CHAPTER 4

DETAILED HUMAN HEALTH RISK ASSESSMENT METHODOLOGY

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4.0 DETAILED HUMAN HEALTH RISK ASSESSMENT METHODOLOGY

The detailed HHRA was conducted using the data collected from Phases 1 and 2, and followed the four major steps of the HHRA framework: i) problem formulation; ii) exposure assessment; iii) hazard assessment; and, iv) risk characterization. The problem formulation step (Phase 1) was previously discussed in detail in Chapter 2, while the additional sampling and analytical work conducted to fill identified data gaps (Phase 2) was outlined in Chapter 3 of this volume.

Included within this chapter are two of the three remaining steps of the HHRA; the exposure assessment and the hazard assessment. The exposure assessment provides a detailed discussion of the methodologies used to estimate exposure under each scenario evaluated as part of the HHRA. The hazard assessment provides details of the toxicological effects associated with exposure to each of the COC and indicate which toxicity reference values (TRVs) were selected to be applied within the risk characterization step. This information was also used in the derivation of provisional trigger concentrations (PTCs). A PTC can be defined as the average COC soil concentration within an exposure unit that corresponds to an acceptable level of risk. Soil PTCs have been derived to assist in determing if biomonitoring may be appropriate to more accurately assess exposure. The PTCs are derived to help focus the efforts of a biomonitoring program on those areas or properties that may be of the greatest concern. Additional details on PTCs are provided in Chapter 5.

4.1 Exposure Assessment

The exposure assessment evaluates data related to all COC, receptors and exposure pathways identified during the problem formulation phase of the HHRA using a multimedia approach. The multimedia approach takes into account all potential exposure to COC from the different sources or media (*i.e.,* soil, air, dust, water, food, *etc*.) which receptors could come in contact with as part of their daily activities.

The primary objective of the exposure assessment is to predict, using site-specific data and a series of conservative assumptions, the rate of exposure (*i.e.,* the quantity of chemical and the rate at which that quantity is received) of the selected receptors to COC *via* the various exposure scenarios and pathways identified in the problem formulation step. The rate of exposure to chemicals from the various pathways is usually expressed as the amount of chemical taken in per body weight per unit time (*e.g.,* µg chemical/kg body weight/day). The assessment of risk *via* the inhalation pathway can be assessed through a direct comparison of ambient air concentrations to health-based air standards.

The degree of exposure of receptors to chemicals in the environment depends on the interactions of a number of parameters, including:

- The concentrations of COC in various environmental media;
- The physical-chemical characteristics of the COC which affect their environmental fate and transport and determine such factors as efficiency of absorption into the body of a given external exposure;
- The influence of site-specific environmental characteristics, such as geology, soil type, topography, hydrology, hydrogeology, local meteorology and climatology *etc.* on a chemical's behaviour within environmental media; and,
- The physiological and behavioural characteristics of the receptors (*e.g.,* respiration rate, soils/dust intake, time spent at various activities and in different areas).

The derivation of the exposure point concentrations (EPCs) for COC in each media type used to assess exposure is described in Section 4.1.1.

4.1.1 Media Concentration Data Selected for Use in the HHRA

The derivation of an appropriate EPC (*i.e*., the concentration of a chemical in any environmental medium to which a receptor could reasonably be expected to be exposed over an extended period of time) is important to the overall exposure assessment. As recommended by the U.S. EPA Risk Assessment Guidance for Superfund (U.S. EPA, 1989), the upper 95% confidence interval on the arithmetic mean of the data set (*i.e*., the 95% UCLM) should be used to represent the EPC. This is considered to be a reasonable estimate of the concentration to which a receptor might be exposed over a significant amount of time. When enough data are present, the 95% UCLM incorporates the central tendency (*i.e*., the arithmetic mean) and the uncertainty surrounding the arithmetic mean.

EPCs were developed for each COC in all environmental media of concern. All data that were less than the method detection limit (MDL) were conservatively assumed to be present at the MDL value unless otherwise indicated. The 95% UCLM concentrations were calculated using ProUCL, software developed by Lockheed Martin under contract with the U.S. EPA. ProUCL tests the data set for normality, lognormality, and gamma distributions using parametric and non-parametric methods to calculate a conservative and stable 95% UCLM (U.S. EPA, 2004b). The U.S. EPA has peer reviewed this software and endorses its use as a tool in contaminated site risk assessments. ProUCL output summaries for each COC in all environmental media are provided in Appendix N.

4.1.1.1 Surface Soil Concentrations

Data collected as part of the Jacques Whitford (JW) residential soil sampling program was used to derive EPCs for COC in soil for each of the four communities of interest (COI) assessed in the HHRA (*i.e*., West Flin Flon, East Flin Flon, Creighton, and Channing). All data were separated into each of the four COI. For sampling locations that had multiple samples taken (*i.e*., from the front yard, back yard, garden, and/or sandbox), an arithmetic mean of these values was used to represent the concentration for that location. Since the EPCs were used to estimate exposure *via* incidental ingestion and direct dermal contact with residential soils, only surface soil samples were considered in the derivation of the EPCs (*i.e*., samples collected in the 0-2.5 or 0-5 cm bgs profile). The EPCs represent the 95% UCLM surface soil concentration for each community (Table 4-1).

Table 4-1 Exposure Point Concentrations for COC Measured in Residential Surface Soil (µg/g)

Location	*Arsenic*	*Cadmium*	*Copper*	*Lead*	*Mercury*	*Selenium*
West Flin Flon						
# Samples	77	77	77	77	77	77
Mean	67	25	2,157	332	106	32
Maximum	237	71	7,810	820	971	286
EPC (95% UCLM)	77	28	2,800	370	130	39
East Flin Flon						
# Samples	63	63	63	63	63	63
Mean	16	14	794	141	6	4.1
Maximum	33	27	2,050	333	18	12
EPC (95% UCLM)	17	16	870	160	7.2	4.6
Creighton						
# Samples	29	29	29	29	29	29

Table 4-1 Exposure Point Concentrations for COC Measured in Residential Surface Soil (µg/g)						
Location	***Arsenic***	***Cadmium***	***Copper***	***Lead***	***Mercury***	***Selenium***
Mean	64	14	689	164	6.0	5.6
Maximum	300	32	1,800	456	24	18
EPC (95% UCLM)	88	16	850	250	8.9	7.2
Channing						
# Samples	10	10	10	10	10	10
Mean	17	10	384	112	2.8	2.0
Maximum	36	21	700	266	7.0	4.0
EPC (95% UCLM)	22	14	510	160	4.1	2.6

4.1.1.2 Ambient Air Concentrations

The combined influence of local wind patterns in the Flin Flon-Creighton area and the proximity to the HBMS complex creates unique conditions for different communities within this area. While residents from these communities may spend time working or spending leisure time away from their primary residence, their exposure to COC in ambient air may be most accurately characterized using concentrations measured near their home. To address the variability in ambient air concentrations throughout the study area, the selection of ambient air data for use in the exposure assessment for each of the COI was based on their proximity to ambient air monitoring stations and the influence of the predominate wind direction on the dispersion of emissions from the HBMS complex.

Given that the most frequent wind direction is from the northwest (approximately seven months of the year), ambient air in the western portion of Flin Flon (*i.e.*, west of Ross Lake) is likely heavily influenced by direct releases of emissions from the stack. Air monitors stationed on the Provincial building and Ruth Betts School are located downwind of the HBMS complex and data collected at these stations are considered to be reflective of exposure conditions for residents of the community of West Flin Flon (refer to Figure 3-1). Based on a comparison of metals associated with Total Suspended Particulates (TSP) at the Provincial building and Ruth Betts, concentrations of several metals are higher in samples collected at the Provincial building. For the purpose of estimating conservative EPCs for residents of West Flin Flon, the HHRA utilized data collected from the monitors located on the Provincial building. Although only TSP data are available for this location, chemical-specific correlating factors based on historical paired TSP and PM_{10} data from the Provincial Building were used to predict concentrations of COC associated with the respirable PM_{10} fraction for the community of West Flin Flon based on recent TSP measurements at the Provincial Building. This is described in further detail below.

The community of East Flin Flon (*i.e.*, east and northeast of Ross Lake) is not likely influenced by direct stack emissions to the same extent as West Flin Flon as a result of the predominate wind direction from the northwest. Since there are no air monitoring stations located to the north or east of Ross Lake, data collected from air monitors outside of this community must be utilized in the HHRA. Given that the Provincial building is located directly downwind from the HBMS complex and is the closest in proximity to the stack, it is not anticipated that data collected from this monitor are an accurate reflection of ambient air quality in East Flin Flon. Therefore, the HHRA utilized data collected from the monitors located on Ruth Betts to estimate long-term ambient air EPCs for residents of the community of East Flin Flon. These data were also used to represent air quality for residents of Channing located southeast of Ruth Betts.

The community of Creighton is located to the southwest of the HBMS complex. Although it is not directly downwind of the HBMS complex and will not receive the same level of direct smelter

emissions as the community of West Flin Flon, it does receive particulate loadings from wind-blown dusts originating from the tailings impoundment. Data collected from air monitors located on the Creighton School are considered to provide an accurate representation of ambient air quality in this community and were utilized within the HHRA.

HBMS currently operates air sampling stations at Ruth Betts School in Flin Flon and at Creighton School in Creighton. HBMS reports data for TSP, PM_{10}, and $PM_{2.5}$ and metals associated with each of these fractions. Manitoba Conservation operates a monitor on the Provincial building and analyzes for TSP and metals associated with TSP. As described in the *Correlation of Metal Content in TSP and PM_{10}* supplement provided in Appendix I, the concentrations of COC associated with the PM_{10} component of outdoor air is the most relevant for predicting exposure *via* the inhalation pathway (Health Canada, 2006a).

Prior to the initiation of the HHRA, the HBMS monitoring program included the analysis of only four of the six COC (*i.e.*, arsenic, cadmium, copper, and lead). Beginning in September 2007, HBMS increased the number of metals to be analyzed to include all six COC. Samples were collected on a weekly basis. For the HHRA, the 95% UCLM concentration was calculated for each COC associated with the PM_{10} component in samples collected at Ruth Betts (n=57) and Creighton School (n=59) for one year from September 2007 to August 2008. Measured concentrations were adjusted by subtracting the concentrations of each COC measured in the filter blanks. Use of the most recent data was considered to be the most relevant for the purpose of predicting exposure and risk under current conditions and moving into the future. For comparative purposes however, the annual average concentrations dating back to 2002 were calculated and are summarized in Table 4-2. Based on annual average concentrations of arsenic, cadmium, copper, lead, and zinc from 1998 to 2007/2008, concentrations have generally declined or remained constant over this period.

Table 4-2 Annual Average COC Concentrations Associated with PM10 in Ambient Air (µg/m^3)

Location	*Arsenic*	*Cadmium*	*Copper*	*Lead*
Ruth Betts School				
1998	0.09	0.11	0.42	0.81
1999	0.04	0.06	0.23	0.60
2000	0.04	0.05	0.17	0.41
2001	0.05	0.06	0.50	0.24
2002	0.02	0.02	0.35	0.15
2003	0.04	0.02	0.33	0.16
2004	0.01	0.00	0.13	0.03
2005	0.02	0.01	0.18	0.07
2006	0.02	0.02	0.13	0.06
2007/2008[b]	0.02	0.01	0.10	0.05
Creighton School[a]				
2003	0.01	0.04	0.15	0.42
2004	0.01	0.04	0.13	0.42
2005	0.01	0.06	0.13	0.42
2006	0.01	0.01	0.05	0.17
2007/2008[b]	0.005	0.002	0.05	0.18

[a] Measurements are from the dichotomous air sampler.
[b] Samples were collected from September 2007 to August 2008.

