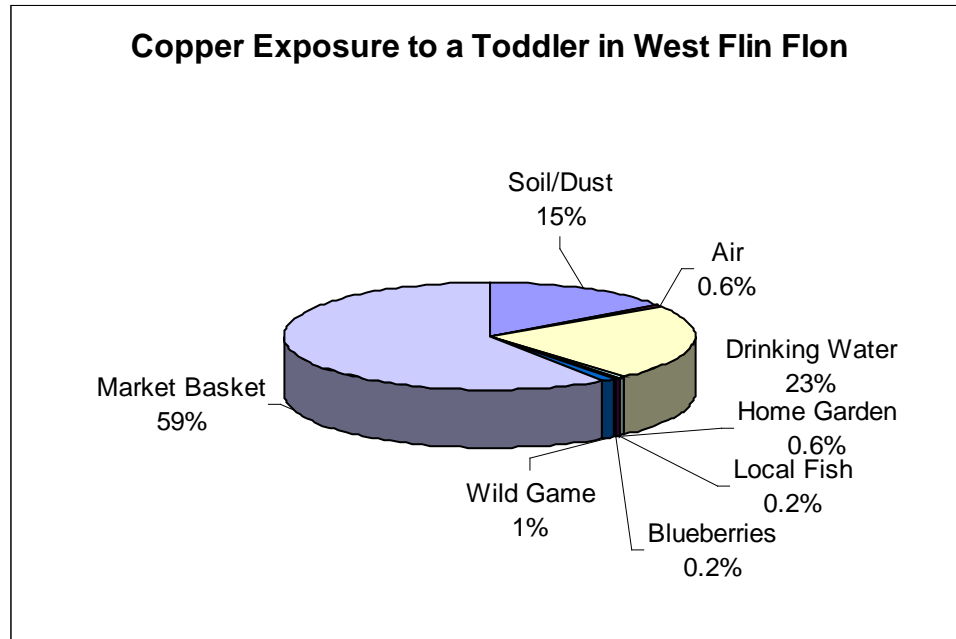


Figure 5-11 Contributions of Media to Total Copper Exposure for a Toddler Living in West Flin Flon

A detailed breakdown of the pathway-specific exposures for residents of West Flin Flon in each of the 5 age classes is provided in Table 5-17. Details for receptors in other communities are provided in Appendix M.

Exposure Pathway	Environmental Media Concentrations		Percent of Lifetime Total EDI	Estimated Daily Intakes (EDI) (µg/kg bw/day)					
	Value	Units		Infant	Toddler	Child	Teen	Adult	Lifetime
Inhalation of Fine Particulate	0.84	µg/m ³	0.65%	0.22	0.47	0.37	0.22	0.19	0.22
Dermal Contact – Outdoors	2,800	µg/g	0.014%	0.011	0.0078	0.0059	0.0047	0.0045	0.0049
Dermal Contact – Indoors	2,300	µg/g	0.0041%	0.0036	0.0025	0.0017	0.0013	0.0013	0.0014
Outdoor Soil Ingestion	2,800	µg/g	3.2%	4.5	9.0	1.1	0.62	0.53	1.1
Indoor dust Ingestion	2,300	µg/g	1.3%	1.9	3.8	0.47	0.26	0.22	0.46
Home Garden Root Vegetables	1.6	µg/g ww	0.22%	0.031	0.14	0.12	0.089	0.063	0.074
Home Garden Other Vegetables	2.0	µg/g ww	0.62%	0.34	0.36	0.30	0.20	0.19	0.21
Local Wild Blue Berries	2.1	µg/g ww	0.45%	0.15	0.15	0.15	0.15	0.15	0.15
Local Wild Game	2.0	µg/g ww	2.6%	0	0.92	0.91	0.90	0.91	0.90
Local Fish	0.31	µg/g ww	0.61%	0.00	0.21	0.21	0.21	0.21	0.21
Drinking Water	520	µg/L	33%	19	19	13	8.7	11	11
Market Basket Contribution	NA	µg/g	57%	51	49	36	21	15	19
Summary									
Estimated Daily Intake (µg/kg/day)			--	77	83	52	33	28	34
Inhalation Route Only			0.65%	0.22	0.47	0.37	0.22	0.19	0.22
Direct Soil/Dust Contact			4.6%	6.5	13	1.6	0.89	0.75	1.6
Market Basket Foods			57%	51	49	36	21	15	19
Drinking Water			33%	19	19	13	8.7	11	11
Local Foods (Home Garden, Blueberries, Wild Game and Fish)			4.5%	0.53	1.8	1.7	1.6	1.5	1.5

Hazard Quotients

HQ estimates for residents living in each of the COI ranged from 0.19 to 0.93, with the highest HQs predicted for residents of West Flin Flon (Table 5-18). All HQs were below the acceptable HQ value of 1.0. These HQs were derived using the EPC for soil and dust selected to be representative of typical exposure to residents in each of the COI. Receptors living at locations with concentrations of copper in soil and dust that are higher or lower than the EPC are anticipated to be subject to risks that are subsequently higher or lower than those predicted for the general community population. HQs for the toddler are higher than those predicted for other receptors as a result of the elevated soil ingestion rate assumed for children of this age.

<i>Receptor</i>	<i>East Flin Flon</i>	<i>West Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>	<i>Typical Background</i>
Infant	0.81	0.86	0.65	0.81	0.58
Toddler	0.84	0.93	0.68	0.83	0.58
Child	0.51	0.52	0.41	0.51	0.38
Teen	0.32	0.33	0.26	0.32	0.23
Adult	0.28	0.28	0.19	0.28	0.16

Due to the significant contribution of market basket foods to the total copper exposure, predicted risk levels for receptors living in each of the COI are less than two-fold higher than those predicted under the Typical Background scenario (Figure 5-12).

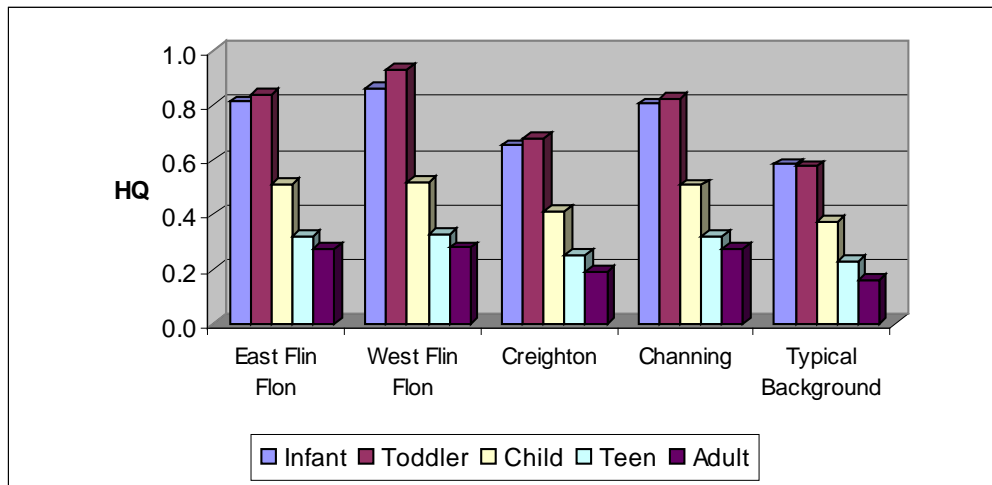


Figure 5-12 Predicted Hazard Quotients as a Result of Exposure to Copper

Exposure to copper *via* inhalation may elicit adverse effects within sensitive respiratory tissues in addition to its contribution to the total internal dose which can result in hepatotoxicity and gastrointestinal effects. As a result, the contribution of exposure *via* inhalation was included in the total multimedia assessment using the oral TDI, as well as through a comparison of the ambient air EPC to the air standard (1 µg/m³) protective of adverse effects to respiratory tissues during chronic exposure. This comparison produces a concentration ratio (CR). Concentration ratios that are at or below 1.0 are considered to be acceptable. This assessment indicated that concentrations of copper in ambient air in each of the COI are below the air standard protective of adverse respiratory effects (Table 5-19).

	<i>East Flin Flon</i>	<i>West Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>
EPC ($\mu\text{g}/\text{m}^3$)	0.22	0.84	0.058	0.22
CR	0.22	0.84	0.058	0.22

The difference in exposure to copper between residents of the COI and the Typical Background scenario can be attributed primarily to concentrations in soil and drinking water which are notably higher in each of the COI. Accordingly, the difference in HQ estimates between residents of the COI and the Typical Background scenario can primarily be attributed to direct soil/dust pathways, and the consumption of drinking water in communities receiving municipal water supply from Flin Flon (Figure 5-13). For receptors living in West Flin Flon, CRs associated with the inhalation of copper in air are higher than experienced by receptors in other communities but are still lower than the acceptable risk level. Contribution to the HQ from exposure through the consumption of local foods (*i.e.*, wild game, fish, blueberries, and home garden vegetables) is insignificant relative to the total HQ.

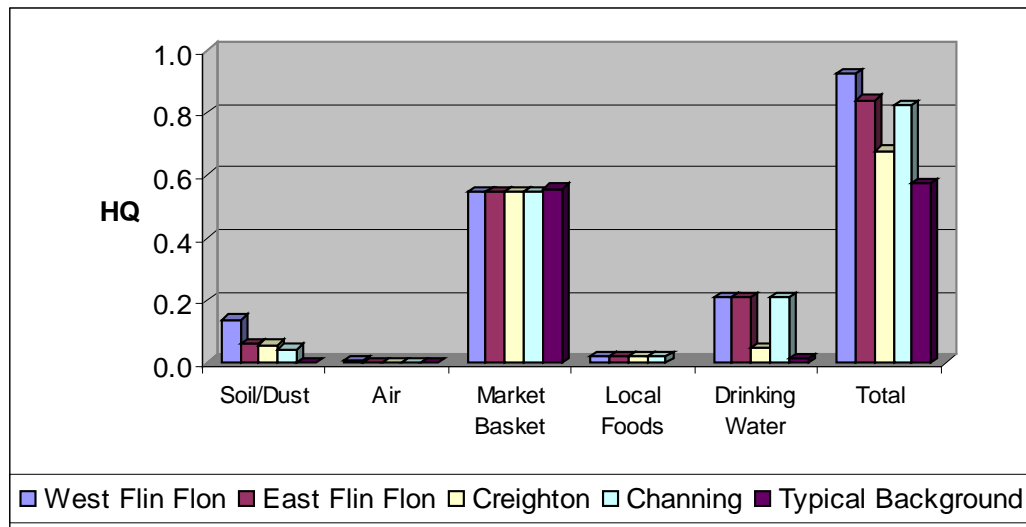


Figure 5-13 Pathway-Specific Hazard Quotients for a Toddler in each COI as a Result of Exposure to Copper

Therefore, using the community-based EPCs for copper-impacted environmental media, the predicted daily exposures for receptors living in each of the COI are not anticipated to result in the occurrence of unacceptable health risks. However, given that concentrations of copper in soil are highly variable throughout each of the COI, consideration should be given on a property by property basis. This is discussed further following the derivation of the residential soil PTC.

Derivation of a Residential Copper Soil PTC

Exposure to copper through the consumption of market basket foods is a significant source. Contribution from this pathway is assumed to be similar throughout each of the four COI. Since concentrations of copper in drinking water are higher in Flin Flon, and concentrations in air are highest in West Flin Flon, environmental parameters associated with the community of West Flin Flon were used to back-calculate a soil PTC that is protective of an HQ of 1.0. This value is considered to be protective of receptors living within each of the COI. An iterative process was used in which exposure to copper from air, drinking water, local foods, and market basket foods

remained constant while the concentration in soil was adjusted to produce a total EDI of 90 µg/kg/day and an HQ of 1.0. Since concentrations of copper in indoor dust are a function of the concentrations in outdoor soil, dust concentrations were automatically adjusted during this process.

Given that the TDI for the toddler is 90 µg/kg/day and the predicted exposure from market basket foods is 49 µg/kg/day, the RTDI to be allocated to exposure from soil/dust, drinking water, local foods, and air is 41 µg/kg/day. The predicted daily exposure of a toddler living in West Flin Flon *via* drinking water (19 µg/kg/day), local foods (1.8 µg/kg/day), and air (0.47 µg/kg/day) totals 21 µg/kg/day. Assuming that exposure to copper remains constant from all sources other than soil/dust, the RTDI to be allocated to soil/dust *via* ingestion and dermal exposure is 20 µg/kg/day for the toddler. Conservatively assuming 100% bioaccessibility of copper in soil, a soil concentration of 5,000 µg/g (and an associated indoor dust concentration of 3,200 µg/g) produces an exposure of 20 µg/kg/day and results in a total HQ of 1.0 (Table 5-20).

<i>Media</i>	<i>Exposure (µg/kg/day)</i>	<i>Media-Specific HQ</i>
Air	0.47	0.0053
Drinking Water	19	0.21
Local Fish	0.21	0.0023
Local Wild Game	0.92	0.010
Blue Berries	0.15	0.0017
Home Garden Vegetables	0.50	0.0055
Market Basket	49	0.55
EDI without Soil/Dust	70	0.78
TDI	90	-
RTDI allocated to Soil/Dust	20	0.22

A comparison of the copper soil PTC of 5,000 µg/g with the results of the residential soil sampling program indicates that only 5 of the 183 properties sampled were in excess of the soil PTC (Table 5-21). All of these locations were in the community of West Flin Flon. Although these properties each contained a maximum concentration (5,260, 5,360, 5,470, 5,530 and 7,810 µg/g) in excess of the PTC, the HQs associated with these concentrations range from 1.0 to 1.1 assuming chronic exposure to these levels. Given this, and the minor contribution of soil and dust to the total daily copper exposure, levels of copper in Flin Flon area soils are not anticipated to pose health risks to human health within any of the COI.

	<i>West Flin Flon</i>	<i>East Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>	<i>Total</i>
# of Properties Sampled	77	66	30	10	183
# of Properties >5,000 µg/g	5 (6.5%)	0	0	0	5 (2.7%)

Overall, no health risks are anticipated to Flin Flon area residents as a result of exposure to copper. Risk management measures or soil remediation are not considered to be necessary to prevent or reduce human health risks associated with exposure to copper in residential soils.

5.2.4 Lead

The assessment of lead exposure was completed using the excel-based HHRA exposure model used for all COC as well as the U.S. EPA IEUBK model. The Health Canada TRV of 3.6 µg/kg/day was used to assess risks based on the exposure predicted using the HHRA exposure model. The assessment completed using the IEUBK model predicted the probability of exceeding both a BLL of 10 µg/dL, as well as a BLL of 5 µg/dL. The BLL of 5 µg/dL has more recently been suggested to be the level at which a decrease in cognitive ability may occur in young children and is the level currently in use in Ontario. Blood lead monitoring data is the most effective indication of recent exposure levels to lead from all sources. Given that blood lead data for the Flin Flon area is not available, the IEUBK model was used as an additional tool as it is widely acknowledged as a very effective method of assessing risks to young children from exposure to lead. As such, the IEUBK results were considered in the derivation of the PTC for lead. The results of both assessments are presented in the following sections.

5.2.4.1 Exposure and Risk Estimated using the HHRA Exposure Model

Estimated Exposure to Lead

The primary source of exposure to lead for residents of each COI was through direct soil/dust pathways (*i.e.*, incidental ingestion and dermal contact) and through the consumption of market basket food items. For the toddler, incidental ingestion of soil/dust accounted for 37 to 45% of the total daily exposure (Figure 5-14). Differences in the contribution of soil and dust to the total daily lead exposure is a reflection of the different concentrations of lead measured in COI-specific soils. Market basket foods accounted for 32 to 42% of the total daily exposure for toddlers. Although market basket exposure was assumed to be the same for residents in each of the four COI, the percent contribution of market basket foods to total exposure differed as a result of differences from exposure to community-specific environmental media concentrations (*i.e.*, soil, dust, air, and drinking water). Lead content in market basket food items is unrelated to environmental contamination in the Flin Flon area and is reflective of foods consumed throughout Canada and North America. The contribution of lead ingested in drinking water was also notable, representing 7 to 10% of the total daily exposure for toddlers. In the community of West Flin Flon, exposure to lead *via* inhalation was also a notable source (9% of the total daily exposure for toddlers).

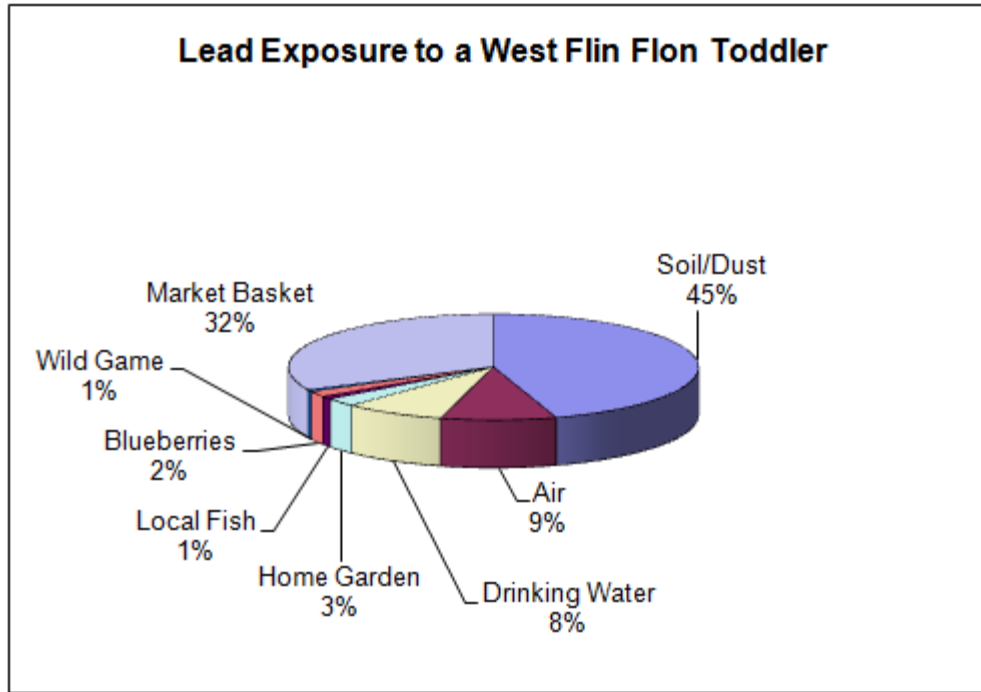


Figure 5-14 Contributions of Media to Total Lead Exposure for a Toddler Living in West Flin Flon as Predicted by the HHRA Model

A detailed breakdown of the pathway-specific exposures for residents of West Flin Flon in each of the five age classes is provided in Table 5-22. Details for receptors in other communities are provided in Appendix M.