For the derivation of EPCs, recent PM_{10} data are available from monitors located on Ruth Betts to be used for the communities of East Flin Flon and Channing, and from monitors located on

the Creighton School, to be used for the community of Creighton. However, as discussed previously, the monitors located on the Provincial building currently only collect data associated with TSP. Since it is recommended that data collected from the Provincial building be used in the exposure assessment for the community of West Flin Flon, concentrations of COC measured in TSP at the Provincial building will be adjusted to estimate concentrations that are anticipated to be associated with the PM_{10} component. As described in the *Correlation of Metal Content in TSP and PM_{10}* supplement provided in Appendix I, correlating factors were developed by using concurrent TSP and PM_{10} monitoring data from the Provincial building for arsenic, cadmium, copper, and lead from March 1991 to December 1998. These data were used to establish correlating factors that express the concentration of a specific metal in PM_{10} as a function of the concentration of the same metal in TSP (Table 4-3). These factors allowed the HHRA to use the most recent TSP data collected from the Provincial building to predict metal concentrations within the respirable particulate fraction in the assessment of inhalation exposure to residents of West Flin Flon.

The analysis of this data indicates that this approach is an effective tool to predict metal concentrations in PM_{10} assuming that the current relationship between PM_{10} and TSP is relatively consistent with that of the 1990s. Since TSP data collected by Manitoba Conservation at the Provincial Building is only available for arsenic, cadmium, copper, and lead, concentrations of mercury and selenium in ambient air for the community of West Flin Flon will be based on measurements of content in PM_{10} at Ruth Betts and the Creighton School.

Table 4-3 Correlating Factors Used to Predict COC Content of PM10 Based on Concentrations Measured in TSP

Metal	***PM_{10} / TSP Factor***
Arsenic	0.85
Cadmium	0.90
Copper	0.56
Lead	0.89

Concentrations of arsenic, cadmium, copper, and lead associated with TSP as measured at the Provincial Building from June 2007 to June 2008 (n=210) were converted to predicted concentrations associated with the PM_{10} fraction using the correlating factors presented in Table 4-3. Manitoba Conservation indicated that concentrations of COC associated with filter blanks were at or near the MDLs, therefore, measured concentrations were not adjusted by subtracting the concentrations of each COC measured in the filter blanks. The 95% UCLM concentrations were then calculated to represent annual average EPCs for the community of West Flin Flon. These values are presented in Table 4-4 along with the 95% UCLMs generated for concentrations measured within the PM_{10} fraction at Ruth Betts, used to represent the annual average EPCs for East Flin Flon and Channing, and the Creighton School, used to represent the annual average EPCs for Creighton.

Table 4-4 Exposure Point Concentrations for COC Measured in Outdoor Air* ($\mu g/m^3$)

COC	***East Flin Flon and Channing (n=210)***	***West Flin Flon (n=210)***	***Creighton (n=59)***
Arsenic			
Mean	0.016	0.054	0.0049
Maximum	0.22	0.74	0.03
EPC (95% UCLM)	0.040	0.084	0.0085
Cadmium			
Mean	0.011	0.036	0.0023
Maximum	0.12	0.66	0.018
EPC (95% UCLM)	0.026	0.070[a]	0.0046

Table 4-4 Exposure Point Concentrations for COC Measured in Outdoor Air* (µg/m^3)			
COC	***East Flin Flon and Channing (n=210)***	***West Flin Flon (n=210)***	***Creighton (n=59)***
Copper			
Mean	0.1	0.71	0.051
Maximum	0.82	4.2	0.28
EPC (95% UCLM)	0.22	0.84	0.058
Lead			
Mean	0.046	0.19	0.018
Maximum	0.39	2.4	0.14
EPC (95% UCLM)	0.10	0.34[a]	0.034
Mercury			
Mean	0.000054	-	0.00055
Maximum	0.00032	-	0.0042
EPC (95% UCLM)	0.000094	0.016[b]	0.0013
Selenium			
Mean	0.0051	-	0.0022
Maximum	0.089	-	0.020
EPC (95% UCLM)	0.014	0.052[b]	0.0042

* Air data considered in the derivation of the EPCs 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.

[a] Concentration is the 97.5% UCLM as recommended by ProUCL.

[b] Concentration is based on the EPC predicted for Creighton adjusted according to the relationship between EPCs derived for arsenic, cadmium, copper, and lead for West Flin Flon and Creighton (*i.e.*, an adjustment factor of 12.4 was applied to the EPCs for Creighton).

Since samples collected at the Provincial Building were not analyzed for mercury or selenium, the EPCs for these COC for the community of West Flin Flon were estimated based on measured concentrations from Ruth Betts and the Creighton School. Based on a comparison of the EPC concentrations for arsenic, cadmium, copper, and lead derived for West Flin Flon and Creighton, on average, the EPCs were 12.4 times higher for West Flin Flon relative to those derived for Creighton. Applying a 12.4-fold factor to the EPCs for mercury and selenium in Creighton would produce estimated EPCs for West Flin Flon of 0.016 and 0.052 µg/m^3 for mercury and selenium, respectively. Similarly, based on a comparison of the EPC concentrations for arsenic, cadmium, copper, and lead derived for East Flin/Channing and West Flin Flon, on average, the EPCs were 3.0 times higher for West Flin Flon relative to those derived for East Flin Flon/Channing. Applying a 3.0-fold factor to the EPCs for mercury and selenium in East Flin Flon/Creighton would produce estimated EPCs for West Flin Flon of 0.00028 and 0.042 µg/m^3 for mercury and selenium, respectively. To be conservative, the higher of the EPCs predicted based on these two relationships were selected for use in the HHRA.

4.1.1.3 Indoor Air Concentrations

Indoor air concentrations were assumed to be equal to measured outdoor air concentrations. This is thought to be a conservative assumption as a number of recent studies (Chao and Wong, 2002; Komarnicki, 2005; Molnar *et al.*, 2005) demonstrate that outdoor concentrations of heavy metals can be significantly greater than measured indoor air concentrations. Lower indoor air concentrations appear to be a result of outdoor air filtration as the outdoor air infiltrates indoor environments and dilution with the existing indoor aerosol. Although there do appear to be some minor indoor sources of heavy metals, their contribution does not appear to be significant compared to the contribution of outdoor air.

4.1.1.4 Drinking Water Concentrations

Drinking water for residents of Flin Flon and Creighton is provided through separate municipal resources. Drinking water for Flin Flon is taken from Cliff Lake, which is supplied water from Trout Lake (also called Embury Lake) through active pumping. Drinking water for Creighton is taken from Douglas Lake.

HBMS provided measured concentrations of each COC in drinking water collected on a bi-weekly basis from August 2007 to July 2008 for three locations in Flin Flin (a residential location (FF-1), the Flin Flon Water Treatment Plant (FF-2), and the Vocational Centre) and one location in Creighton (a residential location (CR-1)). These data are considered to be representative of any potential variability in drinking water quality that may result from seasonal fluctuations occurring in the source water body. Since there is a limited number of sample locations included within this program, members of the Technical Advisory Committee (TAC) decided that an additional short-term study in which multiple locations throughout Flin Flon and Creighton were included in a single sampling event should be completed to ensure that these data were representative of drinking water quality throughout the communities.

Jacques Whitford conducted a residential drinking water sampling program on a number of homes, schools, and daycares in Flin Flon (36) and Creighton (11) on March 6 and 7, 2008 to assess the current metal status of tap drinking water (JW, 2008). For residential locations and daycares, drinking water samples were collected from the most frequently used tap, typically the kitchen. In schools, water samples were collected from a public water fountain (JW, 2008).

Tables 4-5 and 4-6 provide the mean, maximum, and 95% UCLM concentrations for COC in drinking water collected in Flin Flon and Creighton, respectively, as part of the HBMS bi-weekly sampling program and the JW single event sampling study. Faucets were run for a sufficient time to allow for flushing of water from plumbing prior to the collection of samples. All samples that contained COC below the MDL were assumed to be present at the MDL. Although the HBMS sampling program included two locations in Creighton, samples collected from the water treatment facility were taken from the water supply prior to treatment and distribution. Therefore, samples collected from this location were not considered to be reflective of water that would be consumed by residents and were not considered in the current assessment.

Table 4-5 Exposure Point Concentrations for COC Measured in Flin Flon Drinking Water (µg/L)

COC	*HBMS Sampling Program*			*JW Sampling Program*	*Selected EPC for HHRA*
	Flin Flon-1 (n=55)	*Flin Flon-2 (n=40)*	*Vocational Centre (n=54)*	*Sample Locations in Flin Flon (n=36)*	
Arsenic					
Mean	2.8	2.9	2.7	2.8	3.0
Maximum	3.8	3.6	3.4	3.4	
95% UCLM	2.9	3.0	2.8	2.9	
Cadmium					
Mean	1.2	1.2	1.2	1.0	1.3
Maximum	2.0	2.1	2.0	1.1	
95% UCLM	1.2	1.3	1.2	1.0	

Table 4-5 Exposure Point Concentrations for COC Measured in Flin Flon Drinking Water (µg/L)					
COC	***HBMS Sampling Program***			***JW Sampling Program***	***Selected EPC for HHRA***
	Flin Flon-1 (n=55)	***Flin Flon-2 (n=40)***	***Vocational Centre (n=54)***	***Sample Locations in Flin Flon (n=36)***	
Copper					
Mean	96	76	180	190	520
Maximum	200	120	540	2,800	
95% UCLM	100	81	200	520	
Lead					
Mean	2.5	2.1	2.8	1.5	4.6
Maximum	8.2	3.6	14	27	
95% UCLM	3.3	2.3	3.4	4.6	
Mercury					
Mean	0.052	0.053	0.051	0.1[a]	0.056
Maximum	0.13	0.1	0.08	0.1[a]	
95% UCLM	0.055	0.056	0.052	0.1[a]	
Selenium					
Mean	1.5	1.1	1.3	0.32	1.8
Maximum	8.0	3.0	6.0	0.6	
95% UCLM	1.8	1.2	1.5	0.36	

[a] All concentrations were below a laboratory detection limit of 0.1 µg/L.

With the exception of elevated maximum concentrations of copper and lead in the JW study, the mean, maximum, and 95% UCLM concentrations were fairly consistent across the HBMS sampling locations and the compiled JW results. To be conservative, the highest 95% UCLM concentration for each COC was selected as the EPC for the assessment of exposure of all Flin Flon and Channing residents *via* the consumption of drinking water. The exception to this is for mercury in which an elevated MDL was reported for all samples collected within the JW study. All concentrations were less than the MDL of 0.1 µg/L, which is significantly higher than the 95% UCLM concentrations measured at each of the three HBMS sampling locations. As a result, the next highest 95% UCLM concentration was selected as the EPC for mercury.

Table 4-6 Exposure Point Concentrations for COC Measured in Creighton Drinking Water (µg/L)			
COC	***HBMS Sampling Program***	***JW Sampling Program***	***Selected EPC for HHRA***
	Creighton- 1 (n=55)	***Sample Locations in Creighton (n=11)***	
Arsenic			
Mean	1.9	2.0	2.2
Maximum	2.4	2.7	
95% UCLM	1.9	2.2	
Cadmium			
Mean	0.2	0.31	0.89
Maximum	0.3	1.0	
95% UCLM	0.21	0.89	
Copper			
Mean	35	49	124
Maximum	91	146	
95% UCLM	40	124	
Lead			
Mean	1.8	0.78	3.1
Maximum	12	2.5	
95% UCLM	3.1	1.2	
Mercury			

Table 4-6 Exposure Point Concentrations for COC Measured in Creighton Drinking Water (µg/L)			
COC	***HBMS Sampling Program***	***JW Sampling Program***	***Selected EPC for HHRA***
	Creighton- 1 (n=55)	***Sample Locations in Creighton (n=11)***	
Mean	0.051	0.1[a]	0.052
Maximum	0.08	0.1[a]	
95% UCLM	0.052	0.1[a]	
Selenium			
Mean	1.0	0.38	1.1
Maximum	2.0	0.6	
95% UCLM	1.1	0.45	

[a] All concentrations were below a laboratory detection limit of 0.1 µg/L.

As described for the Flin Flon data, the highest 95% UCLM concentration for each COC was selected as the EPC for the assessment of exposure of all Creighton residents *via* the consumption of drinking water. The exception to this is for mercury in which an elevated MDL was reported for all samples collected within the JW study. All concentrations were less than the MDL of 0.1 µg/L, which is notably higher than the mean, maximum, and 95% UCLM concentrations measured at the HBMS sampling location. As a result, the 95% UCLM concentration measured at the HBMS sampling location was selected as the EPC for mercury.