Table 5-22 Estimated Total Daily Intakes for Lead – West Flin Flon

<i>Exposure Pathway</i>	<i>Environmental Media Concentrations</i>		<i>Percent of Lifetime Total EDI</i>	<i>Estimated Daily Intakes (EDI) (µg/kg bw/day)</i>					
	<i>Value</i>	<i>Units</i>		<i>Infant</i>	<i>Toddler</i>	<i>Child</i>	<i>Teen</i>	<i>Adult</i>	<i>Lifetime</i>
Inhalation of Fine Particulate	0.34	µg/m ³	14%	0.087	0.19	0.15	0.090	0.076	0.090
Dermal Contact – Outdoors	370	µg/g	0.61%	0.0084	0.0062	0.0047	0.0038	0.0036	0.0039
Dermal Contact – Indoors	265	µg/g	0.15%	0.0024	0.0017	0.0012	0.00091	0.00086	0.00095
Outdoor Soil Ingestion	370	µg/g	13%	0.35	0.69	0.087	0.048	0.040	0.084
Indoor Dust Ingestion	265	µg/g	4.7%	0.13	0.25	0.031	0.017	0.015	0.030
Home Garden Root Vegetables	0.033	µg/g ww	0.24%	0.00064	0.0028	0.0024	0.0018	0.0013	0.0015
Home Garden Other Vegetables	0.28	µg/g ww	4.7%	0.047	0.050	0.042	0.028	0.027	0.030
Local Wild Blue Berries	0.51	µg/g ww	5.9%	0.038	0.037	0.037	0.038	0.038	0.037
Local Wild Game	0.025	µg/g ww	1.8%	0.0	0.012	0.011	0.011	0.011	0.011
Local Fish	0.031	µg/g ww	3.3%	0.0	0.021	0.021	0.021	0.021	0.021
Drinking Water	4.6	µg/L	16%	0.17	0.17	0.11	0.077	0.10	0.10
Market Basket Contribution	NA	µg/g	36%	0.77	0.68	0.43	0.24	0.16	0.23
Summary									
Estimated Daily Intake (µg/kg/day)			--	1.6	2.1	0.93	0.58	0.49	0.64
Inhalation Route Only			14%	0.087	0.19	0.15	0.090	0.076	0.090
Direct Soil/Dust Contact			19%	0.48	0.95	0.12	0.070	0.059	0.12
Market Basket Foods			36%	0.77	0.68	0.43	0.24	0.16	0.23
Drinking Water			16%	0.17	0.17	0.11	0.077	0.10	0.10
Local Foods (Home Garden, Blueberries, Wild Game and Fish)			16%	0.086	0.12	0.11	0.099	0.098	0.10

Hazard Quotients

HQ estimates for residents living in each of the COI ranged from 0.10 to 0.59, with the highest HQs predicted for residents of West Flin Flon (Table 5-23). All HQs were below the acceptable HQ value of 1.0. These HQs were derived using the EPC for soil and dust selected to be representative of typical exposure to residents in each of the COI. Receptors living at locations with concentrations of lead in soil and dust that are higher or lower than the EPC are anticipated to be subject to risks that are subsequently higher or lower than those predicted for the general community population. HQs for the toddler are higher than those predicted for other receptors as a result of the elevated soil ingestion rate assumed for children of this age.

<i>Receptor</i>	<i>East Flin Flon</i>	<i>West Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>	<i>Typical Background</i>
Infant	0.38	0.44	0.38	0.38	0.24
Toddler	0.45	0.59	0.46	0.45	0.21
Child	0.22	0.26	0.20	0.22	0.14
Teen	0.14	0.16	0.13	0.14	0.078
Adult	0.12	0.14	0.10	0.12	0.058

Due to the significant contribution of market basket foods to the total lead exposure, predicted HQs for receptors living in each of the COI are generally only two-fold higher than those predicted under the Typical Background scenario (Figure 5-15).

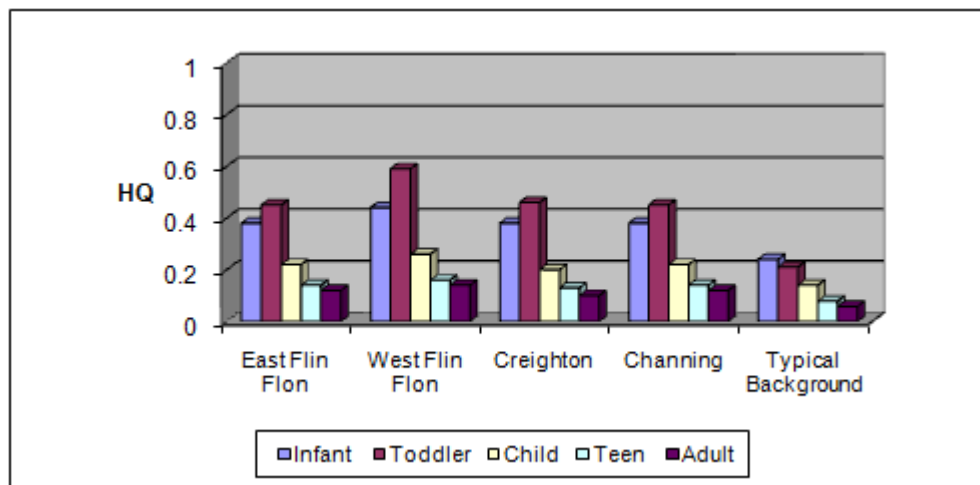


Figure 5-15 Predicted Hazard Quotients as a Result of Exposure to Lead

The difference in exposure to lead between residents of the COI and the Typical Background conditions can be attributed primarily to concentrations in soil which are notably higher in the COI. Accordingly, the difference in HQs between residents of the COI and the Typical Background scenario can primarily be attributed to direct soil/dust pathways. For receptors living in West Flin Flon, HQs associated with the inhalation of lead in air are higher than experienced by receptors in other communities but are still significantly lower than the acceptable risk level. Contribution from exposure through the consumption of local foods (*i.e.*, wild game, fish, blueberries, and home garden vegetables) is minor relative to the total HQ (Figure 5-16).

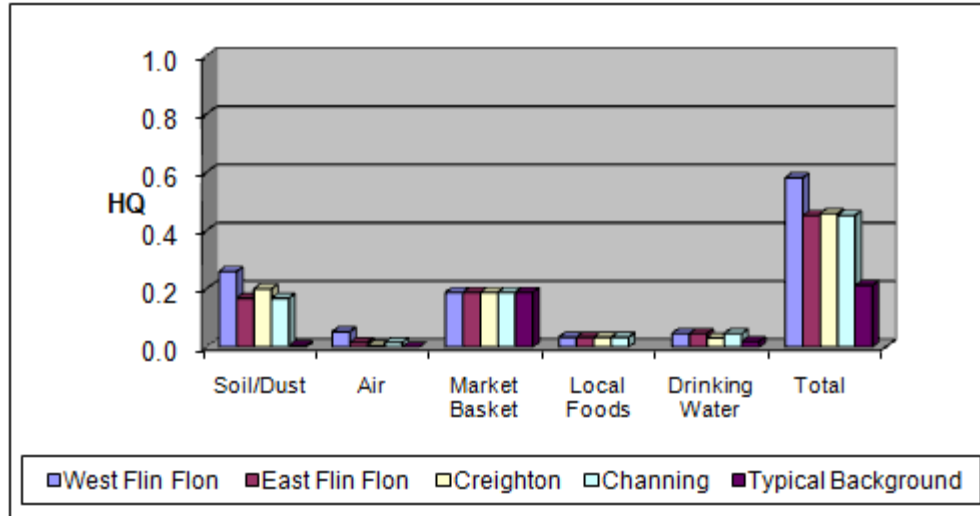


Figure 5-16 Pathway-Specific Hazard Quotients for a Toddler in each COI as a Result of Exposure to Lead

Therefore, using the community-based EPCs for lead-impacted environmental media, the predicted daily exposures for receptors living in each of the COI are not in excess of the Health Canada TRV and are not likely to increase BLL. However, given that direct contact with soil and dust is a significant source of lead exposure, and that concentrations of lead in these media are highly variable throughout each of the COI, consideration should be given on a property by property basis. This is discussed further following the derivation of the PTC for lead.

5.2.4.2 Exposure and Risk Estimated using the IEUBK Model

Using the U.S. EPA IEUBK model, BLLs were predicted for receptors in seven age categories for each of the four COI using the EPCs for environmental media in each community. The predicted BLLs represent the geometric mean for each age category calculated assuming a geometric standard deviation (GSD) of 1.6. This value considers biological and behavioural differences in receptors, variability in repeat sampling, variability resulting from sampling locations, and analytical variability (U.S. EPA, 2002).

Predicted BLLs for all age categories for all four COI and the Typical Background scenario, as well as the overall geometric mean concentrations, were well below 10 µg/dL (Table 5-24). The community of West Flin Flon had higher BLLs than other COI, but the highest predicted concentration of 5.6 µg/dL for the one to two years of age category was still well below 10 µg/dL. The probabilities of exceeding a BLL of 5 and 10 µg/dL at the EPC for each COI are also presented in Table 5-24.

Age Categories (years)	West Flin Flon	East Flin Flon	Creighton	Channing	Typical Background
0 to 1	4.8	4.1	4.1	4.1	0.8
1 to 2	5.6	4.8	4.7	4.8	0.7
2 to 3	5.2	4.4	4.4	4.4	0.6
3 to 4	5.0	4.3	4.2	4.3	0.6
4 to 5	4.2	3.6	3.5	3.6	0.5
5 to 6	3.6	3.1	3.0	3.1	0.5
6 to 7	3.2	2.8	2.7	2.8	0.5
Geometric Mean	4.5	3.8	3.8	3.8	0.66
95 th Percentile BLL	9.7	8.2	8.2	8.2	1.4
Probability of exceeding a BLL of 5 µg/dL	41%	29%	28%	29%	0%
Probability of exceeding a BLL of 10 µg/dL	4.4%	2.1%	1.9%	2.1%	0%

In Table 5-24, the 95th percentile BLLs were calculated using the following equation as recommended by U.S. EPA (2002):

$$X_{95} = GM \times GSD^{Z_{95}}$$

where:

- X_{95} = Blood lead level at the 95th percentile (µg/dL)
- GM = Geometric mean blood lead level (µg/dL)
- GSD = Geometric standard deviation of the distribution (1.6)
- Z_{95} = Z-score corresponding to the 95th percentile of the standard normal cumulative distribution (1.645)

Assuming homogeneous concentrations of lead in environmental media and diet, and incorporating the GSD of 1.6 to account for variability, the probability density for West Flin Flon receptors shows that approximately 4.4% of the population of children up to the age of seven are predicted to have BLLs greater than the level of concern (10 µg/dL) (Figure 5-17).

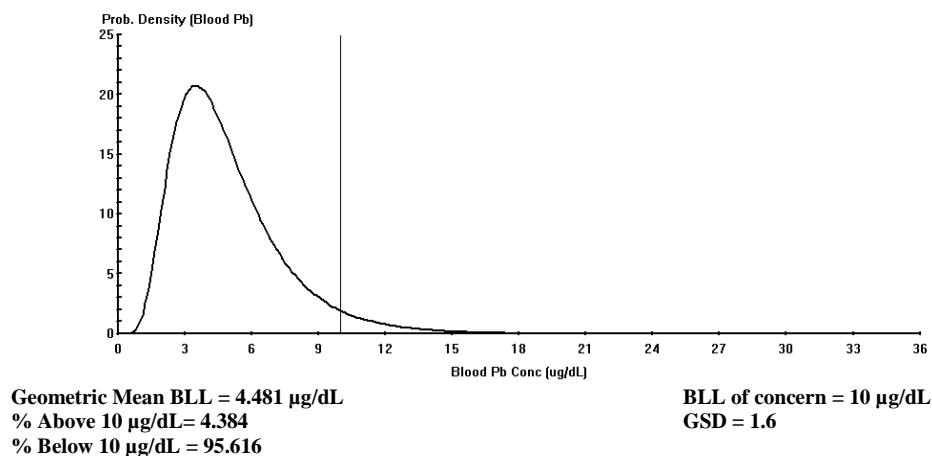


Figure 5-17 Probability Density of Blood Lead Concentrations (µg/dL) in West Flin Flon Children Relative to a Blood Lead Level of Concern of 10 µg/dL

Since there is current research to indicate that cognitive ability in children may be adversely affected at BLLs of <math><10\ \mu\text{g}/\text{dL}</math>, the probability density for children in West Flin Flon was also presented relative to a BLL of concern of

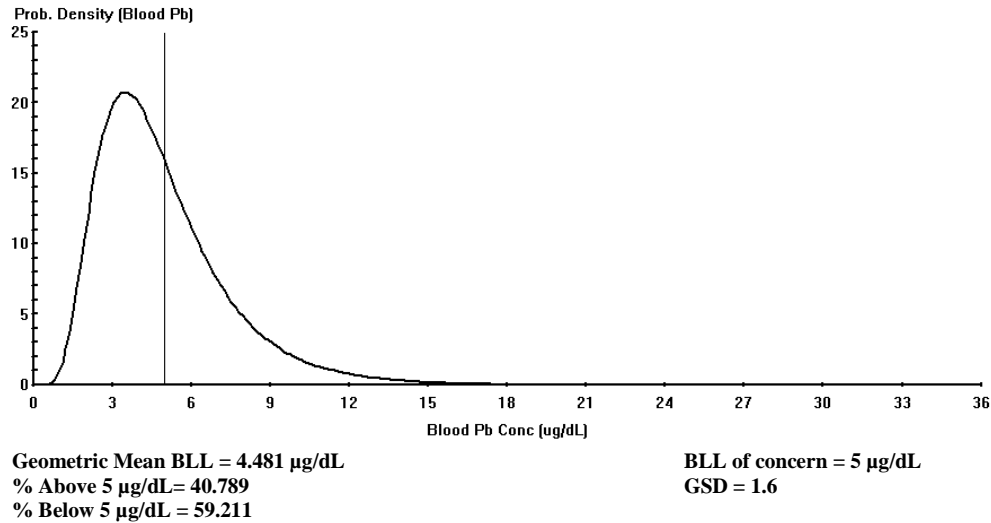


Figure 5-18 Probability Density of Blood Lead Concentrations ($\mu\text{g}/\text{dL}$) in West Flin Flon Children Relative to a Blood Lead Level of Concern of $5\ \mu\text{g}/\text{dL}$

The EPCs of lead in soils varied among the COI assessed ($160\ \mu\text{g}/\text{g}$ in East Flin Flon and Channing, $250\ \mu\text{g}/\text{g}$ in Creighton, and $370\ \mu\text{g}/\text{g}$ in West Flin Flon). This range in values is partly reflected in the predicted BLLs (Figure 5-19). The occurrence of elevated concentrations of lead in indoor dust in communities with lower outdoor soil concentrations reduces the difference in predicted BLLs among the COI despite large differences in soil EPCs.

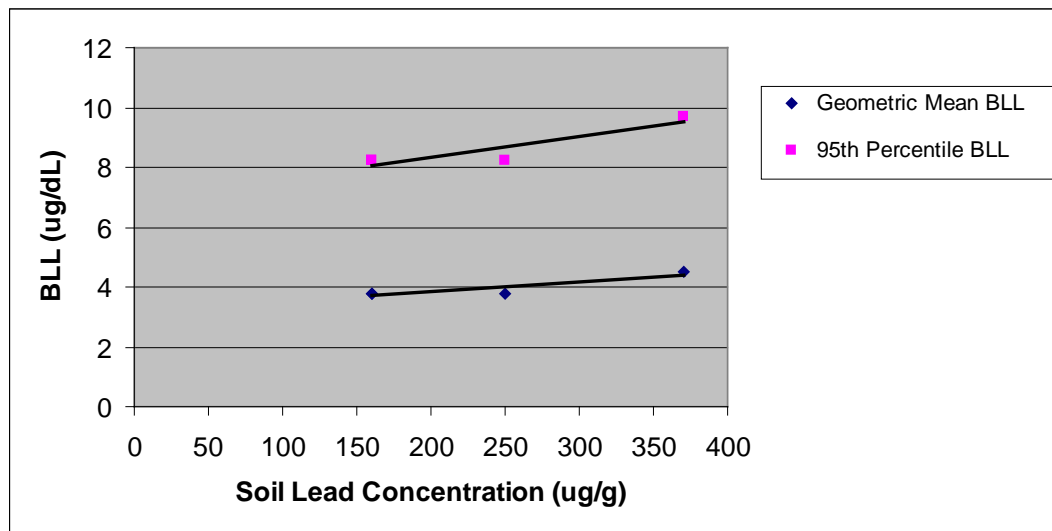


Figure 5-19 Predicted Blood Lead Concentrations Associated with the Range of Soil Concentrations Among the Four COI

The percent contribution to total BLL from each of the four exposure pathways was similar for all four COI but differed from the distribution predicted using the HHRA model. This is primarily a result of two factors. Firstly, the market basket exposure data used in the IEUBK model is based on a recent update provided by the U.S. EPA designed to reflect the current lead content of North American food items. As a result, these values are notably lower than those used in the HHRA model resulting in a lower contribution of diet to total lead exposure. It should be noted that the dietary contribution presented in Table 5-25 represents exposure from the consumption of both market basket and local foods. Secondly, the assumed soil/dust ingestion rates set as default in the IEUBK model are significantly higher than those recommended by Health Canada (2006) (refer to Chapter 7, Table 7-10). These two factors result in a significantly higher contribution of soil and dust to the total daily lead exposure than predicted using the HHRA model. The contributions from each media as predicted by the IEUBK model are shown for West Flin Flon receptors in Table 5-25. Since the dietary input used in the IEUBK model is lower than that used in the HHRA model and the assumed ingestion rates for soil and dust are higher than those recommended by Health Canada (2006), the predicted contribution of soil and dust to total lead exposure is notably higher within the IEUBK model estimates relative to the HHRA model estimates.

Age Categories (years)	Air	Diet	Water	Soil and Dust
0 to 1	0.81%	15.8%	4.7%	78.8%
1 to 2	0.86%	11.1%	7.5%	80.5%
2 to 3	1.50%	11.9%	7.7%	79.0%
3 to 4	1.58%	12.4%	7.7%	78.3%
4 to 5	1.92%	15.8%	10.0%	72.2%
5 to 6	2.79%	17.5%	11.1%	68.5%
6 to 7	2.83%	19.6%	11.6%	66.0%

Soil and dust contributions were the dominant source of BLLs for receptors of all age categories, followed by diet and drinking water. Contributions from air were minor relative to the other exposure sources (Figure 5-20).

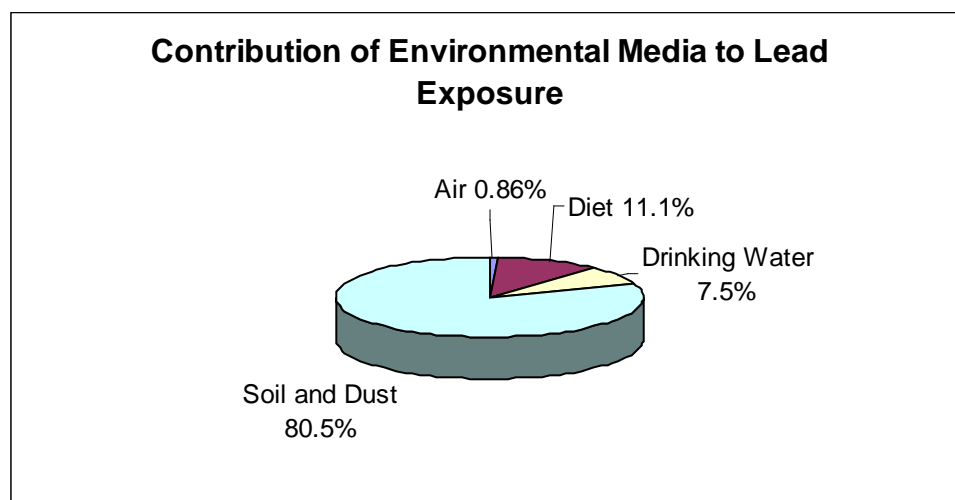


Figure 5-20 Contributions of Environmental Media to Lead Exposure in Children Aged 1 to 2 Years in West Flin Flon Based on the IEUBK Model

Derivation of a Residential Lead PTC

Although exposure to lead through the inhalation of particulates in air and from the consumption of drinking water may vary from community to community, these sources have a relatively minor contribution to total lead intake. The exposure to lead through the consumption of market basket and local foods is notably higher however, contribution from these sources is anticipated to be similar throughout each of the four COI. It can therefore be reasoned that the derivation of a single lead PTC should be applicable to each of the four COI. Since concentrations of lead in air are highest in West Flin Flon, environmental parameters associated with this community were used to back-calculate a residential PTC. Using the HHRA exposure model, an iterative process was used in which concentrations of lead in air and drinking water, and total dietary lead intake, remained constant while the concentration in soil was adjusted. Since the IEUBK MSA module accurately predicted concentrations of lead in indoor dust in West Flin Flon, concentrations of lead in indoor dust were assumed to be a function of the concentrations in outdoor soil and dust concentrations were automatically adjusted during this process.

The HHRA exposure model, the Health Canada TDI and the predicted exposure levels were used to derive a residential PTC. Given that the TDI is 3.6 µg/kg/day and the predicted exposure from market basket foods for the toddler is 0.68 µg/kg/day, the RTDI to be allocated to exposure from soil/dust, drinking water, local foods, and air is 2.9 µg/kg/day. Since lead exposure may also occur through contact with consumer products (*i.e.*, toys, furniture, *etc.*), 20% of the RTDI was allocated to consumer products (*i.e.*, 0.58 µg/kg/day). The predicted daily exposure of a toddler living in West Flin Flon *via* drinking water (0.17 µg/kg/day), local foods (0.12 µg/kg/day), and air (0.19 µg/kg/day) totals 0.48 µg/kg/day. Assuming that exposure to lead remains constant from all sources other than soil/dust, the RTDI to be allocated to soil/dust *via* ingestion and dermal exposure is 1.8 µg/kg/day for the toddler. This represents 50% of the TDI, which is lower than the OMOE (1994) allocation of 64% of the TDI to soil/dust. A soil concentration of 700 µg/g (and an associated indoor dust concentration of 520 µg/g) produces an exposure of 3.6 µg/kg/day and results in a total HQ of 1.0 (Table 5-26).

Media	Exposure (µg/kg/day)	Media-Specific HQ
Air	0.19	0.053
Drinking Water	0.17	0.046
Local Fish	0.021	0.0057
Local Wild Game	0.012	0.0032
Blue Berries	0.037	0.010
Home Garden Vegetables	0.053	0.015
Market Basket	0.68	0.19
EDI without Soil/Dust	1.2	0.33
TDI	3.6	-
RTDI allocated to Consumer Products (20% of RTDI)	0.58	0.16
RTDI allocated to Soil/Dust	1.8	0.50

Therefore, based on the HHRA exposure model and the Health Canada TDI of 3.6 µg/kg/day, a PTC of 700 µg/g was derived.

Also using environmental parameters associated with West Flin Flon, the IEUBK model was used to back-calculate PTCs that are protective of a 5% probability of exceeding BLLs of 5 and 10 µg/dL. An iterative process was used in which concentrations of lead in air and drinking water, and total dietary lead intake, remained constant while the concentration in soil was adjusted until each of the two criteria were met. Using the IEUBK MSA module, concentrations

of lead in indoor dust are a function of the concentrations in outdoor soil, therefore, dust concentrations were automatically adjusted during this process.

Using the IEUBK model, an outdoor soil concentration of 370 µg/g (and an associated indoor dust concentration of 290 µg/g) was found to result in a 5% probability of exceeding a BLL of 10 µg/dL (Figure 5-21).

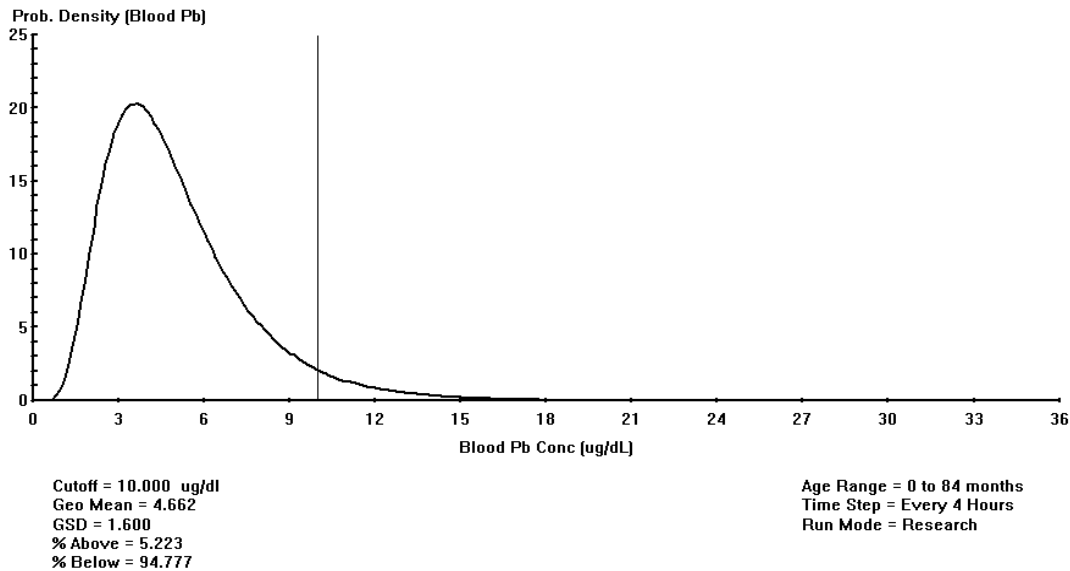


Figure 5-21 Probability density of blood lead concentrations associated with a 5% probability of exceeding 10 µg/dL (outdoor soil concentration of 370 µg/g)

An outdoor soil concentration of 98 µg/g (and an associated indoor dust concentration of 100 µg/g) was found to result in a 5% probability of exceeding a BLL of 5 µg/dL (Figure 5-22).

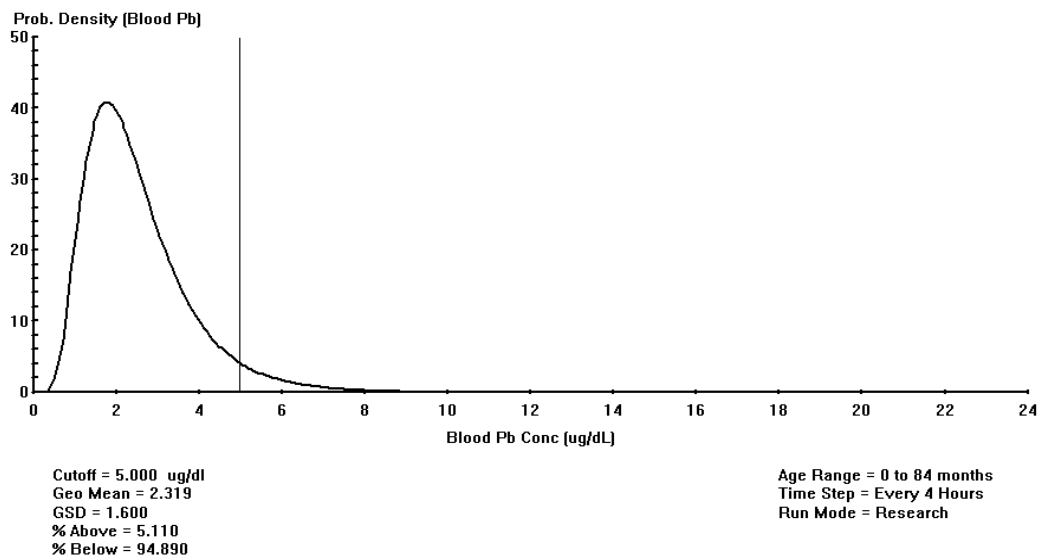


Figure 5-22 Probability density of blood lead concentrations associated with a 5% probability of exceeding 5 µg/dL (outdoor soil concentration of 98 µg/g)

Therefore, using the IEUBK model, a potential PTC of 98 µg/g was derived to be protective of a 5% probability of exceeding a BLL of 5 µg/dL, and a potential PTC of 370 µg/g was derived to be protective of a 5% probability of exceeding a BLL of 10 µg/dL.

Since there are a number of unique issues associated with the assessment of risk from exposure to lead, a weight-of-evidence discussion is presented to provide further context on the results of the HHRA and the selection of the final PTC.

Weight-of-Evidence Approach for the Derivation of a Lead PTC

As with exposures to arsenic, a simple evaluation of ambient soil and dust concentrations of lead in the COI may not be sufficient to provide an adequate and accurate basis on which to develop a reasonable PTC. As part of an overall weight-of-evidence approach, the following lines of evidence were reviewed and evaluated to aid in the development of an appropriate lead PTC:

- Exposure, risk, and PTC predictions from the HHRA Exposure Model;
- Predicted probabilities of exceeding BLL of concern using the IEUBK model; and,
- The empirical relationship between lead in soil and BLLs and how this information has formed the basis for PTC values derived at other sites.

An additional critical line of evidence would be site-specific blood lead data, if it were available for children within the study area.

The following section provides an overview of the lines of evidence used to establish the recommended PTC value for lead in the Flin Flon-Creighton area.

Table 5-27 provides the potential PTCs mathematically derived for lead using the HHRA and IEUBK models. Also included in the table are site-specific soil lead criteria previously established by the U.S. EPA (2001b).

Table 5-27 Provisional Trigger Concentrations (PTCs) and Soil Risk Management Levels (SRML) for Lead (µg/g)			
IEUBK Model Derived PTC	HHRA Model Derived PTC	U.S. EPA SRML	
		Play area	Bare soil Remainder
370 (protective of a 5% probability of exceeding a BLL of 10 µg/dL)	700	400	1,200

An examination of the information provided in Table 5-27 indicates that the HHRA model-derived PTC (700 µg/g) (based on the assumptions inherent in the HHRA) is significantly lower than the U.S. EPA Soil Risk Management Level (SRML) for soils other than those within children's play areas but greater than the value for children's play areas. The PTC predicted to be protective of a 5% probability of exceeding a BLL of 10 µg/dL using the IEUBK model (370 µg/g) is similar to the U.S. EPA SRML for children's play areas. It is noted that the Canadian Council for Ministers of the Environment (CCME, 1996) provide a soil lead criterion of 140 µg/g, however, this criterion is considered a screening value and not an intervention level. The Ontario Ministry of Environment (Ontario Regulation 153/04) provides a generic soil standard of 200 µg/g. Again, this standard is not considered to be an intervention level but rather used for the purposes of screening to determine if lead is found at levels that may require further evaluation.

Section 403 of the Toxic Substances Control Act (TSCA) (U.S. EPA, 2001b), established a lead standard for bare residential soil (400 µg/g in play areas based on the play area bare soil sample and an average of 1,200 µg/g in bare soil in the remainder of the yard). U.S. EPA utilized a weight-of-evidence evaluation in the derivation of the criteria. The derivation considered risk reduction rather than simply the selection of a standard based solely on model-based probability. A BLL of 10 µg/dL was considered as the BLL of concern while the environmental level of concern was established based on a 1 to 5% probability of an individual child's exceeding the BLL of concern. Other considerations included the large degree of uncertainty in selection of the BLL of concern and in relating environmental lead levels to BLLs. Economics were also considered. The U.S. EPA (2001b) indicated that to arrive at a soil-lead hazard level they *"sought to determine, with consideration of the uncertainty of the scientific evidence regarding environmental lead levels at which health effects would result, those conditions for which the Agency (U.S. EPA) had sufficient confidence in the likelihood of harm that abatement seemed warranted to achieve the associated level of risk reduction."*

In March 2001, the OMOE (2001b) developed a fact sheet related to lead contamination issues. In addition to providing background information related to lead exposure and toxicity, the fact sheet provides a commentary on risks related to soil-borne lead. The fact sheet indicates that there is minimal risk from exposure to soil with lead levels below 200 µg/g. Furthermore, it indicates that when soil lead levels are greater than 400 µg/g in bare soil areas of a child's play area or greater than 1,000 µg/g elsewhere on the property, the OMOE strongly advises that measures be taken to reduce or minimize the exposure of children. The fact sheet provides guidance on minimizing exposures for children. The fact sheet also indicates that there are minimal risks related to consuming home grown vegetables in soils containing less than 200 µg/g lead and that vegetables grown in soil containing greater than 1,000 µg/g lead should not be consumed. In May 2008, OMOE withdrew the 2001 fact sheet (OMOE, 2001b) and it was superseded with a revised fact sheet (OMOE, 2008). The revised fact sheet no longer provides SRMLs beyond the generic Ontario soil standard for lead of 200 µg/g.

Relationship between Lead in Soil and Blood Lead Levels

Recent scientific literature suggests that exposure to lead may cause adverse neurological changes in children at BLLs lower than 10 µg/dL. Soil and dust is a major exposure pathway for lead. Therefore, understanding the relationship between soil and dust levels with corresponding BLLs in children is important for development of environmental standards. The primary literature was reviewed to identify studies in which an empirical approach was used to investigate this relationship. The empirical approach generates a slope factor (µg/ Pb/dL blood/µg Pb/g soil) based on the correlation between measured soil lead concentrations and the BLLs in children assumed to be exposed to the soil (Stern, 1994). Empirical slopes reflect site-specific and study-specific exposure scenarios; therefore, these slope factors may not be generalized unless the factors that mediate soil lead levels and BLLs are taken into consideration (Stern, 1994). In addition, the relationship between lead intake and BLL is sublinear for higher intake levels (U.S. EPA, 1986); therefore, linear slopes derived from sites with high soil lead levels will underestimate the relationship (Stern, 1994).