It should be noted that concentrations of copper and lead in drinking water were significantly higher than concentrations measured in surface water, including within the lakes that serve as the source of municipal water. For example, concentrations of copper and lead measured in Embury Lake (which is the source of surface water for Flin Flon) in August 2008 were 17 and <0.5 µg/L, respectively. The EPCs for these COC in treated Flin Flon drinking water were 520 and 4.6 µg/L, respectively. It appears that plumbing is likely contributing to the overall levels of copper and lead in drinking water obtained from the tap. Although this indicates that smelter-related emissions may have a relatively minor contribution to the total copper and lead content in drinking water, contributions from local plumbing is still an important factor to consider when predicting overall community exposure to these COC.

4.1.1.5 Vegetable Garden Produce Concentrations

In 2002, Manitoba Conservation designed and completed a home garden sampling program to measure concentrations of metals in home-grown vegetables. Nine home gardens from the Flin Flon area, located at varying distances and directions from the HBMS complex, were selected to characterize the potential influence of smelter-related emissions (Figure 4-1). In addition, a garden located in the community of Cranberry Portage was selected to be representative of an area that is minimally impacted by smelter emissions. A garden in the town of The Pas was selected to represent a non-impacted control site (Jones and Henderson, 2006).

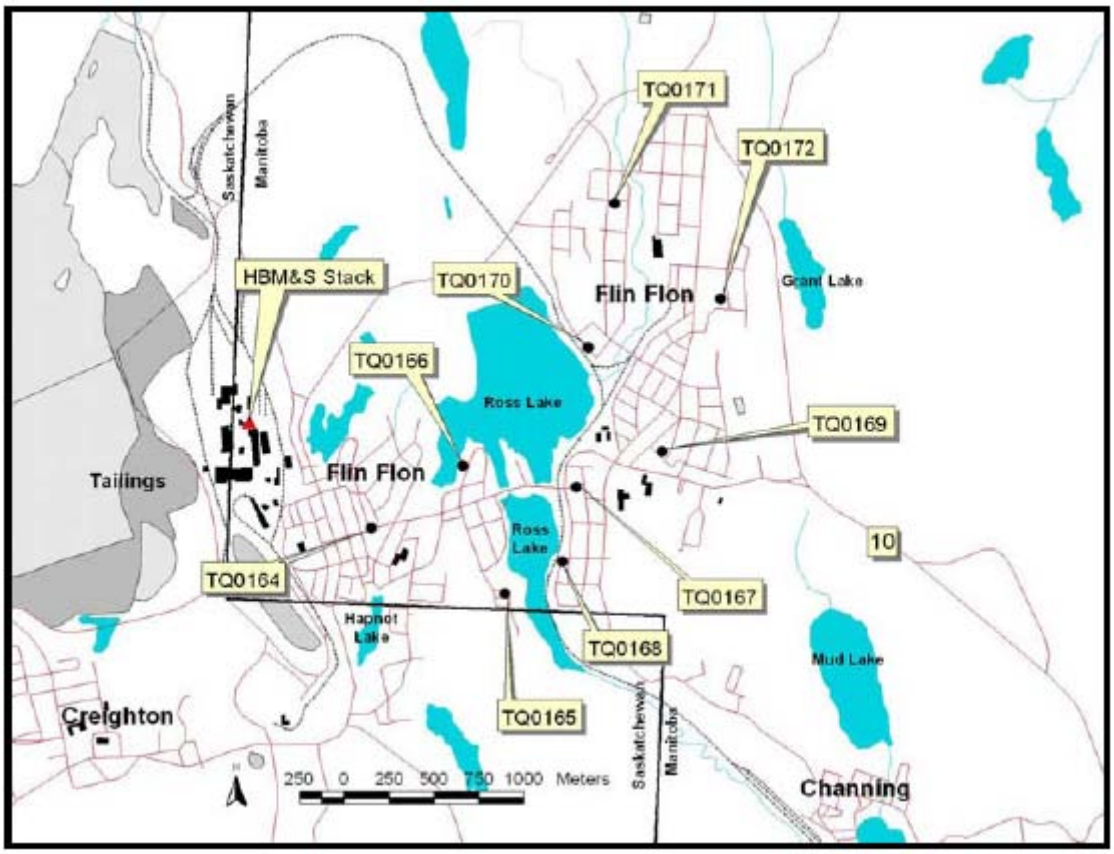

Figure 4-1 Map of Home Garden Sampling Locations Included within the Manitoba Conservation Study (Jones and Henderson, 2006)

To assess exposure and risk associated with the consumption of home garden produce, the HHRA utilized the information gathered from the Jones and Henderson (2006) study. Concentrations were provided for both washed and unwashed above-ground vegetables, however, for the purpose of deriving EPCs for the HHRA, it could be reasonably anticipated that vegetables were washed prior to consumption, therefore only washed samples were considered. Data collected from gardens in Cranberry Portage and The Pas were not included in the current assessment of exposure to residents of Flin Flon, Creighton, and Channing. Concentrations of COC were measured in lettuce (n=27), tomatoes (n=25), beans (n=23), potatoes (n=25) and carrots (n=27).

Since consumption rates recommended by Richardson (1997) are given for all “root vegetables” and all “other vegetables”, the data for carrots and potatoes were pooled to establish EPCs for root vegetables, and data for lettuce, tomatoes, and beans were pooled to establish EPCs for other vegetables. All COC concentrations that were below the MDL were conservatively assumed to be present at the MDL. Concentrations provided in dry weight were converted to wet weight concentrations using the reported sample-specific moisture content.

Since inorganic arsenic is the toxicologically relevant form for the assessment of risk, measured concentrations of total arsenic (including both organic and inorganic forms) in home garden vegetables were adjusted by multiplying the total concentration by the recommended vegetable-specific fraction of inorganic arsenic. Recommended fractions were available for beans (0.57), carrots (0.53), potatoes (0.29), and tomatoes (0.09) but not for lettuce, therefore, a fraction of

1.0 was conservatively assumed for lettuce (Schoof *et al*., 1999). The EPCs are the 95% (or the 97.5% or 99%) UCLM concentrations for above-ground (n=75) and below-ground (n=52) vegetables (Table 4-7). Due to elevated variability in some data sets, ProUCL recommended the use of 97.5% or 99% UCLMs over the 95% UCLM.

Table 4-7 Exposure Point Concentrations for COC Measured in Home Garden Vegetables (µg/g ww)

COC	*Concentration*
Above-Ground Vegetables	
Arsenic[c]	0.12[a]
Cadmium	0.24[b]
Copper	2.0
Lead	0.28[b]
Mercury	0.0082[b]
Selenium	0.30[b]
Below-Ground Vegetables	
Arsenic[c]	0.012
Cadmium	0.051
Copper	1.6
Lead	0.033
Mercury	0.0025
Selenium	0.30[b]

[a] Represents the 99% UCLM as recommended by ProUCL.
[b] Represents the 97.5% UCLM as recommended by ProUCL.
[c] Arsenic concentrations represent the inorganic fraction only.

Since data were only available for gardens in Flin Flon, the same EPCs were used to assess exposure to each of the four communities. Co-located soil concentrations are available for each garden sampled which could allow for the derivation of regressions to describe the relationship between concentrations in soil and each vegetable group sampled. This would allow for the derivation of community-specific home garden vegetable concentrations in each of the four communities. However, use of these regression equations involves an underlying assumption that the measured concentrations in produce are directly attributed to concentrations in soil and does not account for the influence of direct deposition to above-ground produce. In addition, although measured soil concentrations in front and back yards vary between each of these communities, concentrations in home garden soils are not anticipated to possess the same level of variability since home garden soils are generally amended to improve growing conditions. Therefore, use of the measured home garden vegetable concentrations in Flin Flon were considered appropriate for the assessment of exposure to residents in each of the four communities.

4.1.1.6 Fish Tissue Concentrations

Given that sport-fishing during both the summer and winter months is an important recreational activity in Flin Flon and Creighton, consumption of local fish may be a potentially significant route of exposure to metals released by the HBMS complex. When assessing exposure to chemicals as a result of the consumption of fish, there are several important factors to consider. Characterizing the concentrations of COC in fish should focus on those tissues which are most likely to be consumed by humans. While many studies include the analysis of concentrations in both liver and muscle tissue, it is common to assume that humans will generally only consume the muscle tissues and discard the organs and skin. Predicting concentrations of metals in muscle tissue based on measured concentrations in surface water and sediment can often be

problematic due to several confounding factors associated with site-specific conditions and the limited rates of accumulation of many metals. Significant uncertainty can be reduced through the collection and analysis of fish tissues collected from lakes that are known to be frequented by local fishermen.

Based on the results of the local food survey, a sampling program was developed for the collection and analysis of common sport fish from lakes at varying direction and distance from the smelter that are reported to be frequently used for fishing. This program was completed by Stantec in August 2008. Results of this program were combined with data collected by Manitoba Water Stewardship during the same sampling period. Northern pike, walleye (pickerel), lake trout, yellow perch, cisco, and whitefish were collected from 11 lakes and fillets were analyzed for each of the COC (Figure 4-2; note: Jan Lake is located outside of the range of this map). Tables 4-8 and 4-9 present the 95% UCLM concentrations for individual fish species from all lakes, and for individual lakes for all species, respectively.

Table 4-8 95% UCLM Concentrations for COC in Local Fish for Individual Species (µg/g ww)

Species	*Arsenic*[a]	*Cadmium*	*Copper*	*Lead*	*Total Mercury*	*Selenium*
Northern Pike	0.0073	0.0086	0.26	0.028	0.30	1.4
Walleye	0.0059	0.010	0.23	0.028	0.45	1.3
Yellow Perch	0.0088	0.005	0.16	0.034	0.057	2.9
Lake Whitefish	0.038	0.0095	0.52	0.024	0.026	1.7
Lake Trout	0.013	0.017	0.46	0.027	0.49	0.85
Cisco	0.013	0.005	0.47	0.02[b]	0.002[b]	4.2

[a] Concentrations of arsenic in fish tissues were adjusted to represent the inorganic fraction by applying a factor of 0.068 (representative of freshwater finfish (Schoof and Yager, 2007) to the total concentration.

[b] Indicates that all concentrations were below the MDL. The concentration presented is the MDL.

Table 4-9 95% UCLM Concentrations for COC in Fish for Individual Lakes (µg/g ww)

Lake	*Arsenic*[a]	*Cadmium*	*Copper*	*Lead*	*Total Mercury*	*Selenium*
Phantom	0.0068[b]	0.0094	0.36	0.030[b]	0.39	0.47
Bakers Narrows	0.0068[b]	0.0050[b]	0.42	0.030[b]	0.18	0.85
Amisk	0.0068[b]	0.0050[b]	0.45	0.030[b]	0.36	0.22
Big Island	0.0068[b]	0.023	0.34	0.030[b]	0.56	1.2
Denare Beach	0.0068[b]	0.0055	0.29	0.030[b]	0.60	0.23
Embury	0.022	0.024	0.54	0.030[b]	0.48	0.84
Hamell	0.012	0.017	0.19	0.030[b]	0.54	1.7
Kisseynew	0.0068[b]	0.0050[b]	0.17	0.030[b]	0.40	0.30
Jan	0.0068[b]	0.0050[b]	0.22	0.030[b]	0.30	0.19
Schist	0.0096	0.0047	0.33	0.024	0.0076	3.2
Athapapuskow	0.021	0.0049	0.31	0.021	0.23	1.3

[a] Concentrations of arsenic in fish tissues were adjusted to represent the inorganic fraction by applying a factor of 0.068 (representative of freshwater finfish (Schoof and Yager, 2007) to the total concentration.

[b] Indicates that all concentrations were below the MDL. The concentration presented is the MDL.

Although it is recognized that recreational anglers are likely to spend the majority of their time angling within a few isolated locations, the derivation of EPCs were based on data for fish collected from all lakes included in the sampling program to be representative of an overall community-based estimate (Table 4-10). Since the local food survey indicated that the most commonly consumed fish is walleye, the 95% UCLM concentrations for walleye were selected

as the EPC when they exceeded the 95% UCLM for all fish species (note: this occurred for mercury only). Refer to Appendix E for the complete set of data for fish tissue concentrations.

Table 4-10 Exposure Point Concentrations for COC in Fish (µg/g ww)

Parameter	*Arsenic*[a]	*Cadmium*	*Copper*	*Lead*	*Total Mercury*	*Selenium*
# Samples	166	166	166	166	212	166
Mean	0.011[c]	0.014[c]	0.26	0.040[c]	0.17	1.2
Maximum	0.052	0.037	0.77	0.07	1.4	5.1
95% UCLM for all fish	0.0097	0.0084	0.31	0.031	0.25[b]	1.6
EPC	0.0097	0.0084	0.31	0.031	0.45	1.6

[a] Concentrations of arsenic in fish tissues were adjusted to represent the inorganic fraction by applying a factor of 0.068 (representative of freshwater finfish (Schoof and Yager, 2007)) to the total concentration.
[b] Concentration represents the 97.5% UCLM as recommended by ProUCL.
[C] Value represents mean of detected values only. The 95% UCLM was derived considering values below detection.

Although for the majority of samples, the analysis of mercury was for total mercury, nine samples were analyzed for both total and methyl mercury (Table 4-11). For some samples, the concentration of methyl mercury exceeded the total mercury concentration. This is assumed to be the result of standard laboratory error and variability in analysis. Since by definition it is not possible to have a higher concentration of methyl mercury than total mercury in a given sample, it was conservatively assumed that the fraction was equal to 1.0 for these samples. The 95% UCLM fraction of total mercury that was in the form of methyl mercury was calculated to be 0.96 and was applied to the 95% UCLM concentration calculated for total mercury to appropriately allocate this concentration to inorganic mercury and methyl mercury within the exposure assessment.

Table 4-11 Methyl Mercury Content in Local Fish Tissue Samples

Lake	*Sample*	*Total Mercury (µg/g ww)*	*Methyl Mercury (µg/g ww)*	*Fraction Methyl Mercury*
Phantom	PL-01-FIL	0.13	0.08	0.62
Bakers Narrows	BN-11-FIL	0.25	0.255	1.00[a]
Amisk	AL-20-FIL	0.53	0.282	0.53
Big Island	BI-32-FIL	0.24	0.153	0.64
Denare Beach	DB-47-FIL	0.56	0.642	1.00[a]
Embury	EL-54-FIL	0.32	0.279	0.87
Hamell	HL-62-FIL	0.56	0.549	0.98
Kisseynew	KL-73-FIL	0.22	0.106	0.48
Jan	JL-82-FIL	0.30	0.328	1.00[a]
Average	-	-	-	0.79
95% UCLM				**0.96**

[a] The methyl mercury concentration exceeded the total mercury concentration, therefore, the sample was assumed to be 100% methyl mercury.

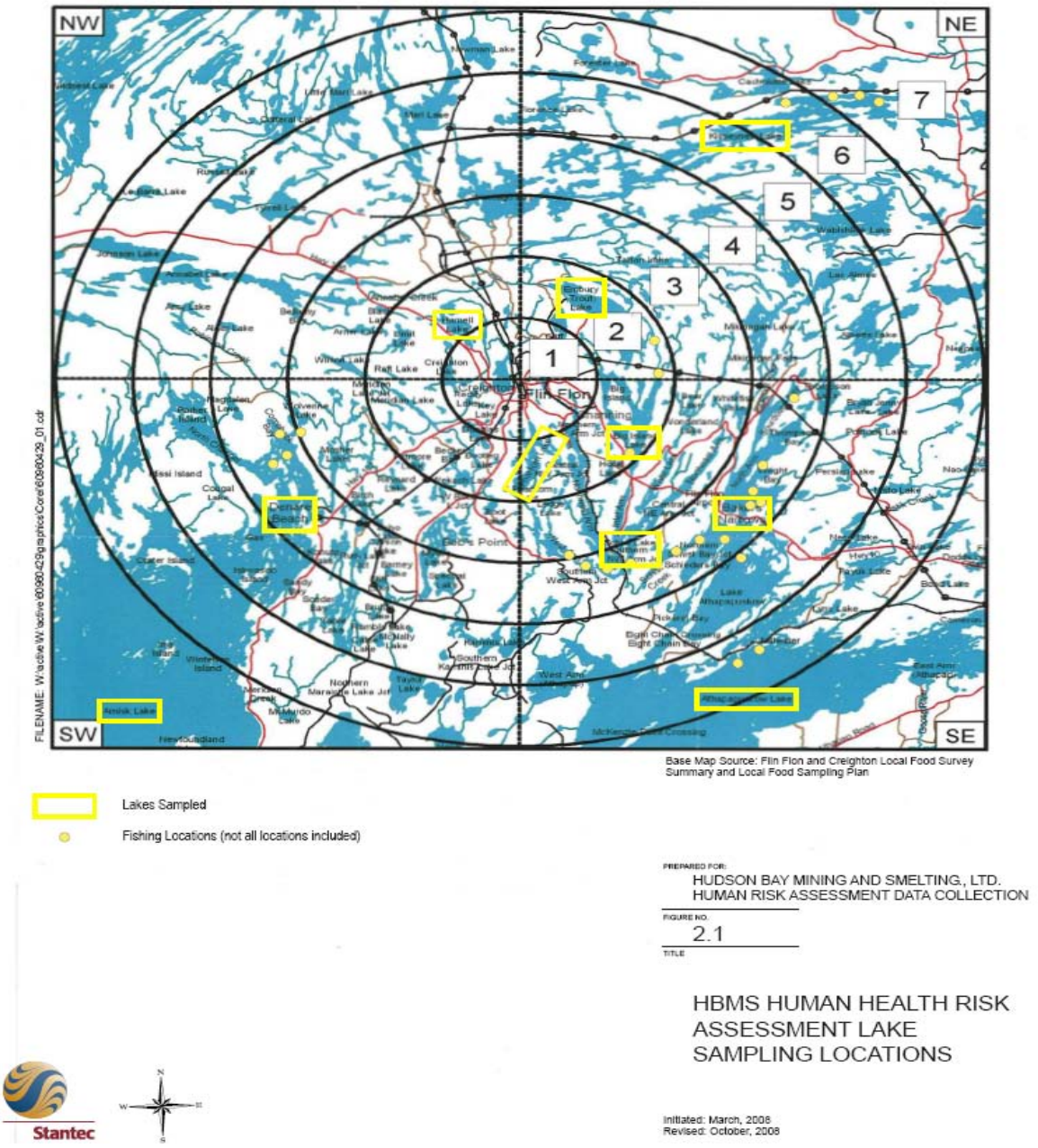

Figure 4-2 Human Health Risk Assessment Lake Sampling Locations

Lakes closer to the facility would be expected to have higher levels of all COC which are related to emissions from the facility operations. However, the results of the fish study do not demonstrate this trend for mercury. Rather, mercury levels remain consistent in fish regardless of the lake from which they were sampled. Wren and Stokes (1988) sampled fish, invertebrates and aquatic mammals in the Sudbury area and found that low mercury levels in the tissues were related to high levels of selenium. Selenium, which originated from the smelters in the vicinity, interfered with assimilation of mercury into the fish tissues by decreasing the rate of uptake from food and water (Rudd *et al*., 1980; Turner and Swick, 1983; Nriagu and Wong, 1983). Chen *et al*. (2001) suggested that selenium may have an antagonistic effect on mercury uptake. This was demonstrated using perch (*Perca flavescens*) and walleye (*Stizosedion vitreum*) that were

sampled from lakes in the Sudbury area that were at distances between 4 to 204 km from the smelters. Results showed that there were strong inverse relationships between the concentrations of selenium and mercury in the fish tissues. As the concentrations of selenium increased in the muscle tissues, the concentrations of mercury decreased exponentially. As well, selenium concentrations decreased with increasing distance from the smelter. Further studies by Belzile *et al*. (2005) examined the bioassimilation of mercury by other aquatic biota including zooplankton, mayflies, amphipods, and young-of-the-year perch. Results concurred with those by Chen *et al*. (2001), showing mercury bioassimulation decreased with increasing proximity to the smelter, and increasing selenium concentrations in the water. However, clearer correlation trends were reported with methylmercury and selenium in the tissues. A potential mechanism for this interaction was proposed to be the existence of a preferential affinity for selenium binding over mercury binding, therefore increasing the selenium concentrations in the muscle tissue. In addition to the antagonistic effect that selenium may be having on mercury, it has also been suggested that the high levels of selenium may be inhibiting sulfate reducing bacteria which ultimately affects methylation of mercury in the sediments which in turn affect overall assimilation of mercury. Belzile *et al*. (2005) concurred with the same opinion from previous studies that selenium may simply reduce the methylation rate of mercury and the bioaccumulation of methylmercury in the sediment species by decreasing the solubility of mercury (Jackson, 1991; Jin *et al*., 1997; 1999; Nuutinen and Kukkonen, 1998). It is hypothesized that a similar interaction between selenium and mercury is occurring in the Flin Flon area as was observed in the Sudbury area.

4.1.1.7 Indoor Dust Concentrations

While spending time indoors, receptors are assumed to be exposed to COC in indoor dust *via* incidental ingestion and dermal contact. The residential indoor dust sampling program provided measured concentrations of COC in indoor dust at 15 locations in West Flin Flon, 14 locations in East Flin Flon, 8 locations in Creighton, and 1 location in Channing (Table 4-12).

Table 4-12 Concentrations of COC Measured in Indoor Dust (µg/g)

Location	*Arsenic*	*Cadmium*	*Copper*	*Lead*	*Mercury*	*Selenium*
West Flin Flon						
# Samples	15	15	15	15	15	15
Mean	56	22	2,137	225	4.6	9.2
Maximum	91	46	3,250	398	12	19
95% UCLM	64	28	2,418	265	6.3	12
East Flin Flon						
# Samples	14	14	14	14	14	14
Mean	32	14	1,345	163	1.1	3.2
Maximum	57	25	2,880	606	2.6	5.2
95% UCLM	37	16	1,630	320	1.3	3.7
Creighton						
# Samples	8	8	8	8	8	8
Mean	55	11	1,016	166	1.4	3.9
Maximum	138	18	1,640	422	4.2	8.0
95% UCLM	81	14	1,245	264	2.3	5.5
Channing						
# Samples	1	1	1	1	1	1
Mean	-	-	-	-	-	-
Maximum	42	14	1,700	139	0.99	5.0
95% UCLM	-	-	-	-	-	-
All Samples						
# Samples	38	38	38	38	38	38
Mean	47	17	1,598	188	2.5	5.8

Table 4-12 Concentrations of COC Measured in Indoor Dust (µg/g)						
Location	***Arsenic***	***Cadmium***	***Copper***	***Lead***	***Mercury***	***Selenium***
Maximum	138	46	3,250	606	12	19
95% UCLM	53	19	1805	218	3.5	6.9

The results of this sampling program were used to assess exposure resulting from incidental ingestion and dermal contact with indoor dust. Regression equations were derived to express the relationship between co-located indoor dust concentrations and outdoor soil concentrations. Since the outdoor soil sampling program provided results for a greater number of locations than the indoor dust sampling program, these regression equations can be used to predict indoor dust concentrations for any location with measured outdoor soil concentrations. They can also be used to predict the indoor dust concentrations that are expected to be found in homes at the EPC soil concentration used in the HHRA. Using the EPC soil concentrations and the soil-to-dust regression equations, the EPC indoor dust concentrations were predicted for arsenic, cadmium, copper, mercury, and selenium (Table 4-13).