The empirical results of the primary literature show that a BLL of approximately 5 µg/dL results from exposure to soil containing lead concentrations ranging from 500 to 1,500 µg/g (Angle *et al.*, 1984; Steele *et al.*, 1990; Stern, 1994; Lewin *et al.*, 1999; Johnson and Bretsch, 2002; Mielke *et al.*, 2007) (Table 5-28).

Lewin *et al.* (1999) examined the relationship between the concentrations of lead in soil and BLLs in children residing near four Superfund sites in the U.S by calculating a slope factor for the dose-response curve of children. The data was taken from concurrent investigations of populations near four National Priorities List sites where smelting and/or mining existed (ATSDR, 1995). In total, there were 1,015 measurements of blood lead in children (6 to 71 months) and lead soil samples from corresponding households. A slope factor was calculated by applying a multivariate linear regression model to double-log transformed soil lead and blood levels. The appropriateness of the regression model and data transformation were verified by statistical tests (Lewin *et al.*, 1999). After adjusting for income, education of the parents, presence of a smoker, sex and dust lead, a slope factor of 0.1388 was derived. Strengths of this study include a large sample size, household-specific environmental data, control of covariates, and strong quality control procedures.

Lewin *et al.* (1999) predicted BLLs in children based on household-specific soil lead concentrations in three models: high-risk, low-risk, and no covariate (Table 5-28). Overall, concentrations of lead in soil ranging from 500 to 1,500 µg/g resulted in BLLs ranging from 4.1 to 9.8 µg/dL. The high-risk population encompassed children who were male, and who lived in households with low income and education levels, without air conditioning, and that contained a smoker. For this population, soil concentrations of 500 µg/g and 1,500 µg/g resulted in predicted BLLs of 8.4 and 9.8 µg/dL, respectively. The low-risk population was defined as children who were female, and who lived in households with high income and education levels, with air conditioning, and with non-smokers. In this population, soil concentrations of 500 and 1,500 µg/g resulted in predicted BLLs of 4.1 and 4.9 µg/dL, respectively. A third model using a regression model without factoring in the covariates yielded predicted BLLs of 6.1 and 7.6 µg/dL from soil concentrations of 500 and 1,500 µg/g, respectively. The BLLs from the no covariate model were thought to be over-estimated.

Steele *et al.* (1990) examined thirteen epidemiological studies that investigated the relationship between soil/dust lead and BLLs in children residing in urban and smelter areas, and in regions near mine wastes from inactive smelter sites in the U.S. Overall, slope factors ranged between 0.76 and 8.1 µg/dL per 1,000 µg/g of soil lead (Table 5-28). The majority of the slopes were calculated by the U.S. EPA (1986) using a basic linear model assuming normal distribution. A fraction of the studies reported slopes that did not take other sources of exposure into account. The slopes from the studies conducted in regions with active smelters and urban areas encompassed the entire range of values reported, and the U.S. EPA (1986) estimated an overall slope value 2 µg/dL per 1,000 µg/g of soil lead. The slopes reported from studies in areas with inactive smelter sites were in the low end of the overall range (0 to 4 µg/dL per 1,000 µg/g of soil lead), and a mean slope value of 1.7 µg/dL per 1,000 µg/g of soil lead was estimated. The soil concentrations resulting in BLLs of 5 and 10 µg/dL were calculated using the slope factor range (0.67 to 8.1) reported by Steele *et al.* (1990). A BLL of 5 µg/dL corresponds to a soil concentration ranging from 620 to 660 µg/g.

A soil-specific increase in BLLs in young children exposed to residential soils in the U.S. was derived by Stern (1994). This approach defines an absolute contribution of lead from a single medium (soil), and it is assumed that this concentration will have a uniform effect across an exposed population, independent of other factors such as the blood lead distribution, or lead contribution from other sources (Stern, 1994). The approach employs a mechanistic model which estimates the total change in BLL from ingestion exposure to soil and soil-derived dust under steady-state conditions, with various input parameters. A slope factor of 10 µg/dL per 1,000 µg/g of soil lead was reported, which results from a BLL of 2 µg/dL in children with soil lead concentrations of 200 µg/g (Stern, 1994). Using this slope factor, soil concentrations of 500 and 1,000 µg/g resulting in BLLs of 5 and 10 µg/dL were calculated (Table 5-28).

Angle *et al.* (1984) applied a linear model to blood lead data in 1,074 children ages 1 to 18 years in urban and suburban areas in Omaha. Lead sources from house dust, air, and soil were incorporated in the model. A slope factor of 6.8 $\mu\text{g Pb/dL blood} / 1,000 \mu\text{g Pb/g soil}$ was reported. Soil concentrations of 735 and 1,470 $\mu\text{g/g}$ were calculated using the slope factor, resulting in BLLs of 5 and 10 $\mu\text{g/dL}$, respectively (Table 5-28). Von Lindern *et al.* (2003) also utilized a linear regression model to derive a slope factor of approximately 4 $\text{Pb/dL blood} / 1,000 \mu\text{g Pb/g soil}$. This study encompassed data from the Bunker Hill Superfund Site in Idaho near an abandoned lead/zinc smelting complex. Yard, neighbourhood, and community soil sources were analyzed in relation to blood lead levels in children, and the overall slope factor derived from the model was additive. Using the slope factor, a BLL of 5 $\mu\text{g/dL}$ resulted in a calculated soil concentration of 1,250 $\mu\text{g/g}$.

Mielke *et al.* (2007) derived a relationship between pooled soil lead and child blood lead data from census tracts of residential communities within metropolitan areas of New Orleans between 2000 and 2005. There was a highly significant curvilinear association between the soil and child blood lead data. Based on the curvilinear model, a median blood lead level of 5.9 $\mu\text{g/dL}$ resulted from exposure to a median concentration of 500 $\mu\text{g/g}$ soil lead. A median soil level of 300 $\mu\text{g/g}$ was associated with a predicted median BLL of 5 $\mu\text{g/dL}$. At higher lead soil concentrations (1,000 to 1,500 $\mu\text{g/g}$), median BLLs ranging from 7.5 to 8.7 $\mu\text{g/dL}$ were reported. Due to the non-linear nature of the relationship between blood and soil lead, a single slope factor was not reported. It was noted that below 100 $\mu\text{g/g}$ of lead in soil, BLLs increased 1.4 $\mu\text{g/dL}$ per 100 $\mu\text{g/g}$, and above 300 $\mu\text{g/g}$ of lead in soil, BLLs increased 0.32 $\mu\text{g/dL}$ per 100 $\mu\text{g/g}$ (Mielke *et al.*, 2007).

Johnson and Bretsch (2002) derived a logarithmic model of soil lead concentrations and BLLs in children (0 to 6 years) similar to the non-linear association reported by Mielke *et al.* (2007). Geo-referenced data sets were merged by a geographic clustering method, covering a 3 km^2 area in Syracuse, New York. A highly significant correlation was found in the model, where soil lead values ranged from 50 to 350 $\mu\text{g/g}$, and BLLs ranged from approximately 4 to 10 $\mu\text{g/dL}$. A slope factor was not derived in this study. The range of values reported by Johnson and Bretsch (2002) are similar to empirical results from other studies reviewed that show a BLL of approximately 5 $\mu\text{g/dL}$ resulting from exposure to soil containing lead concentrations ranging from 500 to 1,500 $\mu\text{g/g}$ (Table 5-28).

Concentrations of lead in soil less than 500 $\mu\text{g/g}$ were found to result in BLLs of 5 $\mu\text{g/dL}$ in children living near mine waste, inactive smelter sites, and urban areas (Table 5-29). Jin *et al.* (1997) summarized 22 cross-sectional studies of populations in areas with polluted soil and three prospective studies of soil lead abatement trials. Concentrations of lead in soil less than 500 $\mu\text{g/g}$ were found to result in BLLs of approximately 5 $\mu\text{g/dL}$ in children living near mine waste and inactive smelter sites (Table 5-29).

Louekari *et al.* (2004) measured BLLs in children aged 0 to 6 years living near a former smelter in Finland, and reported corresponding lead soil concentrations from home yards and day-care centres. BLLs ranging from <2.1 to 5 $\mu\text{g/dL}$ (average 2.7 $\mu\text{g/dL}$) were reported in 10 children living in the most contaminated areas near the former smelter, and corresponding soil lead concentrations ranged from 160 to 434 $\mu\text{g/g}$ (average 242 $\mu\text{g/g}$). In other areas near the site with lower lead soil concentrations (15 to 81 $\mu\text{g/g}$; average 40 $\mu\text{g/g}$), BLLs in 42 children ranged from <2.1 and 4.1 $\mu\text{g/dL}$ (average 2.1 $\mu\text{g/dL}$). In reference areas, soil lead was approximately 20 $\mu\text{g/g}$, and BLLs in children were <2 $\mu\text{g/dL}$. The BLLs reported in this study were much less than those measured when the smelter was operational.

In a random cross-sectional survey of children living near a former smelting operation in Midvale, Utah, Lanphear *et al.* (2003) reported child BLLs slightly above 5 µg/dL in association with soil lead concentrations slightly above 500 µg/g. A number of lead sources were correlated with blood lead, and a highly significant association was reported between soil lead that was collected from the perimeter of house foundations and blood lead in 6- to 72-month old children. Prior to soil abatement, a mean BLL of 5.6 µg/dL, with a 95% confidence interval of 4.9 to 6.3 µg/dL, was reported in association with a mean soil lead concentration of 542 µg/g with a 95% confidence interval of 466 to 631 µg/g.

Ren *et al.* (2006) also found that concentrations of lead in soil less than 500 µg/g resulted in BLLs of approximately 5 µg/dL in children living in urban areas (Table 5-29). The study measured lead in child blood and soils at ten kindergartens in Shenyang, China where lead pollution resulted primarily from automobile exhaust and industry emissions. Concentrations of lead in the soil at kindergartens ranged from 53 to 350 µg/g, and BLLs in children aged 3 to 5 years ranged from approximately 1 to 5 µg/dL (Table 5-29). The BLLs were lower in the younger children, with levels ranging from approximately 1 to 1.85 µg/dL in 3-year olds, 1 to 2.3 µg/dL in 4-year olds, and up to a maximum of 5 µg/dL in 5-year olds.

Additional studies describing BLLs in children living in urban populations in the 1970s and 1980s or living near actively emitting lead smelters were excluded from the current analysis. Urban studies were conducted during the 1970s and 1980s when lead additives were commonly used in gasoline and measured soil lead concentrations were extremely low indicating other sources of exposure. In addition, populations near active lead smelters were also omitted as elevated lead concentrations in air invalidate the relationship between soil lead levels and BLLs in children and the current HHRA accounted for air-borne exposures.

Slope Factor ($\mu\text{g Pb/dL blood/ 1,000 } \mu\text{g Pb/g soil}$)	Receptor	Description	Blood Lead Concentration ($\mu\text{g/dL}$)	Soil Concentration ($\mu\text{g/g}$)	Reference	
10	Children	Based on residential soil concentrations in the U.S. The calculated slope factor was assumed to be linear.	5	500 ^d	Stern, 1994	
			10	1,000 ^d		
0.1388	High Risk Children ^a	Four concurrent investigations of populations residing near four National Priorities List sites in the U.S. A natural logarithm regression was assumed.	8.4	500	Lewin <i>et al.</i> , 1999	
			9.2	1,000		
			9.8	1,500		
	No Covariate Children ^b	Four concurrent investigations of populations residing near four National Priorities List sites in the U.S. A natural logarithm regression was assumed.	6.0	500	Lewin <i>et al.</i> , 1999	
			7.1	1,000		
			7.6	1,500		
	Low Risk Children ^c	Four concurrent investigations of populations residing near four National Priorities List sites in the U.S. A natural logarithm regression was assumed.	4.1	500	Lewin <i>et al.</i> , 1999	
			4.6	1,000		
			4.9	1,500		
	No slope factor reported/derived	Children	Census tract data from residential communities in New Orleans, LA, 2000-2005. A curvilinear model was utilized.	5	300	Mielke <i>et al.</i> , 2007
				5.9	500	
				7.5	1,000	
8.7				1,500		
6.8	Children	Based on urban/suburban soil concentrations in Omaha. The calculated slope factor was assumed to be linear.	5	735 ^d	Angle <i>et al.</i> , 1984	
			10	1,470 ^d		
4	Children	Based on paired blood lead/ soil samples from the Bunker Hill Superfund Site in Idaho. The calculated slope factor is assumed to be linear.	5	1,250 ^d	Von Lindern <i>et al.</i> , 2003	
			10	2,500 ^d		
No slope factor reported/derived	Children	Aggregation of geo-referenced datasets from Syracuse, New York. A logarithmic regression model was assumed.	4 to 10 ^e	50 to 350	Johnson and Bretsch, 2002	
1.5	Children	Reanalysis of 1855 children less than 72 months of age from 11 studies (only 6 relevant to soil). Children from ore-processing sites were included	1.5	1000	Succop <i>et al.</i> , 1998	

Slope Factor ($\mu\text{g Pb/dL blood} / 1,000 \mu\text{g Pb/g soil}$)	Receptor	Description	Blood Lead Concentration ($\mu\text{g/dL}$)	Soil Concentration ($\mu\text{g/g}$)	Reference
2.4	Children	Analysis of 183 urban children enrolled in a random sample cross sectional study.	2.4	1000	Lanphear <i>et al.</i> , 1998
0.76 to 8.1	Children	Thirteen epidemiological investigations of populations residing in urban towns and towns with operating smelters in the U.S. The calculated slope factors were assumed to be linear.	5	620 to 6600 ^d	Steele <i>et al.</i> , 1990
			10	1,200 to 13,000 ^d	

^a High risk population; defined as children who did not have air conditioning, who lived with a smoker, were male and were from low income households.

^b A simple no-covariate regression model was used.

^c Low risk population; defined as children who had air conditioning, lived with non-smokers, were female and were from high income households.

^d The soil concentrations resulting in a blood lead concentration of 5 and 10 $\mu\text{g/dL}$ were calculated using the slope factor. It was assumed that the calculated slope factors were linear (e.g., [(slope factor / desired blood lead concentration (5 and 10 $\mu\text{g/dL}$)) = (1,000 $\mu\text{g/g soil} / \text{calculated soil concentration (x) ($\mu\text{g/g}$))].$

^e Levels approximated from graphical representation.

Site (Year)	Age Group	Blood Lead Level ($\mu\text{g/dL}$)	Soil Lead Concentration ($\mu\text{g/g}$)	Reference
Telluride, CO (1987)	0 to 71 mths	6.2	178	Jin <i>et al.</i> , 1997
Clear Creek/Central City, CO (1990)	0 to 71 mths	5.9	201	
Socorro, New Mexico (1990)	All ages	5.8	317	
Granite City, IL (1991)	6 to 14 yrs	5	338	
Montreal, PQ (1990)	6 to 71 mths	5.6	430	
Granite City, IL (1991)	6 to 71 mths	6.9	449	
Smuggler Mt., Aspen, CO (1990)	6 to 14 yrs	1.8	544	
Smuggler Mt., Aspen, CO (1990)	6 to 71 mths	2.6	641	
Palmerton, PA (1991)	6 to 71 mths	6.5	691	
Leadville, CO (1987)	6 to 71 mths	8.7	1,034	
Tikkurila, Finland (1996-1999)	0 to 6 yrs	2.7 (<2.1 to 5) ^a	242	Louekari <i>et al.</i> , 2004
		2.1 (<2.1 to 4.1) ^a	40	
		<2 (<2.1 to 2.5) ^a	20	
Midvale, UT (1989)	6 to 72 mths	5.6	542	Lanphear <i>et al.</i> , 2003
Shenyang, China (2003)	3 to 5 yrs	1 to 5 ^d	135 (53 to 350) ^a	Ren <i>et al.</i> , 2006

^a Average, minimum and maximum values are presented

^b Levels approximated from graphical representation

A blood lead screening study (Decou *et al.*, 2001) was also commissioned by the Regional Niagara Public Health Department in 2001 to determine exposure to and potential health impacts of lead on Port Colborne, Ontario, and specifically the Eastside Community which had demonstrated elevated lead concentrations in soils (arithmetic mean of 203 $\mu\text{g/g}$ and a maximum of 1,350 $\mu\text{g/g}$). In total, 1,065 individuals were screened, with approximately one-third of all participants from the Eastside Community. The geometric means and their confidence

intervals for the BLLs for all participants were well below the 10 µg/dL screening benchmark, with the geometric mean for the Eastside Community reported as 2 µg/dL. While blood lead screening results for children were compared to soil data for collection sites on each particular residential property, no statistical relationship was apparent between the two variables (Decou *et al.*, 2001).

Based on the results of the blood lead screening program, average Eastside Community BLLs were low and similar to those observed in the rest of Port Colborne, as well as other similar Ontario communities. The researchers concluded that children and pregnant women in the Eastside Community were not at an increased risk of lead exposure as compared to other communities in Ontario, even considering the localized elevated soil lead levels. Furthermore, all studied children who lived on properties with surficial soil lead concentrations in excess of 400 µg/g had BLLs less than 10 µg/dL. While the results of the survey indicated that no immediate intervention was required regarding lead soil remediation in the Eastside Community, the Regional Niagara Public Health Department continued to recommend limiting exposure to soil containing known contaminants, such as lead (Decou *et al.*, 2001).