Since there was no statistically significant relationship observed between concentrations of lead in outdoor soil and indoor dust, a site-specific regression equation was not used to predict concentrations of lead in indoor dust. The U.S. EPA's Integrated Exposure, Uptake, and Biokinetics (IEUBK) model provides a Multiple Source Analysis (MSA) module that can be used to predict concentrations of lead in indoor dust based on outdoor soil and outdoor air lead concentrations. The accuracy of using this module to predict indoor dust concentrations was tested by comparing the predicted results to the measured indoor dust concentrations. This involves assigning a value to represent the mass fraction (M_{SD}) of house dust that is derived from outdoor soil. The IEUBK default value of 0.70 g soil/g dust was used for the current assessment. In addition to the contribution of outdoor soil to indoor dust lead levels, the contribution of impacted outdoor air is also considered in the MSA. Again, using the IEUBK default values, an additive increment of 100 µg/g of lead in indoor dust for every 1 µg/m^3 of lead in outdoor air was added to the contribution from outdoor soil. For example, for a given scenario in which the concentration of lead in outdoor soil is 200 µg/g and the concentration in outdoor air is 0.1 µg/m^3, the predicted indoor dust concentration would be 150 µg/g ((200 µg/g x 0.7)+(100 µg/g x 0.1)). Use of the MSA module is preferred over the use of the measured 95% UCLM indoor dust concentration because when deriving PTCs, it allows the model to adjust the indoor dust concentration as the outdoor soil concentration is increased or decreased.

Table 4-13 Predicted Concentrations of COC in Indoor Dust Based on EPC Outdoor Soil Concentrations and Site-Specific Regression Equations and the IEUBK MSA Module for Lead (µg/g)						
Location	***Arsenic***	***Cadmium***	***Copper***	***Lead***[a]	***Mercury***	***Selenium***
West Flin Flon						
Soil EPC	77	28	2,800	370	130	39
Predicted Dust EPC	68	22	2,300	290	5.8	10
East Flin Flon						
Soil EPC	17	16	870	160	7.2	4.6
Predicted Dust EPC	42	17	1,600	120	1.7	4.4
Creighton						
Soil EPC	88	16	850	250	8.9	7.2
Predicted Dust EPC	71	17	1,600	180	1.8	4.9

Table 4-13 Predicted Concentrations of COC in Indoor Dust Based on EPC Outdoor Soil Concentrations and Site-Specific Regression Equations and the IEUBK MSA Module for Lead (µg/g)

Location	*Arsenic*	*Cadmium*	*Copper*	*Lead*[a]	*Mercury*	*Selenium*
Channing						
Soil EPC	22	14	510	160	4.1	2.6
Predicted Dust EPC	46	17	1,400	120	1.6	4.1

[a] The relationship between co-located outdoor soil and indoor dust concentrations of lead was not statistically significant, therefore, a site-specific regression equation was not used to predicted indoor dust concentrations at the EPC outdoor soil concentration. Instead, the dust EPC was predicted using the MSA approach.

To test the validity of using these regression equations to predict EPC indoor dust concentrations, the measured 95% UCLM indoor dust concentrations for each community were compared to the EPCs derived using the regression equations. It should be noted that although the indoor dust lead concentrations predicted using the MSA module and the EPCs for outdoor soil and outdoor air are similar to the measured EPC for indoor dust in West Flin Flon, the MSA module underpredicted concentrations in the other COI. For example, for the community of West Flin Flon, the MSA approach predicted an indoor dust concentration for lead of 290 µg/g based on an outdoor soil concentration of 370 µg/g and an outdoor air concentration of 0.3 µg/m^3. The EPC for indoor dust in West Flin Flon based on measured data was 260 µg/g. However, the MSA-predicted lead concentrations for East Flin Flon (120 µg/g) and Creighton (180 µg/g) were notably lower than the 95% UCLM measured concentrations (320 and 260 ug/g, respectively) (Table 4-14).

Table 4-14 Indoor Dust EPCs Derived Based on Measured Dust Concentrations Versus Predicted Indoor Dust EPCs Derived Based on EPC Outdoor Soil Concentrations and Site-Specific Regression Equations and the IEUBK MSA Module for Lead (µg/g)

Location	*Arsenic*	*Cadmium*	*Copper*	*Lead*	*Mercury*	*Selenium*
West Flin Flon						
Measured Dust EPC	64	28	2,400	260	6.3	12
Predicted Dust EPC	68	22	2,300	290	5.8	10
East Flin Flon						
Measured Dust EPC	37	16	1,600	320	1.3	3.7
Predicted Dust EPC	42	17	1,600	120	1.7	4.4
Creighton						
Measured Dust EPC	81	14	1,200	260	2.3	5.5
Predicted Dust EPC	71	17	1,600	180	1.8	4.9
Channing[a]						
Measured Dust EPC	-	-	-	-	-	-
Predicted Dust EPC	46	17	1,400	120	1.6	4.1

[a] Since only 1 indoor dust sample was available for Channing, an EPC could not be derived based on measured dust data.

Based on the comparison provided in Table 4-14, the measured and predicted indoor dust EPCs are generally similar for arsenic, cadmium, copper, mercury, and selenium. Since a component of the HHRA is to provide back-calculated health-based PTCs for each COC, the regression equations were used to predict EPC indoor dust concentrations within the exposure model to allow for co-ordination between these two variables. Since the IEUBK MSA module tended to underpredict indoor dust lead concentrations for some COI, the 95% UCLM measured values were used to predict exposure to lead in indoor dust (Table 4-15). Given that only one indoor dust sample was collected from Channing, the 95% UCLM for East Flin Flon was used as the EPC for Channing. This was considered to be appropriate given that these communities each had an outdoor soil EPC of 160 µg/g and an assumed outdoor air EPC of 0.10 µg/m^3. Since the MSA module accurately predicted concentrations of lead in indoor dust for the community of West Flin Flon, this relationship was considered in the derivation of the lead PTC.

Table 4-15 Exposure Point Concentrations for COC in Indoor Dust (µg/g)

Location	*Arsenic*	*Cadmium*	*Copper*	*Lead*	*Mercury*	*Selenium*
West Flin Flon						
Indoor Dust EPC	68	22	2,300	260	5.8	10
East Flin Flon						
Indoor Dust EPC	42	17	1,600	320	1.7	4.4
Creighton						
Indoor Dust EPC	71	17	1,600	260	1.8	4.9
Channing						
Indoor Dust EPC	46	17	1,400	320	1.6	4.1

4.1.1.8 Wild Game Tissue Concentrations

The Problem Formulation identified that it is reasonable to expect that individuals within communities of the Flin Flon area consume game tissue obtained from the wild land areas through activities such as hunting and trapping. Game meat concentrations have not been measured to-date in the study area; therefore, predicted tissue concentrations were used to quantify potential exposures from this pathway. Two large mammals (*i.e.*, moose and deer) and two upland birds (*i.e.*, grouse and mallard) were selected as appropriate wildlife receptors for consumption by humans because residents have indicated that the consumption of these animals is popular or frequent.

Predicted wild game tissue concentrations were used in the HHRA in combination with consumption rates provided by the Local Food Consumption Survey to determine exposures to COC from the consumption of local wild game. As a conservative measure, predicted wild game concentrations from within 15 km of Flin Flon were used since metal concentrations from within this area were higher than those predicted for wild game living at greater distances (Table 4-16). Appendix O outlines the methods used to estimate wild game tissue concentrations.

Table 4-16 Exposure Point Concentrations for COC in Wild Game (µg/g ww)

COC	*Deer*	*Moose*	*Grouse*	*Mallard*
Arsenic[a]	0.000040	0.00042	0.0000080	0.000029
Cadmium	0.00019	0.0025	0.30	0.32
Copper	0.54	4.8	0.10	0.12
Lead	0.0096	0.057	0.0034	0.0029

Table 4-16 Exposure Point Concentrations for COC in Wild Game (µg/g ww)				
COC	***Deer***	***Moose***	***Grouse***	***Mallard***
Mercury	0.0019	0.015	0.0021	0.0021
Selenium	0.0014	0.053	0.29	2.5

[a] Predicted concentrations of arsenic in wild game tissues were adjusted to represent the inorganic fraction by applying a factor of 0.01 to the total concentration as recommended for both beef and chicken (Schoof *et al*., 1999).

To derive wild game EPCs it was assumed that 75% of the annual consumption of wild game was large game and 25% was wild birds. These apportionments are similar to the central tendency estimates (CTE) derived for hunting populations in the Sudbury area in which survey respondents indicated that 26% of the annual wild game meals were wild birds with the remaining 74% large game. The average of the concentrations predicted for each COC in deer and moose was used to represent the concentration in large game, and the average of the concentrations predicted in grouse and mallard was used to represent the concentration in wild birds (Table 4-17). It should be noted that the predicted concentration of selenium in the mallard is significantly greater than concentrations predicted in other wild game due to the elevated concentrations of selenium measured in local lakes. This resulted in the prediction of high concentrations of selenium in benthic invertebrates which are the primary source of food for mallards. A discussion of the uncertainties associated with the assumptions used to derive these EPCs are provided in Chapter 7.

Table 4-17 Weighted EPCs for the Consumption of Wild Game (µg/g ww)			
COC	***Weighted Large Game Concentration***	***Weighted Game Bird Concentration***	***Overall EPC***
Arsenic[a]	0.75x0.00023=0.00017	0.25x0.000019=0.0000046	0.00017
Cadmium	0.75x0.0013=0.00098	0.25x0.31=0.078	0.079
Copper	0.75x2.7=2.0	0.25x0.11=0.028	2.0
Lead	0.75x0.033=0.025	0.25x0.0032=0.00080	0.025
Mercury	0.75x0.0084=0.0063	0.25x0.0021=0.00052	0.0068
Selenium	0.75x0.027=0.020	0.25x1.4=0.35	0.37

[a] Predicted concentrations of arsenic in wild game tissues were adjusted to represent the inorganic fraction by applying a factor of 0.01 to the total concentration.

4.1.1.9 Local Blueberries

The climate and landscape of the Flin Flon area promotes an abundance of wild blueberries. As a result, it is common for residents of the Flin Flon and Creighton area to pick large amounts of blueberries to be consumed over the course of the entire year. Concentrations of COC in wild blueberries were measured in samples collected from 13 locations at varying distances and direction from the smelter complex in August 2008 (Figure 4-3). Concentrations of arsenic and selenium were below an MDL of 0.1 µg/g (ww) at all 13 sample locations, and mercury was below an MDL of 0.01 µg/g (ww) at 12 sample locations. EPCs were generated by considering all data collected. All samples that were below the MDL were conservatively assumed to be at the MDL (Table 4-18).

Table 4-18 Exposure Point Concentrations for COC in Local Blueberries (µg/g ww)						
Parameter	***Arsenic[a,b]***	***Cadmium***	***Copper***	***Lead***	***Mercury[b]***	***Selenium[b]***
# Samples	13	13	13	13	13	13
Mean	-	0.0054	0.25	0.049	-	-
Maximum	-	0.014	0.54	0.23	-	-
EPC (95% UCLM)	0.035	0.048	2.1	0.51	0.01	0.1

a Concentrations of arsenic in blueberries were adjusted to represent the inorganic fraction by applying a factor of 0.35 (representative of grapes) to the total concentration.

b All samples were below the MDL,with a single exception in which mercury was at the detection limit, therefore, mean, maximum, and 95% UCLMs could not be generated. The EPC is the MDL.