Selection of a Residential Lead PTC

The wide range of potential PTC or SRML for lead, each with merit, indicate that a detailed weight-of-evidence evaluation is required as part of the PTC development process.

Concentrations of lead in soil less than 500 µg/g were found to result in BLLs of approximately 5 µg/dL in children living in urban areas and near mine waste, inactive smelter sites. Blood lead is a true marker of current exposure, eliminating many of the assumptions and uncertainties inherent in the HHRA, and, in children, generally reflects recent exposure. The current individual action level for intervention for lead is 10 µg/dL (CEOH, 1994), but there is a large volume of literature which suggests that health effects in children and adults occur at concentrations lower than this level (e.g., Lanphear *et al.* 2005; Shih *et al.*, 2006; Bellinger, 2008). The 10 µg/dL value is currently under review by Health Canada, and it is anticipated that Health Canada will reduce the intervention level in the near future (and hence, the Toxicity Reference Value or TRV would also be lowered). While it is anticipated that the level will be lowered, it not possible to confirm what the final accepted intervention level will be at this time. Some other regulatory agencies, such as the Ontario Ministry of Environment, are also in the process of reviewing blood lead intervention levels, and have commenced using a 5 µg/dL blood lead level as the basis of the policy related to their recently published lead air standard (OMOE, 2007). Others, such as the Centre for Disease Control and Prevention in the U.S., indicate that while recent studies suggest adverse health effects associated with concentrations less than 10 µg/dL, they have not lowered the intervention level due to inaccuracies associated with detecting low lead levels in laboratory testing, and that since there is no evidence of a threshold below which adverse effects are not experienced, any decision to establish a new level of concern would be arbitrary and provide uncertain benefits (www.cdc.gov/nceh/lead/faq/changebll.htm). Therefore, major health agencies (e.g., Health Canada, Centre of Disease Control and the U.S. EPA) use the 10 µg/dL limit, despite the new and emerging science which is indicating effects may be occurring at lower levels.

The results of the IEUBK modelling for the current assessment indicates that for children in West Flin Flon, a soil lead concentration of approximately 400 µg/g will result in a mean BLL of 5 µg/dL, and a concentration of 370 µg/g will result in a 5% probability of exceeding a BLL of 10 µg/dL. Based on the weight-of-evidence (*i.e.*, the strong indication provided in the literature that 500 µg Pb/g soil is a safe level for residential properties, and the previously established regulatory SRML for children's play areas of 400 µg Pb/g soil (U.S. EPA, 2001b), and the

relative strength-of-evidence associated with each of these elements, it is concluded that a residential PTC of 370 µg/g based on the IEUBK model results would be appropriate for the Flin Flon area. It was considered to be appropriate to set the PTC for the Flin Flon area slightly lower than those recommended for other areas to account for the greater contribution of air and drinking water to the total exposure that may be experienced in Flin Flon relative to areas such as Sudbury or Port Colborne, Ontario. As the U.S. EPA indicated in the derivation of their lead criteria, consideration of the uncertainty of the scientific evidence regarding environmental lead levels at which health effects would result, a residential soil PTC of 370 µg/g provides a sufficient level of protection to minimize the likelihood of harm to human health.

The number of properties sampled as part of the residential soil sampling study that contained concentrations of lead in excess of a residential PTC of 370 µg/g within each community are presented in Table 5-30.

	<i>West Flin Flon</i>	<i>East Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>	<i>Total</i>
# of Properties Sampled	77	66	30	10	183
# of Properties >370 µg/g	32 (42%)	2 (3%)	4 (13%)	0	38 (21%)

Figure 5-23 illustrates the maximum concentrations of lead in outdoor soil on individual properties relative to the PTC of 370 µg/g.

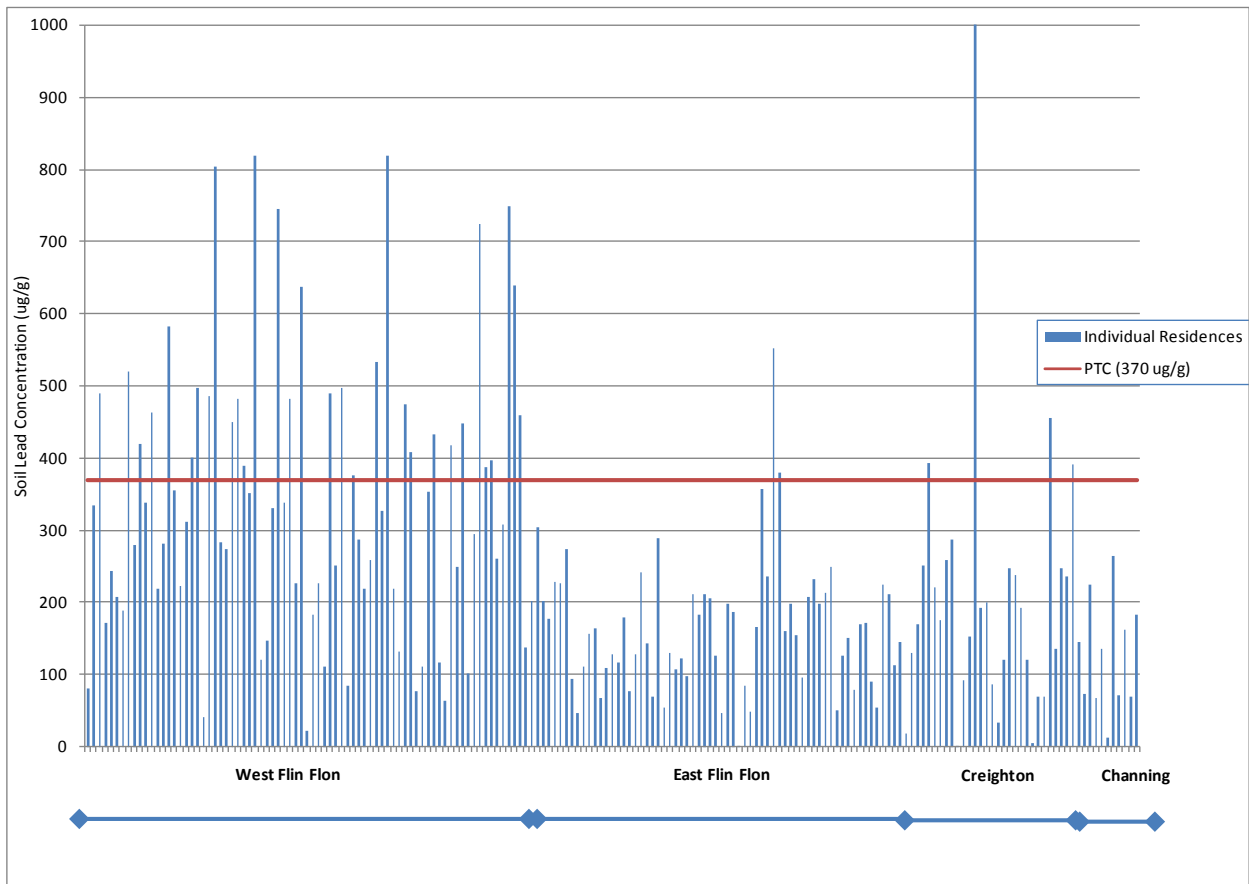


Figure 5-23 Maximum Concentrations of Lead on Individual Residential Properties Relative to the PTC of 370 µg/g.

Approximately 40% of homes sampled in West Flin Flon, 3% in East Flin Flon, and 13% in Creighton are in excess of the soil concentration (370 µg/g) predicted to result in a 5% probability of exceeding a BLL of 10 µg/dL.

Conclusion and Recommendations for Lead

Use of the IEUBK model for a community-based assessment has limitations in that it assumes that all children within a given population will be exposed to homogenous levels of lead under a similar exposure scenario. In reality, there may be significant variability in the lead concentrations in media such as indoor dust and backyard soil. Recognizing this limitation, BLLs for each COI were predicted to represent the geometric mean value for children in each of these communities. Results of this assessment indicate that geometric mean BLLs for each COI at the EPC soil and dust concentrations are well below 10 µg/dL. In addition, the 95th percentile BLLs were also below 10 µg/dL for each COI. While these results may provide a general idea of the average community risk potential, the most appropriate method to assess risk would be to consider soil concentrations on a property by property basis. There are a significant number of residential properties in West Flin Flon that contain concentrations of lead in outdoor soil that are above the residential PTC protective of a BLL of 10 µg/dL as well as a few in East Flin Flon and Creighton. Assuming that the homes sampled as part of the residential soil sampling program are reflective of the distribution of lead throughout each of the COI, these results indicate that there is the potential that approximately 40% of properties in

West Flin Flon contain levels of lead in excess of the PTC. A smaller percentage of properties in East Flin Flon (3%) and Creighton (13%) may also contain levels of lead in soil that are in excess of the PTC.

The assumptions and algorithms used within the IEUBK model are designed to provide a realistic assessment of the BLL in children exposed to lead through a number of exposure pathways. Since a significant percentage of homes in West Flin Flon and Creighton contain soil concentrations in excess of the PTC, the completion of a blood lead survey would be an appropriate method of reducing uncertainty in the exposure assessment and provide a more accurate measure of the levels occurring in young children in these communities. Based on the results of blood lead surveys completed in other communities in Canada such as Port Colborne, Ontario, measured BLLs are typically lower than those predicted using the IEUBK model. A blood lead survey should primarily focus on children up to the age of 7 years as they are the most sensitive to the impaired neurobehavioral development associated with elevated BLLs. Blood lead surveys are generally completed in late summer or early fall to assess children following a period of elevated exposure to outdoor soils. Given that lead has a half-life in blood of approximately 36 days, sampling during this time is likely to capture the highest BLL experienced by children throughout the year.

5.2.5 Mercury

The assessment of exposure and risk to mercury was completed for both organic (methyl mercury) and inorganic forms. While methyl mercury may be found in all forms of environmental media, it is generally recognized to be a small component of the total mercury measured in most. The fraction of methyl mercury in fish, drinking water, and ambient air is considered to be more significant. Therefore, the assessment of exposure and risks from methyl mercury was addressed *via* the consumption of local fish, market basket fish, ingestion of drinking water, and inhalation of ambient air. As recommended by the CCME (1996) within the derivation of the inorganic mercury Canadian Soil Quality Guideline: Human Health Effects document, methyl mercury was assumed to be 100% of the total mercury in market basket fish, 25% of the total mercury in drinking water, and 20% of the total mercury measured in ambient air. Based on the results of the Flin Flon Fish Study completed by Stantec, the 95% UCLM fraction of methyl mercury in local fish was calculated to be 96%. Total mercury measured in soil, dust, home garden produce, blueberries, wild game, and all market basket foods other than fish, was assumed to be 100% inorganic mercury.

5.2.5.1 Inorganic Mercury

Estimated Exposure to Inorganic Mercury

Concentrations of mercury measured in soil in West Flin Flon (EPC=130 µg/g) were significantly higher than those measured in the other COI (EPC=7.2, 8.9, 4.1 µg/g in East Flin Flon, Creighton, and Channing, respectively). As a result, the primary source of exposure to inorganic mercury for residents in West Flin Flon was from soil/dust (83% from outdoor soil and 2% from indoor dust for the toddler) (Figure 5-24). The indoor dust sampling program indicated that concentrations of mercury in indoor dust were much lower than those measured in outdoor soil for co-located samples. As a result, the contribution of indoor dust to inorganic mercury exposure was significantly lower than that from outdoor soil. Within the other COI, the primary source of exposure for the toddler was the consumption of market basket foods, representing 51 to 60% of the inorganic mercury exposure (Figure 5-25). Although market basket exposure was assumed to be the same for residents in each of the four COI, the percent contribution of market basket foods to total exposure differed slightly as a result of differences from exposure to

community-specific environmental media concentrations (i.e., soil, dust, air, and drinking water). Mercury content in market basket food items is unrelated to environmental contamination in the Flin Flon area and is reflective of foods consumed throughout Canada and North America.

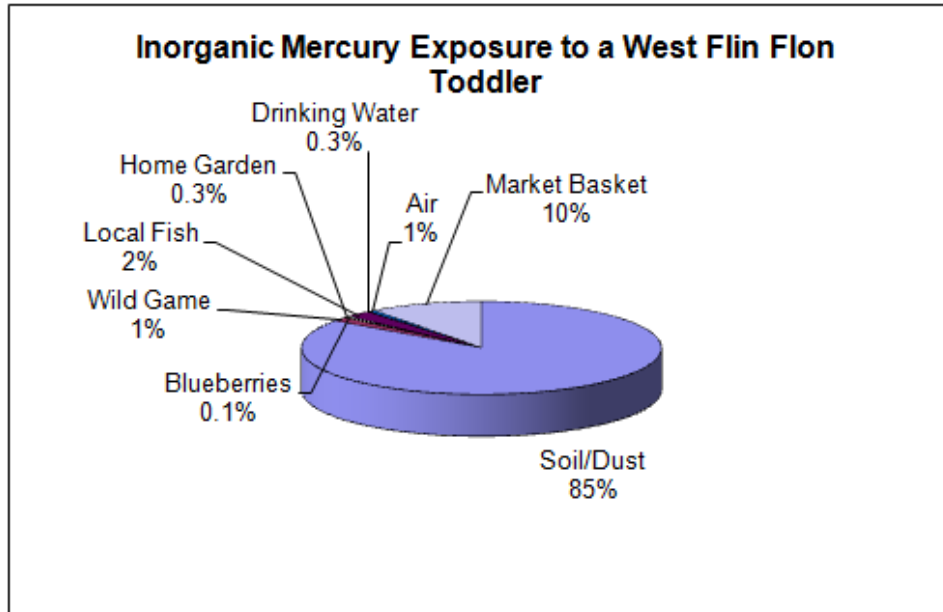


Figure 5-24 Contributions of Media to Inorganic Mercury Exposure for a Toddler Living in West Flin Flon

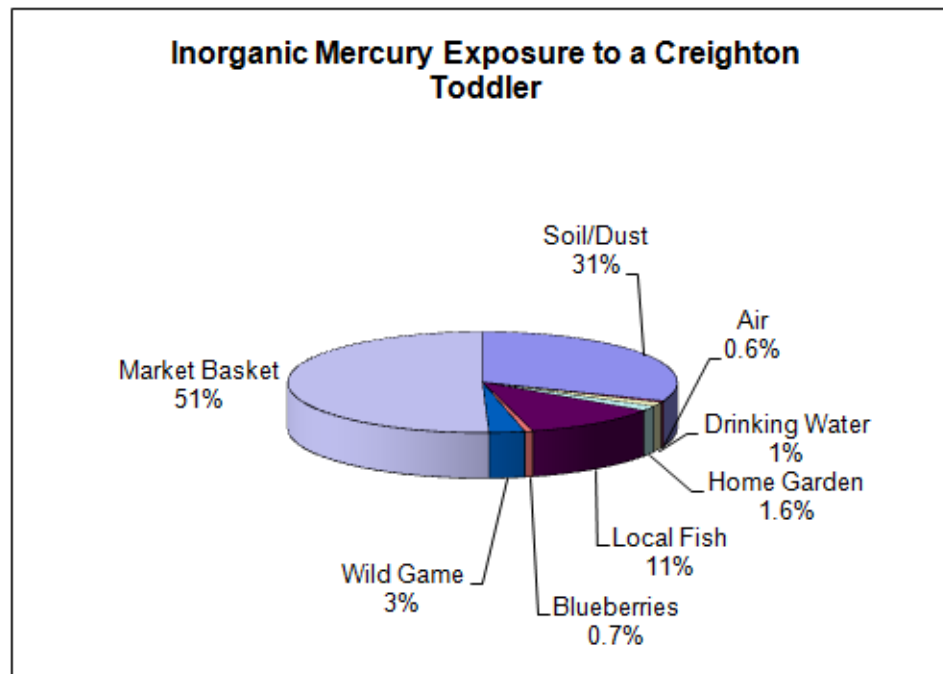


Figure 5-25 Contributions of Media to Inorganic Mercury Exposure for a Toddler Living in Creighton

A detailed breakdown of the pathway-specific exposures for residents of West Flin Flon in each of the 5 age classes is provided in Table 5-31. Details for receptors in other communities are provided in Appendix M.