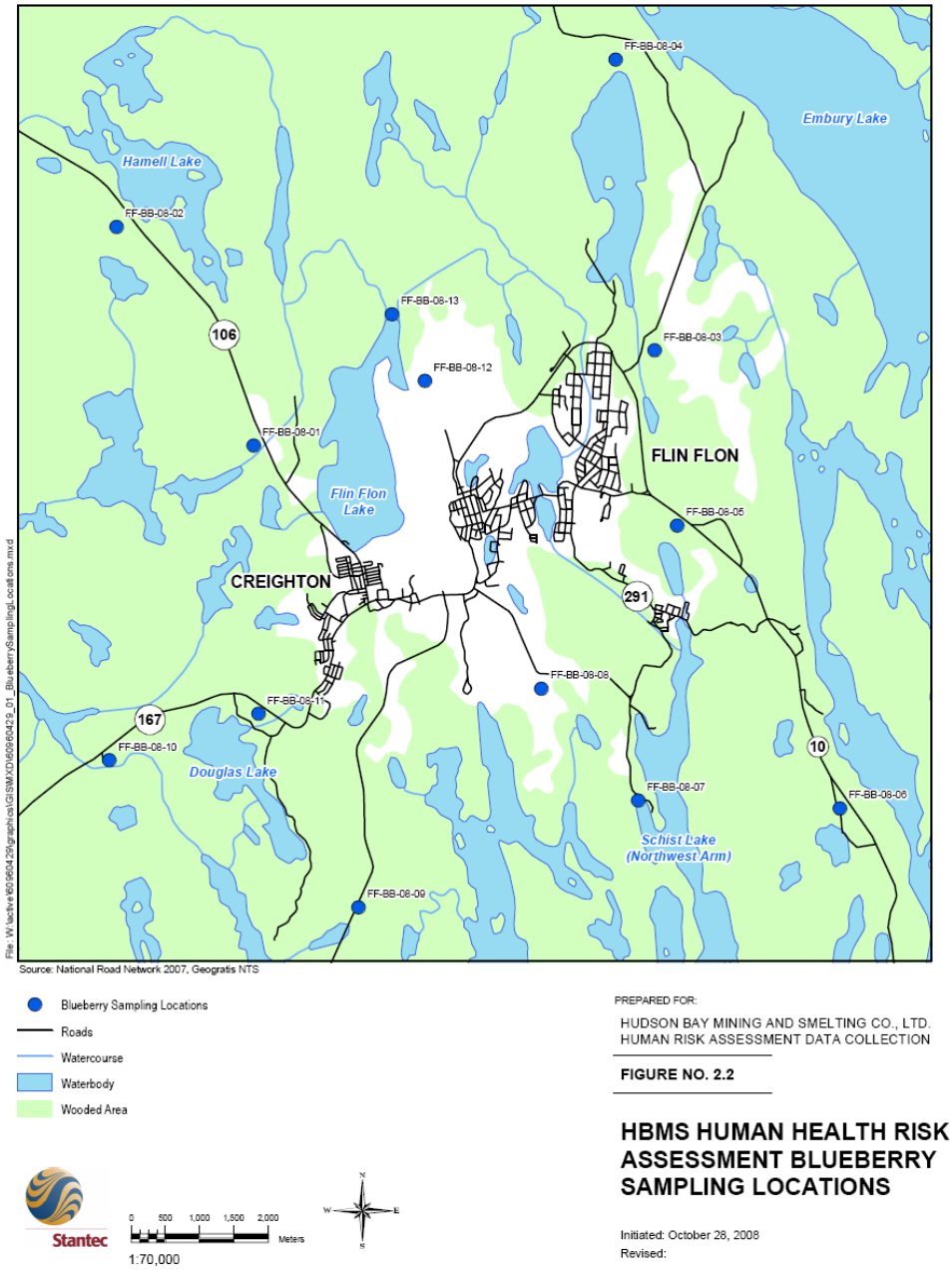

Figure 4-3 HBMS Human Health Risk Assessment Blueberry Sampling Locations

4.1.1.10 Surface Water and Sediment

The natural abundance of lakes in the Flin Flon-Creighton area provides the opportunity for many water-based recreational activities. As a result, the HHRA evaluated the potential exposure and risk levels associated with swimming in surface water of lakes throughout the study area. During the completion of the fish study conducted by Stantec (2009) in the summer of 2008, a surface water sample was collected and anaylzed from each lake. Due to the limited number of surface water samples, as a conservative measure the maximum concentration of each COC measured in all lakes sampled was selected as the EPC for the assessment of exposure while swimming (Table 4-19).

Table 4-19 Maximum Concentrations of COC in Surface Water (n=12)

COC	*Maximum Surface Water Concentration (µg/L)*
Arsenic	8.0
Cadmium	2.2
Copper	19
Lead	1.0
Mercury	0.024[a]
Selenium	5

[a] Concentration is the maximum value from the re-analysis completed by Manitoba Water Stewardship in January 2009.

Three sediment samples were also collected from lakes included in the fish study. Table 4-20 provides the mean, maximum, and 95% UCLM concentrations for COC measured in sediment. The 95% UCLM concentration of each COC was selected as the EPC for the assessment of exposure while swimming.

Table 4-20 Exposure Point Concentrations for COC Measured in Flin Flon Sediments (µg/g)

COC	*Arsenic*	*Cadmium*	*Copper*	*Lead*	*Mercury*	*Selenium*
# Samples	36	37	37	37	37	36
Mean	43	34	560	190	1.0	25
Maximum	260	300	4,800	2,500	7	220
95% UCLM	110[a]	150[b]	2,600[b]	630[a]	2.6[a]	100[b]

[a] Concentration represents the 97.5% UCLM as recommended by ProUCL.
[b] Concentration represents the 99% UCLM as recommended by ProUCL.

4.1.1.11 Snow

Jacques Whitford conducted snow column sampling at select locations on March 8 and 9, 2008 to provide a preliminary view of metal concentrations and metal deposition in snow around the City of Flin Flon (JW, 2008). A limited number of sites (n=12) from the Creighton and Flin Flon areas were selected for sampling based on the availability of non-disturbed snow profiles and relative accessibility. The sites were also selected to be downwind of prevailing northwest winds in the area and to be within the area of the Jacques Whitford residential soil sampling program. Snow column samples were collected in March to ensure total winter accumulation was obtained.

Table 4-21 provides the mean, maximum, and 95% UCLM concentrations for COC measured in snow columns in the Flin Flon and Creighton areas as collected in the Jacques Whitford sampling program. The complete snow column sampling report is provided in Appendix R.

Table 4-21 Exposure Point Concentrations for COC Measured in Flin Flon Snow (µg/L)						
COC	***Arsenic***	***Cadmium***	***Copper***	***Lead***	***Mercury***	***Selenium***
Mean	76	87	2,300	374	1	2
Maximum	147	183	4,940	732	2	3
95% UCLM	96	113	3,000	483	1	2

4.1.2 Background Exposure Assessment

The COC considered in the current HHRA are naturally present within the environment, and/or have a number of anthropogenic sources which are not associated with historic or ongoing emissions from the existing facility operations. As such, everyone is exposed to levels of these chemicals from a number of sources on a daily basis, regardless of where they live. It is, therefore, important to consider "background" exposures and risks in an HHRA to determine the extent to which COI residents are more exposed to chemicals from their environment than would normally be expected in the absence of a major source of COC, such as smelting emissions.

Background or baseline exposures are defined as exposures to chemicals that are not related to the point source or area of impact under assessment. Background sources may be either naturally occurring or anthropogenic (human-made) and contribute to levels of contaminants in food, water, air, soil, and consumer products that humans are commonly exposed to everyday. Background exposures can occur outside of the study area or at other time periods than those defined by the assessment.

The purpose of conducting a background exposure assessment is to determine the contribution of background sources to an individual's total exposure. The remaining proportion of total exposure, after accounting for background exposures, can in part be attributed to exposures originating from historical and current smelting activities in the Flin Flon area. The contributions of these two sources of exposure to the COC are considered in the derivation of PTCs.

Therefore, an additional exposure scenario was conducted to evaluate the degree of exposure of the receptors to the COC without the contribution of the HBMS facility. In this case, such an assessment provides an indication of the exposures experienced under a typical background scenario based on ambient or background concentrations in water, air, soil, dust, and food sources. Predicted typical background exposures can then be compared with the exposures attributed to facility activity to give an indication of total exposure to COC from all known sources. In addition to using background exposure to account for an individual's estimated total daily intake, background assessments can also be used as benchmarks of comparison that aid in determining the significance of the exposures from the study area relative to typical background exposures. Such relative contribution analysis can be useful in putting exposure and risk estimates into perspective, and guiding the development of risk management recommendations (*e.g.,* if study area exposures and risks are estimated to be less than or similar to typical exposures, the need for risk management measures may be reduced or become unnecessary). Evaluation of typical background exposures also assists in the interpretation and validation of predictive modelling data, which increases stakeholder confidence in the overall results of the HHRA process.

Background COC concentrations used in the current background exposure assessment were derived from monitoring programs across Canada. The background COC concentrations, described in more detail below, were used to calculate 95% UCLM values in outdoor air, soil and drinking water for a Typical Background scenario.

4.1.2.1 Data Used in the Typical Background Exposure Scenario

It is important to characterize background sources of exposure to COC when conducting a detailed HHRA. By incorporating background sources of exposure into the assessment, total estimated exposure from all sources (including background) can be compared to the toxicity reference value (TRV) (*e.g.,* a tolerable daily intake (TDI) or reference dose (RfD)), without needing to make decisions about how the TDI or RfD value should be reduced to account for exposures that were not explicitly evaluated. For example, for compounds considered to act *via* a threshold mechanism of toxicity, the entire RfD or TDI can be compared to the site-specific estimated exposure rate, rather than employing an allocation factor and leaving a portion of the TDI or RfD to account for unknown sources of exposure. Secondly, a good understanding of background exposures expected for the typical background scenario can be useful as a contextual framework within which to consider site-related exposures.

The exposure equations used for the typical background assessment are the same as those used to calculate exposure rates of individuals in the Flin Flon area. However, it should be noted that the data used to evaluate typical background exposures has been derived from a variety of generic sources, and the typical background receptor scenario, on its own, would not be appropriate for use in assessing potential health risks to the typical resident. It is simply included in the current assessment to provide a rough comparison point for the evaluation of the area-specific health risk predictions, and to place them into an overall context.

Background Outdoor Air Concentrations

The National Air Pollution Surveillance (NAPS) network monitors air pollutants at monitoring stations across Canada. The NAPS data provide a long-term archive of air pollution data at urban and rural locations in all regions of Canada. The NAPS dataset was selected to derive background outdoor air concentrations for the typical background scenario because it provides the advantage of consistency across chemicals, regions and time periods. The 2006 to 2008 data from monitoring stations in Winnipeg, Manitoba and Saskatoon, Saskatchewan provided the most robust set of data and was selected to represent background air concentrations. All six COC are among the metals analyzed in particulate samples from NAPS monitoring stations; however, only lead, cadmium and selenium are reported for the Manitoba and Saskatchewan monitoring stations (Dann, 2008 pers. comm.). Data from the Toronto, Ontario NAPS monitoring station (185 Judson Street) was utilized for arsenic and copper. No data was identified for mercury within this database, therefore, the Canadian background concentration for mercury provided by CCME (1996a) within the derivation of the human health soil quality guideline was seleted for use in the typical background scenario (Table 4-22).

Table 4-22 Typical Ambient Air Concentrations (µg/m^3)

COC	*Location*	*No. of Samples*	*Arithmetic Mean of PM_{10} samples*
Arsenic	185 Judson Street, Toronto	53	0.001
Cadmium	65 Ellen Street, Winnipeg Saskatoon	96 71	0.0045 0.0055
Copper	185 Judson Street, Toronto	53	0.009
Lead	65 Ellen Street, Winnipeg Saskatoon	96 71	0.0026 0.0022
Mercury	NA	NA	0.01
Selenium	65 Ellen Street, Winnipeg Saskatoon	96 71	0.0015 0.0018

NA Not available.

Background Soil Concentrations

Measured soil concentrations in the Flin Flon/Creighton area are influenced by both regional background concentrations as well as deposition of emissions from the HBMS complex. As a result, it is not possible to distinguish between background concentrations and background plus contamination concentrations in this area. As part of the Manitoba Conservation surface soils study (Manitoba Conservation, 2007), soil samples were collected for two reference locations; Cranberry Portage and Bakers Narrows Provincial Park. The Cranberry Portage location was determined to be less likely impacted by anthropogenic sources and as such this data was selected to be representative of regional background soil concentrations for use within the typical background scenario (Table 4-23).

Table 4-23 Regional Soil Concentrations for Use in the Typical Background Exposure Scenario (µg/g)

COC	*Regional Background Soil Concentrations*
Arsenic	2.5
Cadmium	<0.2
Copper	26
Lead	5
Mercury	0.14
Selenium	0.2

Background Drinking Water Concentrations

Background drinking water data for Manitoba or Saskatchewan was only identified for arsenic. The Saskatchewan Department of Environment and Resource Management provided concentrations of total arsenic in drinking water samples collected from over 550 locations throughout Saskatchewan. The 95% UCLM concentration from 1997 to 2002 (the most recent year of available data) was used to represent the arsenic concentration in drinking water under the typical background scenario. To estimate typical background exposure to other COC in drinking water, monitoring data from over 170 water treatment plants and well supplies across Ontario were used to calculate 95% UCLM values for each COC. Drinking water monitoring data were provided by the Ontario Ministry of the Environment (OMOE) based on information collected by the Drinking Water Surveillance Program (DWSP), a voluntary reporting system operated by the OMOE in cooperation with municipalities across the province (OMOE, 2005a). For the purpose of estimating exposure to COC concentrations in typical drinking water under a typical background scenario, samples collected from distribution systems from 1997 through 2002 were included in the derivation of EPCs. These samples were unfiltered and were analysed for total metal concentrations. Raw untreated water samples were not included in the estimate of exposure to COCs in drinking water because they are typically collected before circulation through the water supply system; and therefore, do not accurately reflect drinking water that would be consumed by a typical resident under a background residential scenario (*i.e.,* from a kitchen faucet).