<i>Exposure Pathway</i>	<i>Environmental Media Concentrations</i>		<i>Percent of Lifetime Total EDI</i>	<i>Estimated Daily Intakes (EDI) (µg/kg bw/day)</i>					
	<i>Value</i>	<i>Units</i>		<i>Infant</i>	<i>Toddler</i>	<i>Child</i>	<i>Teen</i>	<i>Adult</i>	<i>Lifetime</i>
Inhalation of Fine Particulate	0.013	µg/m ³	2.4%	0.0033	0.0072	0.0056	0.0034	0.0029	0.0034
Dermal Contact – Outdoors	130	µg/g	8.2%	0.025	0.018	0.014	0.011	0.010	0.011
Dermal Contact – Indoors	5.8	µg/g	0.12%	0.00044	0.00031	0.00022	0.00017	0.00016	0.00017
Outdoor Soil Ingestion	130	µg/g	36%	0.21	0.42	0.053	0.029	0.024	0.051
Indoor Dust Ingestion	5.8	µg/g	0.82%	0.0047	0.0094	0.0012	0.00065	0.00055	0.0011
Home Garden Root Vegetables	0.0025	µg/g ww	0.083%	0.000048	0.00022	0.00018	0.00014	0.000098	0.00012
Home Garden Other Vegetables	0.0082	µg/g ww	0.63%	0.0014	0.0015	0.0012	0.00081	0.00079	0.00087
Local Wild Blue Berries	0.01	µg/g ww	0.53%	0.00074	0.00074	0.00074	0.00074	0.00074	0.00074
Local Wild Game	0.0068	µg/g ww	2.2%	0	0.0031	0.0031	0.0031	0.0031	0.0031
Local Fish	0.018	µg/g ww	8.7%	0	0.012	0.012	0.012	0.012	0.012
Drinking Water	0.042	µg/L	0.66%	0.0015	0.0015	0.0010	0.00070	0.00089	0.00092
Dental Amalgam	NA	µg/g	24%	0	0	0.016	0.025	0.040	0.034
Market Basket Contribution	NA	µg/g	15%	0.066	0.054	0.035	0.021	0.016	0.020
Summary									
<i>Estimated Daily Intake (µg/kg/day)</i>			--	0.31	0.53	0.14	0.11	0.11	0.14
Inhalation Route Only			2.4%	0.0033	0.0072	0.0056	0.0034	0.0029	0.0034
Direct Soil/Dust Contact			46%	0.24	0.45	0.068	0.041	0.036	0.063
Market Basket Foods and Dental Amalgam			39%	0.066	0.054	0.051	0.046	0.056	0.056
Drinking Water			0.66%	0.0015	0.0015	0.0010	0.00070	0.00089	0.00092
Local Foods (Home Garden, Blueberries, Wild Game and Fish)			12%	0.0022	0.018	0.017	0.017	0.017	0.017

Hazard Quotients

HQ estimates for residents living in each of the COI ranged from 0.22 to 1.8, with the highest risk levels predicted for West Flin Flon (Table 5-32). Health Canada has recommended an oral TDI for inorganic mercury of 0.3 µg/kg/day to be protective of adverse kidney effects. With the exception of the toddler in West Flin Flon, all HQs were equal to or below the acceptable value of 1.0 indicating that adverse effects associated with elevated exposure to inorganic mercury at the EPCs are not anticipated. The highest risk levels were predicted to the toddler as a result of the elevated soil ingestion rate assumed for children of this age. These HQs were derived using the EPCs for several environmental media selected to be representative of typical exposure to residents in each of the COI. Receptors living at locations with concentrations of mercury in media that are higher or lower than the EPC are anticipated to be subject to risks that are subsequently higher or lower than those predicted for the general community population.

<i>Receptor</i>	<i>East Flin Flon</i>	<i>West Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>	<i>Typical Background</i>
Infant	0.28	1.0	0.29	0.26	0.23
Toddler	0.33	1.8	0.35	0.30	0.20
Child	0.25	0.48	0.25	0.24	0.19
Teen	0.22	0.36	0.22	0.22	0.17
Adult	0.25	0.37	0.25	0.25	0.20

Bolded values highlighted in grey exceed the acceptable HQ of 1.0.

Due to the significant contribution of soil in West Flin Flon to total inorganic mercury exposure, predicted HQs for receptors living in this COI are significantly higher than those predicted for other COI and under the Typical Background scenario (Figure 5-26). However, predicted HQs for COI other than West Flin Flon are similar to those predicted under the Typical Background scenario in which market basket foods and dental amalgam (for receptors other than the infant and toddler) account for a significant portion of the total HQ.

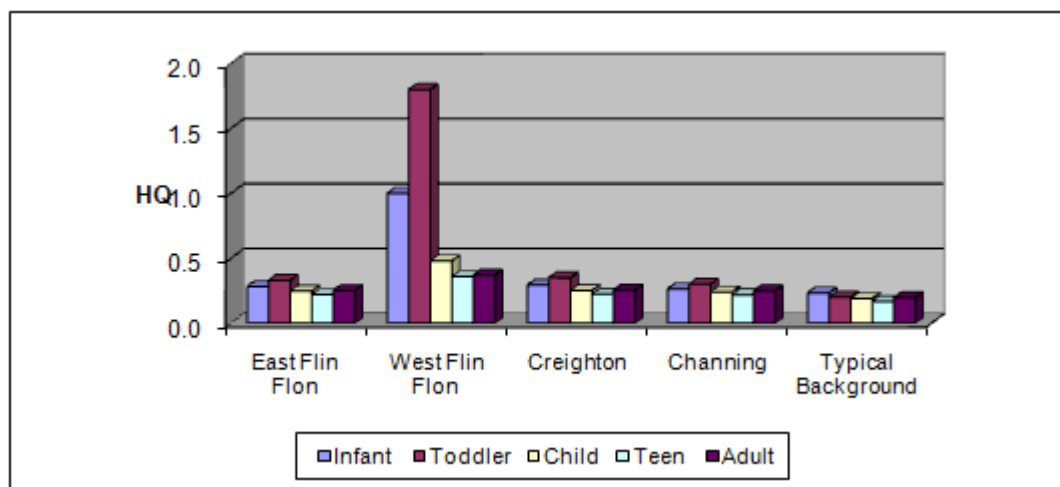


Figure 5-26 Predicted Hazard Quotients as a Result of Exposure to Inorganic Mercury

Within West Flin Flon, exposures of the toddler to inorganic mercury are dominated by contributions from soil. Within each of the other COI, exposures are dominated by contributions from market basket foods (Figure 5-27). HQs associated with the inhalation of air and the consumption of drinking water are minor, accounting for less than 1% and approximately 1% of the total HQ, respectively. The consumption of local blueberries and home garden produce did

not contribute significantly to the total HQ, representing less than 2% of the total HQ per media. The consumption of local fish represented approximately 12% of the total HQ, while contributions from the consumption of local wild game accounted for approximately 4% of the total HQ.

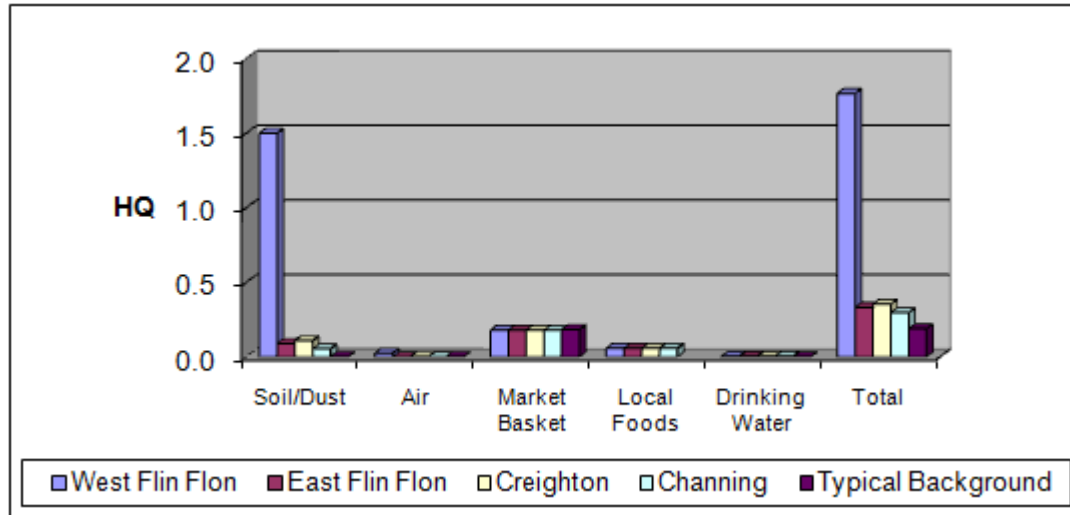


Figure 5-27 Pathway-Specific Hazard Quotients for a Toddler in each COI as a Result of Exposure to Inorganic Mercury

An HQ greater than 1.0 was only predicted for the toddler living in West Flin Flon. This scenario combines the receptor with the greatest soil consumption rate and the COI with the highest concentrations of mercury in soil. Predicted HQs are within the acceptable range for all other receptors.

Derivation of a Residential Inorganic Mercury PTC

Although exposure to inorganic mercury through the inhalation of particulates in air and from the consumption of drinking water may vary from community to community, these sources have a relatively minor contribution to total inorganic mercury intake. The exposure to inorganic mercury through the consumption of market basket and local foods is notably higher however, contribution from these sources is anticipated to be similar throughout each of the four COI. It can therefore be reasoned that the derivation of a single inorganic mercury PTC should be applicable to residential properties in each of the four COI. Since concentrations of mercury in air are highest in West Flin Flon, environmental parameters associated with this community were used to back-calculate a PTC that is protective of an HQ of 1.0. An iterative process was used in which exposure to inorganic mercury for the toddler from air, drinking water, local foods, and market basket foods remained constant while the concentration in soil was adjusted to produce an HQ of 1.0. Since concentrations of mercury in indoor dust are a function of the concentrations in outdoor soil, dust concentrations were automatically adjusted during this process.

Given that the chronic oral TDI for inorganic mercury is 0.3 µg/kg/day and the predicted exposure from market basket foods is 0.054 µg/kg/day (not including 0.082 µg/kg/day allocated to methyl mercury), the RTDI to be allocated to exposure from soil/dust, drinking water, local foods, and air is 0.25 µg/kg/day. Based on the assumptions in the current assessment, the EDI resulting from drinking water (0.0015 µg/kg/day), local foods (0.018 µg/kg/day), and air (0.0072 µg/kg/day) is 0.027 µg/kg/day. Assuming that exposure to inorganic mercury remains constant

from all sources other than soil/dust, the RTDI to be allocated to soil/dust *via* ingestion and dermal exposure is 0.22 µg/kg/day for the toddler. Conservatively assuming 100% bioavailability of inorganic mercury in soil, a soil concentration of 64 µg/g (and an associated indoor dust concentration of 3.6 µg/g) produces a soil/dust-related exposure of 0.19 µg/kg/day and results in a total HQ of 1.0 (Table 5-33). The assumed 100% bioavailability of mercury in soil may have resulted in a significant over-prediction of risk and the derivation of a highly conservative PTC. The soil bioaccessibility study completed for residential soils in Flin Flon/Creighton indicated an average bioaccessibility of 1.2% for total mercury, and a 95% UCLM value of 1.5%, based on a single phase analysis. However, given that the results of *in vitro* bioaccessibility testing for mercury have not been validated with *in vivo* studies, the results of this study were not utilized in the current HHRA.

Media	Exposure (µg/kg/day)	Media-Specific HQ
Air	0.0072	0.025
Drinking Water	0.0015	0.0051
Local Fish	0.012	0.040
Local Wild Game	0.0031	0.010
Blue Berries	0.00073	0.0024
Home Garden Vegetables	0.0017	0.0056
Market Basket	0.054	0.18
EDI without Soil/Dust	0.080	0.27
TDI	0.3	--
RTDI allocated to Soil/Dust	0.22	0.73

A comparison of the inorganic mercury PTC of 64 µg/g with the results of the residential soil sampling program indicates that all properties sampled in East Flin Flon, Creighton, and Channing were below the PTC, and that 40 of the 77 properties sampled in West Flin Flon were in excess of the PTC (Table 5-34).

	West Flin Flon	East Flin Flon	Creighton	Channing	Total
# of Properties Sampled	77	66	30	10	183
# of Properties >64 µg/g	40 (52%)	0	0	0	40 (22%)

Figure 5-28 illustrates the maximum concentrations of mercury in outdoor soil on individual properties relative to the PTC of 64 µg/g.

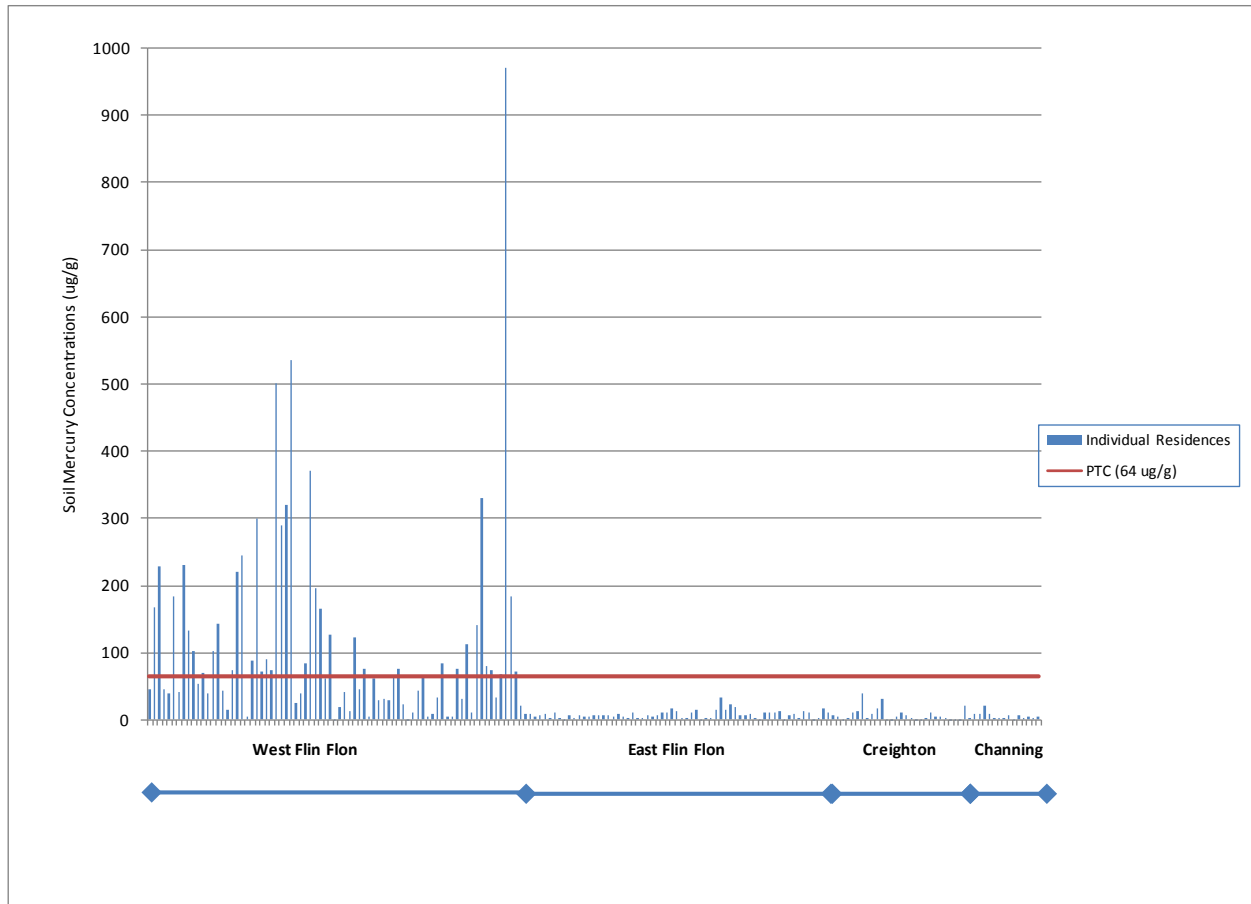


Figure 5-28 Maximum Concentrations of Mercury on Individual Residential Properties Relative to the PTC of 64 µg/g.

Concentrations of mercury in soil found on numerous properties sampled in West Flin Flon may result in the occurrence of adverse health effects in individuals that are highly exposed to impacted soils. Biomonitoring would be an appropriate option to more accurately assess inorganic mercury exposure to individuals in West Flin Flon. Human blood, hair, breast milk, and urine are generally used in measuring mercury exposure (ATSDR, 1999). In the blood, mercury has a short half-life (3 days) thereby making it a useful medium for determining short-term exposures (WHO, 2003). However, long-term consumption of fish containing elevated methyl mercury levels can also be determined through blood analysis. Mercury analysis of hair is useful in that it reveals dietary exposure to methyl mercury, however, this has not been regarded as a suitable indicator for inorganic mercury exposure (WHO, 1991). For long-term, low level exposures to inorganic mercury, measurement through urine samples is the preferred medium (Yoshida, 1985; ATSDR, 1999; WHO, 2003).

5.2.5.2 Methyl Mercury

Estimated Exposure to Methyl Mercury

Exposure to methyl mercury was assumed to occur *via* the consumption of fish from market basket foods, consumption of local fish, consumption of drinking water, and inhalation of ambient air (Figure 5-29). The primary source of exposure to residents in each of the COI was through ingestion of local fish (approximately 78% for the toddler). The second highest source

of methyl mercury exposure was *via* the consumption of market basket fish, representing approximately 22% of the total exposure for a toddler.

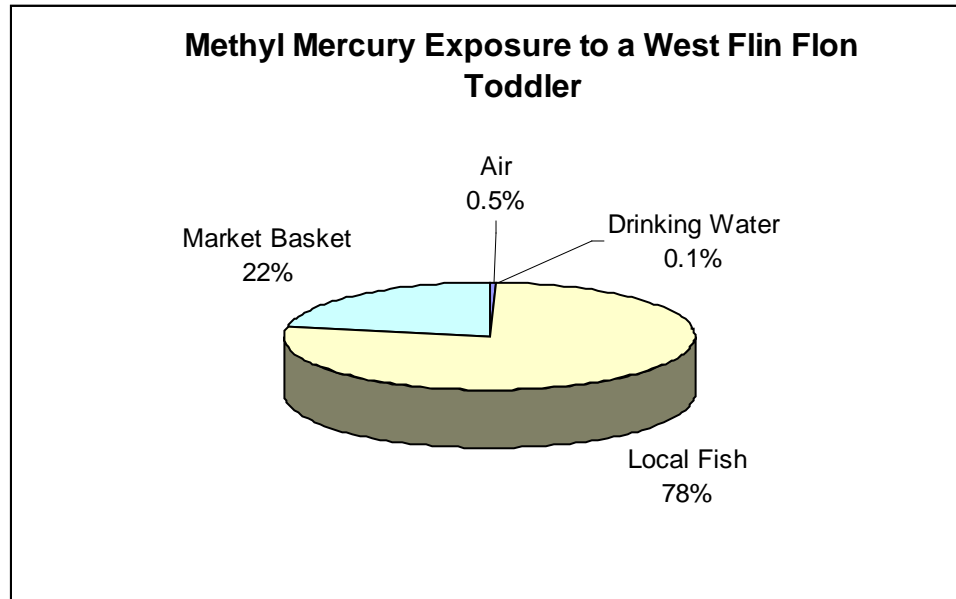


Figure 5-29 Contributions of Media to Methyl Mercury Exposure for a Toddler Living in West Flin Flon

A detailed breakdown of the pathway-specific exposures for residents of West Flin Flon in each of the 5 age classes is provided in Table 5-35. Details for receptors in other communities are provided in Appendix M.

<i>Exposure Pathway</i>	<i>Environmental Media Concentrations</i>		<i>Percent of Lifetime Total EDI</i>	<i>Estimated Daily Intakes (EDI) (µg/kg bw/day)</i>					
	<i>Value</i>	<i>Units</i>		<i>Infant</i>	<i>Toddler</i>	<i>Child</i>	<i>Teen</i>	<i>Adult</i>	<i>Lifetime</i>
Inhalation of Fine Particulate	0.0032	µg/m ³	0.24%	0.00082	0.0018	0.0014	0.00085	0.00072	0.00085
Dermal Contact – Outdoors	-	µg/g	-	-	-	-	-	-	-
Dermal Contact – Indoors	-	µg/g	-	-	-	-	-	-	-
Outdoor Soil Ingestion	-	µg/g	-	-	-	-	-	-	-
Indoor Dust Ingestion	-	µg/g	-	-	-	-	-	-	-
Home Garden Root Vegetables	-	µg/g ww	-	-	-	-	-	-	-
Home Garden Other Vegetables	-	µg/g ww	-	-	-	-	-	-	-
Local Wild Blue Berries	-	µg/g ww	-	-	-	-	-	-	-
Local Wild Game	-	µg/g ww	-	-	-	-	-	-	-
Local Fish	0.43	µg/g ww	81%	0	0.29	0.29	0.29	0.29	0.29
Drinking Water	0.014	µg/L	0.086%	0.00051	0.00051	0.00034	0.00023	0.00030	0.00031
Market Basket Fish	0.29	µg/g	19%	0	0.082	0.084	0.056	0.066	0.067
Summary									
<i>Estimated Daily Intake (µg/kg/day)</i>			-	0.0013	0.37	0.37	0.35	0.36	0.36
Inhalation Route Only			0.24%	0.00082	0.0018	0.0014	0.00085	0.00072	0.00085
Direct Soil/Dust Contact			-	-	-	-	-	-	-
Market Basket Foods			19%	0	0.082	0.084	0.056	0.066	0.067
Drinking Water			0.086%	0.00051	0.00051	0.00034	0.00023	0.00030	0.00031
Local Foods (Home Garden, Blueberries, Wild Game and Fish)			81%	0	0.29	0.29	0.29	0.29	0.29

Hazard Quotients

HQ estimates for residents living in each of the COI ranged from 0.0026 to 1.9, with risk levels consistent throughout each of the COI (Table 5-36). HQ estimates for the infant were significantly lower than those for other age groups because it was assumed that children under the age of 6 months would not consume fish. Although the primary route of exposure for all receptors other than the infant was the consumption of local fish, and each of these receptors consumed the same amount of fish on a per body weight basis, HQs were much higher for the toddler and child as a result of the use of lower RfDs for these age groups (*i.e.*, 0.2 µg/kg/day for the toddler and child relative to 0.47 µg/kg/day for the teen and adult) protective of neurodevelopmental effects.

Receptor	East Flin Flon	West Flin Flon	Creighton	Channing	Typical Background
Infant	0.0026	0.0067	0.0027	0.0026	0.0035
Toddler	1.8	1.9	1.8	1.8	0.42
Child	1.9	1.9	1.9	1.9	0.43
Teen	0.74	0.74	0.74	0.74	0.12
Adult	0.77	0.77	0.77	0.77	0.14

Due to the significant contribution of local fish to the total methyl mercury exposure, predicted risk levels for receptors living in each of the COI are significantly higher than those predicted under the Typical Background scenario (Figure 5-30).

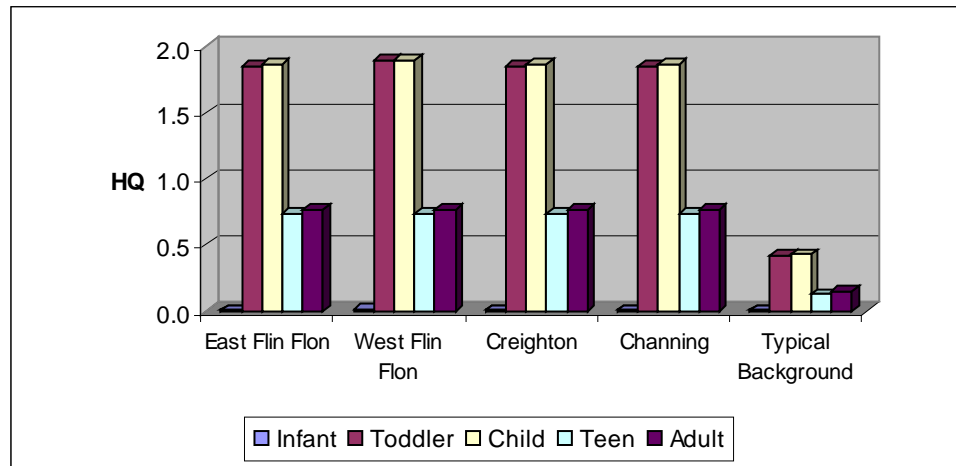


Figure 5-30 Predicted Hazard Quotients as a Result of Exposure to Methyl Mercury

Exposure to methyl mercury, and subsequently risk levels, are dominated by contributions from local and market basket fish (Figure 5-31). Contributions from all other sources are very minor. Methyl mercury content in residential soils are assumed to be negligible, therefore, exposure to methyl mercury through direct soil/dust pathways was not assessed.

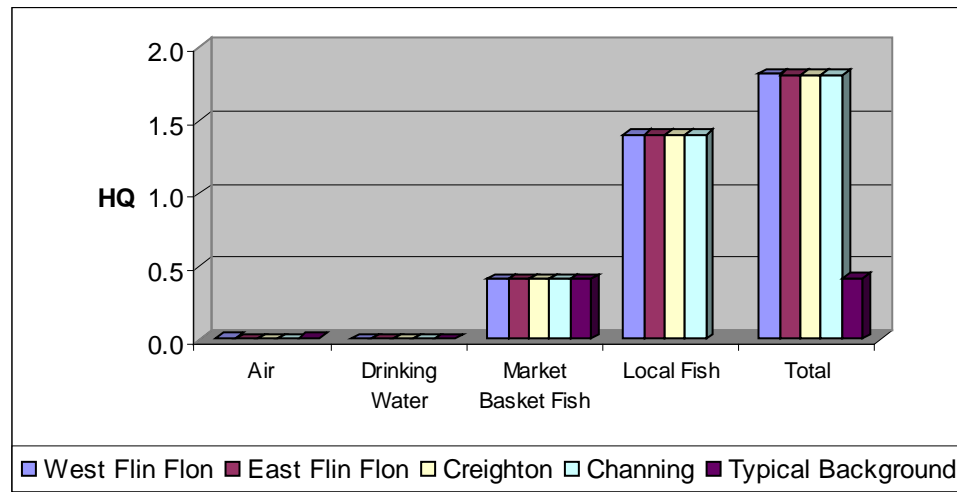


Figure 5-31 Pathway-Specific Hazard Quotients for a Toddler in each COI as a Result of Exposure to Methyl Mercury

Since methyl mercury is not a significant component of residential soils, a soil PTC was not derived. Overall, unacceptable risks may occur to toddlers and children as a result of exposure to methyl mercury under the assumed exposure scenario. This assumes that these receptors will consume 1.5 local fish meals per week, 52 weeks per year at the 95% UCLM concentration (0.43 µg/g ww methyl mercury or 0.45 µg/g ww total mercury). Based on the assumed exposure scenario, and assuming that exposure from drinking water, air, and market basket fish remained constant, the back-calculated local fish concentration that would result in an HQ of 1.0 for the toddler is 0.19 µg/g ww total mercury. As a result of the conservative assumptions regarding local fish consumption rates, this value is significantly lower than the fish consumption guidelines recommended by regulatory agencies. Health Canada (2007) has established a guideline of 0.5 µg/g ww total mercury for commercially sold fish. Manitoba Water Stewardship indicates that sportfish with mercury concentrations ranging from 0.5 to 1.0 µg/g can be consumed up to 4 times per month by members of the general population, but women of child bearing age and children under 12 years old should only consume fish with concentrations below 0.5 µg/g (Manitoba, 2007). Similarly, Saskatchewan Environment and Resource Management (SERM, 1999) indicates that fish with an average mercury concentration of less than 0.5 µg/g may be eaten in unlimited amounts and that children and pregnant women should not consume fish containing mercury concentrations in excess of 0.5 µg/g (SERM, 1999). The 95% UCLM concentration for each fish species, independent of lake where caught, was below the 0.5 µg/g total mercury guideline, with the highest values for walleye (0.45 µg/g) and lake trout (0.49 µg/g) (refer to Chapter 4, Table 4-8). In addition, of the 11 lakes included in the Fish Study, the 95% UCLM concentration of all fish caught for three lakes (0.56 µg/g in Big Island, 0.60 µg/g in Denare Beach, and 0.54 µg/g in Hamell lake) was in excess of 0.5 µg/g guideline (refer to Chapter 4, Table 4-9). It was also noted that there does not seem to be link between methyl mercury fish concentrations and proximity to the facility.

Of the 212 local fish collected and analyzed for total mercury, 17 (or 8%) contained concentrations above the 0.5 µg/g ww guideline, and 80 (or 38%) contained concentrations above the HHRA-derived concentration associated with an HQ of 1.0 (0.19 µg/g ww). Based on this assessment, it is recommended that fish consumption advisories be considered for this area, particularly for sensitive receptors.

5.2.6 Selenium

Estimated Exposure to Selenium

The primary source of exposure to selenium for residents of each COI was through the consumption of market basket food items. Market basket foods accounted for 73 to 75% of the total daily exposure for a toddler (Figure 5-32). Although market basket exposure was assumed to be the same for residents in each of the four COI, the percent contribution of market basket foods to total exposure differed slightly as a result of differences from exposure to community-specific environmental media concentrations (*i.e.*, soil, dust, air, and drinking water). Selenium content in market basket food items is unrelated to environmental contamination in the Flin Flon area and is reflective of foods consumed throughout Canada and North America. Direct exposure to soil and dust was a very minor source of selenium exposure. For the toddler, direct soil/dust exposure accounted for only 0.3 to 2.5% of the total daily selenium exposure. Differences in the contribution of soil and dust to the total daily selenium exposure are a reflection of the different concentrations of selenium measured in COI-specific soils. The contribution of selenium from air, drinking water, home garden vegetables, and local blueberries was also minor. Exposure resulting from ingestion of local fish accounted for approximately 19% of the daily exposure for a toddler. This exposure is based on an assumed local fish consumption rate of 1.5 meals per week for 52 weeks per year. Assuming that receptors consume one serving of local wild game meat per week for 52 weeks per year resulted in approximately 3% of the predicted daily exposure for a toddler.

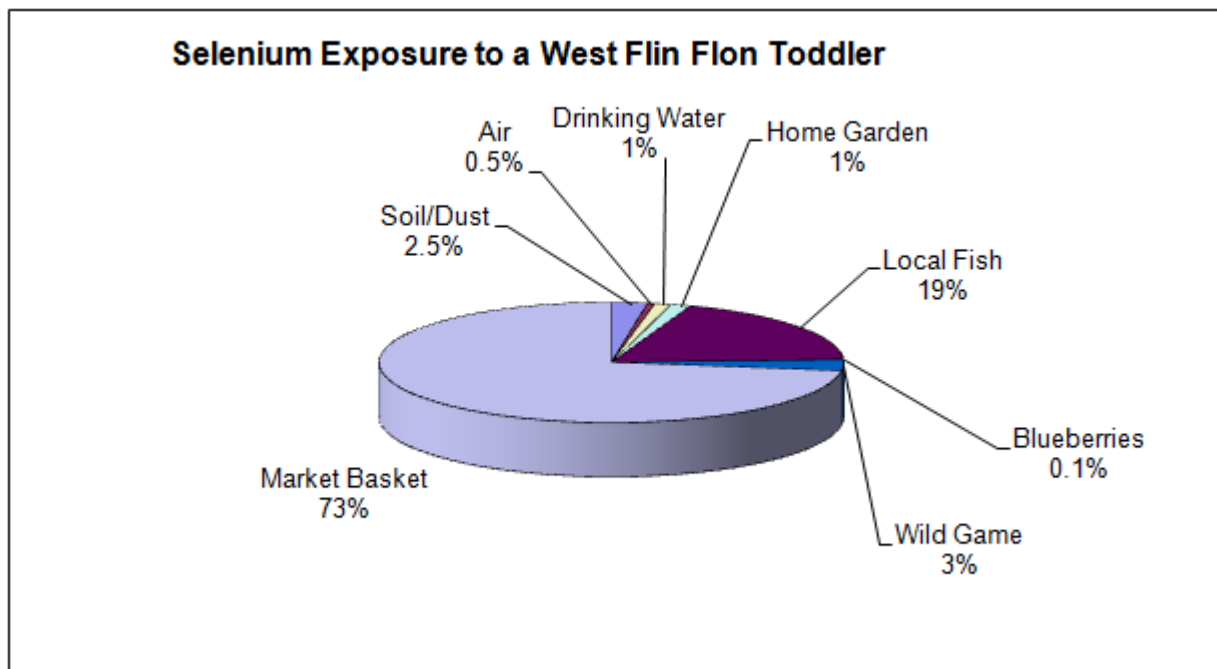


Figure 5-32 Contributions of Media to Total Selenium Exposure for a Toddler Living in West Flin Flon

A detailed breakdown of the pathway-specific exposures for residents of West Flin Flon in each of the 5 age classes is provided in Table 5-37. Details for receptors in other communities are provided in Appendix M.

Exposure Pathway	Environmental Media Concentrations		Percent of Lifetime Total EDI	Estimated Daily Intakes (EDI) (µg/kg bw/day)					
	Value	Units		Infant	Toddler	Child	Teen	Adult	Lifetime
Inhalation of Fine Particulate	0.052	µg/m ³	0.48%	0.013	0.029	0.023	0.014	0.012	0.014
Dermal Contact – Outdoors	39	µg/g	0.0047%	0.00030	0.00022	0.00016	0.00013	0.00013	0.00014
Dermal Contact – Indoors	10	µg/g	0.00043%	0.000032	0.000022	0.000016	0.000012	0.000011	0.000012
Outdoor Soil Ingestion	39	µg/g	0.52%	0.063	0.126	0.016	0.009	0.007	0.015
Indoor dust Ingestion	10	µg/g	0.071%	0.0085	0.0170	0.0021	0.0012	0.0010	0.0021
Home Garden Root Vegetables	0.3	µg/g ww	0.48%	0.0058	0.026	0.022	0.017	0.012	0.014
Home Garden Other Vegetables	0.3	µg/g ww	1.1%	0.051	0.054	0.045	0.030	0.029	0.032
Local Wild Blue Berries	0.1	µg/g ww	0.25%	0.0074	0.0074	0.0074	0.0074	0.0074	0.0074
Local Wild Game	0.37	µg/g ww	5.7%	0	0.17	0.17	0.17	0.17	0.17
Local Fish	1.6	µg/g ww	37%	0	1.1	1.1	1.1	1.1	1.1
Drinking Water	1.8	µg/L	1.4%	0.066	0.065	0.044	0.030	0.038	0.040
Market Basket Contribution	NA	µg/g	53%	3.6	4.2	2.9	1.7	1.1	1.5
Summary									
Estimated Daily Intake (µg/kg/day)			--	3.8	5.7	4.3	3.1	2.5	2.9
Inhalation Route Only			0.48%	0.013	0.029	0.023	0.014	0.012	0.014
Direct Soil/Dust Contact			0.60%	0.072	0.14	0.018	0.010	0.0080	0.017
Market Basket Foods			53%	3.6	4.2	2.9	1.7	1.1	1.5
Drinking Water			1.4%	0.066	0.065	0.044	0.030	0.038	0.040
Local Foods (Home Garden, Blueberries, Wild Game and Fish)			44%	0.064	1.3	1.3	1.3	1.3	1.3

Hazard Quotients

HQ estimates for residents living in each of the COI ranged from 0.43 to 0.92, with risk levels consistent throughout each of the COI (Table 5-38). All HQs were below the acceptable value of 1.0 indicating that adverse effects associated with elevated exposure to selenium at the EPCs are not anticipated. These HQs were derived using the EPCs for several environmental media selected to be representative of typical exposure to residents in each of the COI. Receptors living at locations with concentrations of selenium in media that are higher or lower than the EPC are anticipated to be subject to risks that are subsequently higher or lower than those predicted for the general community population. Risks to the toddler are higher than those predicted for other receptors partly as a result of the elevated soil ingestion rate assumed for children of this age.