Drinking water concentrations reported by the DWSP are available to the public; however, these concentrations must be interpreted with caution. In some cases, negative concentration values were reported in the DWSP data due to corrections made to the analytical results based on blanks or interferences. Concentration values of zero were also reported. For the purpose of calculating a 95% UCLM for COC concentrations in drinking water, concentration values were replaced with a value equal to half the MDL where negative or zero values occurred.

A summary of the estimated 95% UCLM COC concentrations in drinking water under a typical background exposure scenario are presented in Table 4-24.

Table 4-24 Typical Background Drinking Water Concentrations (µg/L)

COC	*No. of Samples*	*95% UCLM*
Arsenic	1,244	3.0
Cadmium	-	-
Copper	2,296	40.8
Lead	2,301	1.89
Mercury	-	0.01[a]
Selenium	2,291	1.58

- Typical background concentrations of cadmium and mercury in drinking water were not identified within the DWSP.
[a] Value represents the background concentration presented by CCME within the derivation of the mercury soil quality guideline.

Background Exposure to Mercury in Dental Amalgam

The CCME (1996a) indicates that exposure to inorganic mercury from dental amalgam may be a significant source of exposure and should be included in the calculation of the estimated daily intake. The estimated daily intake associated with dental amalgam for the child (0.52 µg/day), teen (1.52 µg/day), and adult (2.81 µg/day) were included as part of the predicted exposure to residents of the COI and the Typical Background scenario. The CCME indicates that daily exposures from dental amalgam do not need to be included for the infant or toddler. These daily intakes were converted to exposure on a per body weight basis (*i.e.*, 0.016, 0.025, and 0.040 µg/kg/day for the child, teen, and adult, respectively) using the age-specific body weights recommended by Health Canada (2006a).

4.1.3 Market Basket Estimated Daily Intakes

Food represents a critical pathway of exposure to the COC for the residents of the Flin Flon area. Foods consumed and purchased from grocery stores, supermarkets, butchers, *etc,* are considered to be background sources of exposure and contribute to an individual's total level of exposure to the COC. The exposures to COC through the consumption of store-bought foods is termed the *market basket* estimated daily intake (EDI_{MB}). As part of the HHRA, a literature review was conducted to obtain published data on the concentrations of COC in store-bought foods (*i.e.,* supermarket or market basket food items) which Flin Flon-area residents may be consuming. The details of this literature review can be found in Appendix K.

A market basket EDI (EDI_{MB}) is defined as the estimated daily intake of a chemical that is related to food commonly purchased in the supermarket and other points of purchase (*e.g.,* bakery, butchery), prepared, and consumed by urban Canadians. The purpose of the EDI_{MB} is to incorporate background exposure when characterizing an individual's exposure to COC. This is to ensure that a portion of a chemical's TDI is apportioned to background sources such that the total exposure to background levels, plus soil concentrations at the acceptable benchmark level do not exceed the TDI (CCME, 2005). In the context of the HHRA, the purpose of the EDI_{MB} is 2-fold:

1. To ensure that background sources are included in the exposure assessment of Flin Flon-area residences; and,
2. To ensure that background sources are accounted for when calculating PTCs for the COI.

The purpose of the literature review was to identify the most appropriate food data to characterize Flin Flon-area residents' background exposure to store-bought foods. In Canada, most supermarket foods are from sources distributed across North America and are generally not specific to the location of the supermarket. Thus, food purchased in Flin Flon should resemble the foods purchased in other cities in Canada. The exception is the locally derived food items such as home garden vegetables, wild blueberries, local fish, and wild game. These were assessed separately and explicitly incorporated in the exposure assessment.

The current market basket review was composed of three main tasks: i) identify the key food item categories; ii) determine the estimated daily intake rates for each food category; and, iii) determine the range of COC concentrations in each food category. The information generated from this phase of the study was incorporated into the HHRA model as the EDI_{MB} for each COC.

The food concentrations used in the derivation of the EDI_{MB} were based on the most applicable data available for food purchased in a Canadian supermarket. Food concentrations were calculated as the 95% UCLM when the number of samples available were less than 100 and the mean concentration when more than 100 samples were available.

In order to determine the most appropriate data to use in the Flin Flon HHRA, the following criteria were used:

- Food concentration data were Canadian-specific (if Canadian data were unavailable, the literature search extended to international studies, preferably American);
- Food was purchased from a supermarket or other public point-of-purchase (*e.g.*, bakery, butcher, *etc.*);
- Food was prepared and/or cooked for normal consumption;
- Data were reported with adequate summary statistics (raw data, or at a minimum, the sample number, mean concentration and range);
- The minimum detection limits were adequately low to detect the metal in most of the food items; and,
- The quality of the study design and the comprehensiveness of the data collected were considered appropriate for use in this HHRA.

The databases selected for calculating the EDI_{MB} are summarized in Table 4-25 (refer to Appendix K for the complete datasets).

Table 4-25 Summary of Databases Selected for Use in the Development of the EDIMB

COC	*Location*	*Date*	*Description*	*Reference*
As	Six Canadian cities	1985 and 1988	Canadian Total Diet Study[a]: Total As analyzed in supermarket foods	Dabeka *et al.*, 1993
Cd	Six Canadian cities	1985 to 1988	Canadian Total Diet Study[a]: Total Cd analyzed in supermarket foods	Dabeka and McKenzie, 1992; 1995; CCME, 1996c
Cu	Eight Canadian cities	1993 to 1999 and 2000	Canadian Total Diet Study[a]: Total Cu analyzed in supermarket foods	Health Canada, 2004a; Dabeka and McKenzie, 2005 pers. comm.
Hg	Two Canadian Cities	1998 to 2000	Canadian Total Diet Study[a]: Total Hg analyzed in supermarket foods	Dabeka *et al.*, 2003; Health Canada, 2007a

Table 4-25 Summary of Databases Selected for Use in the Development of the EDIMB				
COC	***Location***	***Date***	***Description***	***Reference***
Pb	Canada	2000	Canadian Total Diet Study[a]: Total Pb analyzed in supermarket foods	Dabeka and McKenzie, 2005 pers. comm.
Se	Canada (Toronto)	1992	Total Diet Study Total Se analyzed in supermarket foods	Dabeka, 1994; CCME, 2007

[a] All non-detected food concentrations were assumed by the authors to be the full detection limit.

For the purposes of applying the food concentrations to the EDI_{MB}, when available the raw data were obtained for all the datasets; non-detect data points were assigned a value of one half of the detection limit and the 95% UCLM of the food categories was calculated. The data used in the derivation of the EDI_{MB} are summarized in Appendix K. A brief summary of each COC is provided herein.

Arsenic

There were a number of Canadian market basket surveys available for arsenic (OMOE, 1987; Dabeka *et al.,* 1993; JWEL, 2004). Some of the market basket studies analyzed total arsenic (*e.g*., Dabeka *et al.,*1993; JWEL, 2004), while others analyzed both total and inorganic forms (OMOE, 1987).

The database selected for use in the Flin Flon HHRA was the Dabeka *et al*. (1993) Canadian Total Diet Study (CTDS) because it fulfilled all of the selection criteria and was found to be the most appropriate for arsenic. In this survey, food was sampled from supermarkets in six Canadian cities[1] and prepared as for normal consumption by Canadians (Dabeka *et al*., 1993). Raw data and summary statistics were available and the detection limits were appropriate, ranging from 0.3 to 1.1 ng/g wet weight. Unfortunately, arsenic was not analyzed in the Canadian TDS data for the period 1993 to 1999, and 2000 due to limited government resources (Dabeka and McKenzie, 2005 pers. comm.). Therefore, the available data are greater than 10 years old.

The more recent Port Colborne database (*i.e*., JWEL, 2004) was not selected because it had inappropriately high detection limits (*i.e*., arsenic was non-detectable in 97% of food samples; detection limit was ~50 ng/g dw [~10 ng/g ww for vegetables[2]]); resulting in highly uncertain estimates of food concentrations. For that analysis, non-detectable arsenic concentrations were assumed to be equal to half the detection limit (JWEL, 2004), an assumption that is typically conservative. This may explain why the mean concentrations for the food categories in the JWEL (2004) data are consistently higher than those in the Dabeka *et al*. (1993) study. Due to a lack of any alternatives, the Port Colborne data were used for arsenic concentrations in infant formula.

Many studies concerned with estimating the dietary intake of arsenic have traditionally been based on surveys of total arsenic in food, including both organic and inorganic forms of arsenic.

[1] The six Canadian cities where food was sampled are: Ottawa (sampled twice), Halifax, Winnipeg, Vancouver and Toronto (Dabeka *et al*., 1993). The authors report no significant differences in the arsenic levels in food items between the cities where the food was collected (Dabeka *et al*., 1993). (Surveys of market basket foods are generally considered to be nationally representative because the foods sampled tend to be nationally distributed.) For this reason, data from multiple cities can be combined to create a larger and more robust database.

[2] Calculated for illustrative purposed only, and assumes 80% moisture content for vegetables.

According to Schoof *et al*. (1999), arsenic concentrations in food were dominated by the relatively non-toxic organic forms of arsenic found in seafood. Schoof *et al.* (1999) conducted a market basket survey of inorganic arsenic in 40 different commodities which were anticipated to provide approximately 90% of the dietary intake of inorganic arsenic. Four samples of each commodity were collected and, analyzed for total and inorganic arsenic. Total arsenic was analyzed using a NaOH digestion and ICP-MS while inorganic arsenic was analyzed using a HCL digestion and hydride AAS. The results provided by Schoof *et al*. (1999) were consistent with other studies, in that total arsenic concentrations among seafood products were highest; however, inorganic arsenic concentrations observed in seafood were not elevated and ranged between less than 1 to 2 ng/g. According to Schoof *et al.* (1999), raw rice was found to have the highest inorganic arsenic content among all food commodities tested.

A more recent study by Schoof and Yager (2007) compiled data on total and speciated arsenic concentrations in fish and seafood. Data was collected from 20 studies from around the world including studies conducted for the purpose of environmental monitoring in contaminated areas. The average fraction of total arsenic that is inorganic was found to be 6.8% for freshwater finfish, 1.1% for marine fish, 1.5% for crustaceans, and 2.0% for molluscs. Using the results of a seafood consumption survey completed by the U.S. EPA (2002a), a weighted inorganic arsenic adjustment factor of 2.4% was derived for market basket fish and shellfish (Table 4-26).

Table 4-26 Derivation of an Inorganic Arsenic Adjustment Factor for Market Basket Fish and Shellfish

Seafood Category	*Fraction of Total Consumption*	*Fraction of Total Arsenic that is Inorganic*	*Contribution to Inorganic Arsenic Adjustment*
Freshwater Fish	0.19	0.068	0.013
Marine Fish	0.37	0.011	0.0041
Crustaceans	0.41	0.015	0.0062
Molluscs	0.03	0.02	0.00060
Total Inorganic Arsenic Adjustment			**0.024 (2.4%)**

Therefore, inorganic arsenic was assumed to represent 2.4% of the total arsenic measured in market basket fish and shellfish.