<i>Receptor</i>	<i>East Flin Flon</i>	<i>West Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>	<i>Typical Background</i>
Infant	0.68	0.70	0.68	0.68	0.67
Toddler	0.90	0.92	0.90	0.90	0.70
Child	0.68	0.68	0.68	0.68	0.48
Teen	0.49	0.50	0.49	0.49	0.30
Adult	0.44	0.44	0.43	0.44	0.22

Due to the significant contribution of market basket foods to the total selenium exposure, predicted risk levels for receptors living in each of the COI are similar to those predicted under the Typical Background scenario (Figure 5-33).

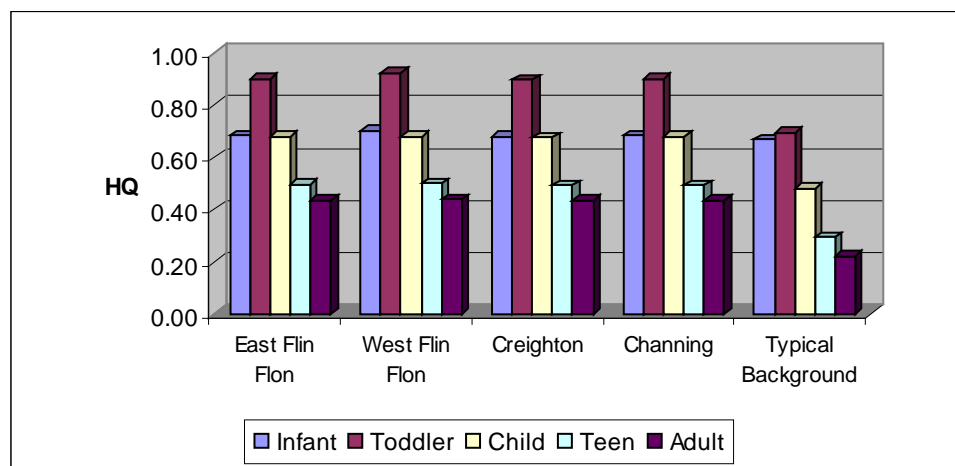


Figure 5-33 Predicted Hazard Quotients as a Result of Exposure to Selenium

Exposure to selenium, and subsequently risk levels, are dominated by contributions from market basket foods (Figure 5-34). The next highest contributions are from the consumption of local fish and wild game, which are assumed to be the same for residents of all four COI. Although the predicted HQs for the infant and toddler are approaching a value of 1.0, increasing or decreasing exposure to selenium in soil/dust, air, drinking water, and local foods would not have a significant effect on the overall risk levels.

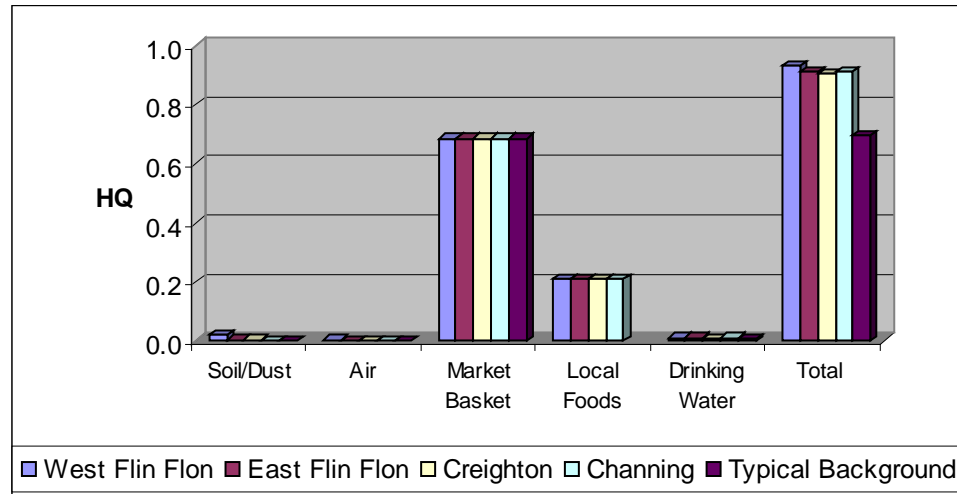


Figure 5-34 Pathway-Specific Hazard Quotients for a Toddler in each COI as a Result of Exposure to Selenium

Derivation of a Residential Selenium Soil PTC

Although exposure to selenium through the inhalation of particulates in air and from the consumption of drinking water may vary from community to community, these sources have a relatively minor contribution to total selenium intake. The exposure to selenium through the consumption of market basket and local foods is notably higher however, contribution from these sources is anticipated to be similar throughout each of the four COI. It can therefore be reasoned that the derivation of a single selenium soil PTC should be applicable to each of the four COI. Since concentrations of selenium in air are highest in West Flin Flon, environmental parameters associated with this community were used to back-calculate a soil PTC that is protective of an HQ of 1.0. An iterative process was used in which exposure to selenium from air, drinking water, local foods, and market basket foods remained constant while the concentration in soil was adjusted to produce an HQ of 1.0. Since concentrations of selenium in indoor dust are a function of the concentrations in outdoor soil, dust concentrations were automatically adjusted during this process.

Given that the chronic oral TDI for the toddler is 6.2 $\mu\text{g}/\text{kg}/\text{day}$ and the predicted exposure from market basket foods is 4.2 $\mu\text{g}/\text{kg}/\text{day}$, the RTDI to be allocated to exposure from soil/dust, drinking water, local foods, and air is 2.0 $\mu\text{g}/\text{kg}/\text{day}$. The predicted daily exposure of a toddler living in West Flin Flon *via* drinking water (0.065 $\mu\text{g}/\text{kg}/\text{day}$), local foods (1.3 $\mu\text{g}/\text{kg}/\text{day}$), and air (0.029 $\mu\text{g}/\text{kg}/\text{day}$) totals 1.4 $\mu\text{g}/\text{kg}/\text{day}$. Assuming that exposure to selenium remains constant from all sources other than soil/dust, the RTDI to be allocated to soil/dust *via* ingestion and dermal exposure is 0.6 $\mu\text{g}/\text{kg}/\text{day}$ for the toddler. Conservatively assuming 100% bioaccessibility of selenium in soil, a soil concentration of 170 $\mu\text{g}/\text{g}$ (and an associated indoor dust concentration of 33 $\mu\text{g}/\text{g}$) produces a soil/dust-related exposure of 0.6 $\mu\text{g}/\text{kg}/\text{day}$ and results in a total HQ of 1.0 (Table 5-39). Although there is a significant level of uncertainty associated with predicting the EDI for market basket foods (*i.e.*, variability and uncertainty associated with food category-specific ingestion rates and selenium concentrations), the daily EDI predicted in the current assessment (4.2 $\mu\text{g}/\text{kg}/\text{day}$ for the toddler) is equal to the EDI predicted by the CCME and used in the derivation of the human health component of the residential soil standard.

<i>Media</i>	<i>Exposure (µg/kg/day)</i>	<i>Media-Specific HQ</i>
Air	0.029	0.0047
Drinking Water	0.065	0.010
Local Fish	1.1	0.18
Local Wild Game	0.17	0.027
Blue Berries	0.0078	0.0012
Home Garden Vegetables	0.08	0.013
Market Basket	4.2	0.68
EDI without Soil/Dust	5.6	0.91
TDI	6.2	--
RTDI allocated to Soil/Dust	0.6	0.097

A comparison of the selenium soil PTC of 170 µg/g with the results of the residential soil sampling program indicates that one sample (286 µg/g in West Flin Flon) was in excess of the PTC (Table 5-40). Although this property contained a concentration of selenium in excess of the PTC, the HQ for the toddler associated with this concentration is 1.1 assuming chronic exposure to this level. Given this, and the minor contribution of soil and dust to the total daily selenium exposure, levels of selenium in Flin Flon area soils are not anticipated to pose health risks to human health within any of the COI.

	<i>West Flin Flon</i>	<i>East Flin Flon</i>	<i>Creighton</i>	<i>Channing</i>	<i>Total</i>
# of Properties Sampled	77	66	30	10	183
# of Properties >170 µg/g	1 (1.3%)	0	0	0	1 (0.55%)

Overall, the health risks to Flin Flon area residents associated with exposure to selenium are expected to be similar to those observed in other parts of Canada and are within risk levels deemed to be acceptable by Health Canada and the CCME.

5.3 Outdoor Commercial/Industrial Workers

Since concentrations of COC in outdoor soil are generally much higher than concentrations measured in indoor dust, and some occupations may require individuals to spend a significant amount of time outdoors, an outdoor worker scenario was evaluated in the HHRA in addition to the residential scenario. Under this scenario, it was conservatively assumed that 100% of an adult worker's daily incidental ingestion and dermal contact exposure was to outdoor soil rather than allocating a portion to indoor dust as done under the residential scenario. This was assumed to occur throughout the year, even during winter months when it is anticipated that snow cover will significantly restrict exposure to outdoor soil. Workers were assumed to spend 10 hours per day, 5 days per week, 48 weeks per year working outdoors. Workers were assumed to be exposed to the 95% UCLM soil concentrations measured on non-residential properties within the Manitoba Conservation (2007) Soils Study (Table 5-41).

COC	95% UCLM Concentration
Arsenic	57
Cadmium	16
Copper	1300
Lead	270
Mercury	63
Selenium	18

In addition to exposure resulting from incidental ingestion and dermal contact with outdoor soil, workers were assumed to be exposed to COC in ambient outdoor air and drinking water (based on EPCs for West Flin Flon), and market basket foods. Pathway-specific exposures for the outdoor worker scenario are provided in Table 5-42.

Pathway	Arsenic	Cadmium	Copper	Lead	Mercury	Selenium	Methyl Mercury
Soil - Dermal Contact	0.0018	0.000017	0.0014	0.0017	0.0033	0.000038	-
Soil - Ingestion	0.0035	0.0030	0.24	0.029	0.012	0.0034	-
Air - Inhalation	0.0051	0.0043	0.051	0.021	0.00078	0.0032	0.00020
Market Basket Foods	0.065	0.18	15	0.17	0.017	1.2	0.067
Drinking Water	0.042	0.018	7.3	0.064	0.00059	0.025	0.00020
Total	0.012	0.21	23	0.29	0.10	1.3	0.07

Non-Cancer Risk Estimates for the Outdoor Worker Scenario

No unacceptable risks were predicted for any COC under an outdoor worker scenario for non-cancer effects (Table 5-43). Given that this scenario assumed that a worker would be exposed to the 95% UCLM concentration in soil every day spent at work during summer and winter months, it is anticipated that COC in soils found throughout the Flin Flon-Creighton area will not result in non-cancer health effects as a result of outdoor occupational activities.

Pathway	Arsenic	Cadmium	Copper	Lead	Mercury	Selenium	Methyl Mercury
Soil - Dermal Contact	0.0064	0.000017	0.000014	0.00048	0.011	0.0000067	-
Soil - Ingestion	0.012	0.0030	0.0024	0.0081	0.039	0.00059	-
Air - Inhalation	0.017	0.0043	0.00051	0.0058	0.0027	0.00056	0.00042
Market Basket Foods	0.22	0.18	0.15	0.046	0.056	0.22	0.14
Drinking Water	0.14	0.018	0.073	0.018	0.0020	0.0044	0.00042
Total	0.39	0.20	0.23	0.078	0.11	0.22	0.14

Cancer Risk Estimates for the Outdoor Worker Scenario

Exposure to arsenic and cadmium have been associated with elevated occurrences of various forms of cancer. The assessment of carcinogenic risks *via* the inhalation of arsenic and cadmium in ambient air for the outdoor worker would be similar to the assessment provided under the residential exposure scenario. In addition, the assessment of carcinogenic risks to arsenic *via* consumption of drinking water and market basket foods would be the same as those predicted under the residential scenario.

Under the outdoor worker scenario, chronic exposure to the 95% UCLM soil concentration is not anticipated to result in CRLs above the acceptable level of 1.0×10^{-5} (Table 5-44). This conservatively assumes that a receptor would spend their entire adult life working outdoors in the Flin Flon-Creighton area.

Pathway	ILCR
Soil - Dermal Contact	2.7E-06
Soil - Ingestion	5.2E-06
Total Soil-Related ILCR	7.9E-06

Conclusions and Recommendations

Overall, despite the assumption that outdoor workers will spend a significant amount of time outdoors, no unacceptable cancer or non-cancer risks are anticipated for individuals working (but not residing) in the Flin Flon area. Concentrations of COC on commercial/industrial properties are not anticipated to result in adverse health effects assuming that adults are the only receptors that would be chronically exposed to soils on these properties. As described under the residential scenario, the residential PTCs are recommended for all properties that are to be used for daycares, retirement homes, schools, residential properties, and parkland.

5.4 Recreational Exposure Pathways

Under a supplemental recreational assessment, it was assumed that receptors may spend a significant portion of the summer months swimming in local lakes. Exposure to COC was assumed to occur *via* incidental ingestion of surface water and sediment, as well as dermal contact of surface water with all skin. Within this assessment, the maximum concentrations of COC in surface water and the 95% UCLM concentrations of sediments measured in lakes sampled as part of the Fish Study (Stantec, 2009) were used to predict exposure while swimming (Table 5-45).

Pathway	Arsenic	Cadmium	Copper	Lead	Mercury	Selenium
Oral Exposure to Surface Water						
Toddler	0.0046	0.0013	0.011	0.00057	0.0027	0.0029
Child	0.0023	0.00063	0.0055	0.00029	0.0014	0.0014
Teen	0.00094	0.00026	0.0022	0.00012	0.00056	0.00059
Adult	0.00060	0.00017	0.0014	0.00008	0.00036	0.00038
Composite	0.0010	0.00028	0.0024	0.00013	0.00061	0.00063
Dermal Exposure to Surface Water						
Toddler	0.00056	0.00015	0.0013	0.000070	0.00034	0.00035
Child	0.00047	0.00013	0.0011	0.000058	0.00028	0.00029
Teen	0.00029	0.000080	0.00069	0.000036	0.00017	0.00018
Adult	0.00021	0.000059	0.00051	0.000027	0.00013	0.00013
Composite	0.00026	0.000072	0.00062	0.000033	0.00016	0.00016

<i>Pathway</i>	<i>Arsenic</i>	<i>Cadmium</i>	<i>Copper</i>	<i>Lead</i>	<i>Mercury</i>	<i>Selenium</i>
Oral Exposure to Sediment						
Toddler	0.015	0.060	1.0	0.14	0.0010	0.040
Child	0.0019	0.0075	0.13	0.018	0.00013	0.0050
Teen	0.0010	0.0041	0.072	0.010	0.000072	0.0028
Adult	0.00087	0.0035	0.060	0.0085	0.000060	0.0023
Composite	0.0018	0.0071	0.12	0.017	0.00012	0.0047
Total Exposure						
Toddler	0.020	0.061	1.0	0.15	0.0010	0.043
Child	0.0046	0.0082	0.14	0.019	0.00014	0.0067
Teen	0.0023	0.0045	0.074	0.010	0.000075	0.0035
Adult	0.0017	0.0037	0.062	0.0086	0.000063	0.0028
Composite	0.0030	0.0074	0.12	0.017	0.00013	0.0055

All HQs associated with incidental ingestion of surface water and sediment, and dermal contact with surface water, while swimming were very minor and are not considered to significantly contribute to overall risks for residential receptors or visitors to the Flin Flon-Creighton area (Table 5-46).

<i>Pathway</i>	<i>Arsenic</i>	<i>Cadmium</i>	<i>Copper</i>	<i>Lead</i>	<i>Mercury</i>	<i>Selenium</i>
Oral Exposure to Surface Water						
Toddler	0.015	0.0013	0.00012	0.00016	0.0092	0.00046
Child	0.0077	0.00063	0.000055	0.000080	0.0046	0.00023
Teen	0.0031	0.00026	0.000022	0.000033	0.0019	0.000094
Adult	0.0020	0.00017	0.000014	0.000021	0.0012	0.000066
Dermal Exposure to Surface Water						
Toddler	0.0019	0.00015	0.000015	0.000020	0.0011	0.000057
Child	0.0016	0.00013	0.000011	0.000016	0.00093	0.000046
Teen	0.0010	0.000080	0.0000069	0.000010	0.00058	0.000029
Adult	0.00071	0.000059	0.0000051	0.0000074	0.00043	0.000023
Oral Exposure to Sediment						
Toddler	0.050	0.060	0.012	0.040	0.0034	0.0064
Child	0.0062	0.0075	0.0013	0.0051	0.00043	0.00079
Teen	0.0034	0.0041	0.00072	0.0028	0.00024	0.00044
Adult	0.0029	0.0035	0.00060	0.0024	0.00020	0.00041
Total Exposure						
Toddler	0.067	0.061	0.012	0.041	0.0035	0.0070
Child	0.015	0.0082	0.0014	0.0052	0.00046	0.0011
Teen	0.0075	0.0045	0.00075	0.0028	0.00025	0.00057
Adult	0.0056	0.0037	0.00062	0.0024	0.00021	0.00050

Carcinogenic Risks Associated with Exposure to Arsenic While Swimming were also considered very minor (Table 5-47).

<i>Pathway</i>	<i>ILCR</i>
Surface Water and Sediment - Ingestion	4.1×10^{-6}
Surface Water - Dermal	3.9×10^{-7}
Total Swimming-Related ILCR	4.6×10^{-6}

Therefore, cancer and non-cancer risks associated with receptors swimming in lakes in the Flin Flon-Creighton area throughout the summer months are considered to be very minor and are not anticipated to result in the occurrence of adverse health effects.

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