The arsenic concentration data used to establish estimated daily intake rates of inorganic arsenic from market basket and local foods (*e.g.,* home garden vegetables, *etc*.) were based on total arsenic measurements (*i.e.,* organic plus inorganic species). As a result, total arsenic concentrations reported for various food groups had to be corrected by the fraction of total arsenic that is present as inorganic species. The Schoof *et al*. (1999) data provided mean concentrations of total and inorganic arsenic in 40 different food commodities. From these data, the fraction of total arsenic which is inorganic could be derived for each food group. As previously indicated, at least four different food types within each commodity were analyzed for total and inorganic arsenic and, therefore, the ratios of inorganic over total arsenic content were developed for each food group within each commodity. The arithmetic mean ratio of different food groups was used to adjust the total arsenic concentration of a particular food group to an inorganic arsenic concentration. Table 4-27 provides data from Schoof *et al*. (1999) and Schoof and Yager (2007) that were used to calculate the mean fraction of inorganic arsenic in different food groups.

Table 4-27 Fraction of Inorganic Arsenic in Various Food Groups			
Food Group	***Total As (µg/g ww)***	***Inorganic As (µg/g ww)***	***Fraction Inorganic***
Fats, Oils, Sweets, Nuts			
Beet sugar	0.0122	0.0035	0.29
Cane sugar	0.0238	0.0044	0.18
Corn syrup	0.006	0.0004	0.07
Butter	0.0018	0.0011	0.61
Soybean oil	0.0018	0.0011	0.61
Salt	0.0048	0.0008	0.17
Beer	0.0027	0.0018	0.67
Peanut butter	0.0436	0.0047	0.11
MEAN VALUE	0.0121	0.00223	0.34
Milk, Yogurt, Cheese			
Milk, skim (non-fat)	0.0026	0.001	0.38
Milk, whole	0.0018	0.001	0.56
MEAN VALUE	0.0022	0.001	0.47
Meat, Poultry, Eggs			
Beef	0.0515	0.0004	0.01
Chicken	0.0864	0.0009	0.01
Pork	0.0135	0.0006	0.04
Eggs	0.0199	0.001	0.05
MEAN VALUE	0.0428	0.00073	0.03
Vegetables			
Beans	0.0021	0.0012	0.57
Carrots	0.0073	0.0039	0.53
Corn	0.0016	0.0011	0.69
Cucumber	0.0096	0.0041	0.43
Lettuce	0.0014	0.0015	1.0[a]
Onions	0.0096	0.0033	0.34
Peas	0.0043	0.0045	1.0[a]
Potatoes	0.0028	0.0008	0.29
Spinach	0.0051	0.0061	1.0[a]
Tomato	0.0099	0.0009	0.09
MEAN VALUE	0.00613	0.00219	0.59
Fruit			
Apple, raw	0.0048	0.0018	0.38
Apple, juice	0.0076	0.0028	0.37
Banana	0.0023	0.0006	0.26
Grapes	0.0102	0.0036	0.35
Grape Juice[c]	0.0141	0.0093	0.16
Orange	0.0016	0.0024	1.0[a]
Orange Juice	0.0048	0.001	0.21
Peaches	0.0034	0.0023	0.68
Watermelon[c]	0.0067	0.0021	0.31
MEAN VALUE	0.0165	0.00378	0.41
Bread			
Corn (meal)	0.0386	0.0044	0.11
Flour	0.0391	0.0109	0.28
Rice	0.303	0.0737	0.24
MEAN VALUE	0.127	0.0297	0.21
Fish			
Saltwater finfish - mean (N=253)	3.58	0.021	0.011[b]
Crustaceans - mean (N=44)	10.7	0.040	0.015[b]

Table 4-27 Fraction of Inorganic Arsenic in Various Food Groups			
Food Group	***Total As (µg/g ww)***	***Inorganic As (µg/g ww)***	***Fraction Inorganic***
Molluscs - mean (N=80)	2.48	0.050	0.020[b]
Freshwater finfish - mean (N=42)	0.304	0.018	0.068[b]
MEAN VALUE (weighted based on diet composition)	-	-	0.024

[a] The mean inorganic arsenic content was reported to be greater than the mean total arsenic content, therefore, an inorganic arsenic content of 100% was assumed.

[b] Mean concentrations are those derived by Schoof and Yager (2007) based on inorganic arsenic content from individual studies rather than mean total arsenic and mean inorganic arsenic concentrations from all studies.

[c] Values have been corrected from original values reported in Schoof *et al*. (1999) by Yost *et al*. (2004).

Cadmium

Canadian market basket data are available for cadmium (Dabeka and McKenzie, 1992; 1995). Dabeka and McKenzie (1992) analyzed the cadmium concentrations in 105 food composites that were collected in 1985 as part the Canadian Health Protection Branch's ongoing total diet program. The food samples, purchased at the retail level in the Ottawa region, were prepared for regular consumption before being combined into composites. Atomic absorption spectrophotometer was used to analyze cadmium concentrations in the food samples.

Dabeka and McKenzie (1995) analyzed the cadmium concentrations in food samples collected at the retail level from Halifax, Montreal, Toronto, Winnipeg and Vancouver. The food samples were prepared for regular consumption, homogenized into 113 composites and then subdivided into eight broader food categories. Cadmium concentrations were determined using atomic absorption spectrometry.

Within the derivation of the Canadian Soil Quality Guideline for inorganic cadmium, the CCME used the results of the Dabeka and McKenzie (1992; 1995) Total Diet Study to derive daily cadmium intake rates for the general population (CCME, 1996c). These values (*i.e.*, 0.58 µg/kg/day for the infant and toddler, 0.46 µg/kg/day for the child, 0.26 µg/kg/day for the teen, and 0.18 µg/kg/day for the adult) were used in the current assessment to represent the EDI from the consumption of market basket foods.

Copper

Canadian market basket data are available for copper (Health Canada, 2004a; JWEL, 2004; Dabeka and McKenzie, 2005 pers. comm.). There was good agreement among the results for the CTDS (Health Canada, 2004b; Dabeka and McKenzie, 2005 pers. comm.). The Port Colborne results were lower than the other databases for many food categories (*e.g.*, fish/seafood, milk and dairy, cereal and grain, root vegetables, sugars and sweets, and alcoholic beverages) and higher for others (*e.g.*, meat and poultry, other vegetables, and fats and oils) but were within the same order of magnitude as the results of the CTDS (JWEL, 2004).

The copper levels for organ meats were significantly higher than the rest of the meat and poultry samples for all three studies. For example, the mean copper concentrations for the meat category with and without the organ meats for three different studies were: 10,911 and 1,342 ng/g in the 2000 CTDS; 3,496 and 1,006 ng/g in the 1993 to 1999 CTDS; and, 21,935 and 685 ng/g in the Port Colborne study (Appendix K for further discussion on organ meats).

The databases selected for use in the Flin Flon HHRA were the consecutive years (1993 to 2000) of the CTDS (Health Canada, 2004a; Dabeka and McKenzie, 2005 pers. comm.) because

they fulfilled all of the selection criteria and were the most appropriate for copper. The datasets were combined to increase the Canadian coverage (eight cities) and the statistical robustness of the data.

Lead

There were a number of Canadian datasets available for lead, all conducted as part of the CTDS (Dabeka and McKenzie, 1995; Health Canada, 2004a; Dabeka and McKenzie, 2005; pers. comm.). The databases selected for use in the Flin Flon HHRA were Dabeka and McKenzie (2005, pers. comm.) because they fulfilled all of the selection criteria and were found to be the most appropriate for lead.

The CTDS lead results for 1993 through to 1999 (Health Canada, 2004a) could not be used because the accuracy of the data at near-detection limit measurements was poor due to the accidental contamination of the samples (Dabeka and McKenzie, pers. comm. 2005). The older Total Diet Study results were also not used because lead concentrations in environmental media and biological tissues/fluids are generally much lower today than in the 1970s and 1980s (ATSDR, 2007a). In addition, older Canadian diet studies (and presumably other studies in which lead was measured in various media) used analytical techniques that may not have been sensitive enough for the prescribed purpose.

Mercury

Canadian market basket data are available for mercury (Dabeka *et al*., 2003; Health Canada, 2007a).

The Dabeka *et al*. (2003) dataset was selected for use in the Flin Flon HHRA because it fulfilled all of the selection criteria and was found to be the most appropriate for mercury. Data from the Health Canada (2007a) Human Health Risk Assessment of Mercury in Fish and Health Benefits of Fish Consumption Report were combined with Dabeka *et al*. (2003) to increase the Canadian coverage and the statistical robustness of the fish and seafood data. Several databases were not used because the method detection limits were not adequately low to detect the metal in most food items.

Selenium

The results of the Dabeka (1994) Total Diet Study were used in the Flon Flon HHRA because it was the only complete source of Canadian data. In July of 1992, commercial foods were purchased in retail outlets in Toronto. This included the collection of 135 food composites, including infant formulae composites. These samples were prepared for typical consumption and analyzed in triplicates by cyclic and pseudocyclic INNA instrumental neutron activation analysis (CCME, 2007). Although the raw data from this study were not obtained, the CCME (2007) Scientific Supporting Document for the derivation of the Canadian Soil Quality Guideline for Selenium contained the average concentrations for each of the 135 food composites. These composites were organized into the 11 food categories used in the current assessment. The average concentration for each food category was used to represent the EPC in the HHRA.

Summary of Market Basket Food Concentrations

Table 4-28 provides a summary of the COC concentrations calculated for the market basket EDI.

Table 4-28 Exposure Point Concentrations of COC in market basket foods (µg/g ww)						
Food Category	***Arsenic[c]***	***Cadmium[b]***	***Copper***	***Lead***	***Mercury***	***Selenium***
Dairy Products	0.0032	-	0.2	0.006	0.00071	0.038
Meat, Poultry and Eggs (without organ meats)	0.00046	-	1	0.0066	0.0011	0.17
Fish and Shellfish	0.0049	-	1.3	0.0069[a]	0.29	0.31
Cereals and Grains	0.0032	-	1.4	0.012	0.00034	0.17
Root Vegetables	0.0043	-	1.1	0.0073	0.00022	0.014
Other Vegetables	0.0093	-	0.9	0.005	0.0059	0.016
Fruit and Fruit Juices	0.0019	-	0.85	0.014	0.00024	0.0083
Fats and Oils	0.0091	-	0.25	0.00038[a]	0.00019	0.012
Nuts and Seeds	0.0073	-	14	0.014[a]	0.001	0.34
Sugar and Candies	0.0077	-	1.4	0.04	0.00019	0.010
Infant Formula	0.0000072	-	0.9	0.0023	0.00023	0.012

- Not available.

[a] Maximum value was used when the calculated 95% UCLM value was greater than the maximum value in the data set.

[b] Insufficient literature-based data on cadmium content in individual market basket foods were available. Therefore, the total food EDI recommended by CCME (1996c) was used in the HHRA (*i.e.*, 0.58 µg/kg/day for the infant and toddler, 0.46 µg/kg/day for the child, 0.26 µg/kg/day for the teen, and 0.18 µg/kg/day for the adult).

[c] Concentrations of arsenic have been adjusted to represent the inorganic fraction only.

4.1.4 Summary of EPC Data used in the HHRA

Table 4-29 provides a summary of the EPC data outlined in the previous sections which were used in the current assessment.

Table 4-29 Summary of Exposure Point Concentrations used in the HHRA						
	Arsenic[a]	***Cadmium***	***Copper***	***Lead***	***Mercury[b]***	***Selenium***
Soil Concentrations µg/g						
West Flin Flon	77	28	2,800	370	130	39
East Flin Flon	17	16	870	160	7.2	4.6
Creighton	88	16	850	250	8.9	7.2
Channing	22	14	510	160	4.1	2.6
Typical Background	2.5	0.2	26	5	0.14	0.2
Dust Concentrations (calculated)[c] µg/g						
West Flin Flon	68	22	2300	260	5.8	10
East Flin Flon	42	17	1600	320	1.7	4.4
Creighton	71	17	1600	260	1.8	4.9
Channing	46	17	1400	320	1.6	4.1
Typical Background[d]	2.5	0.2	26	5	0.1	0.2
Air Concentrations (outdoor and indoor) µg/m^3						
West Flin Flon	0.084	0.07	0.84	0.34	0.016	0.052
East Flin Flon	0.040	0.026	0.22	0.10	0.000094	0.014
Creighton	0.0085	0.0046	0.058	0.034	0.0013	0.0042
Channing	0.040	0.026	0.22	0.10	0.000094	0.014
Typical Background	0.001	0.0055	0.009	0.0026	NA	0.0018
Drinking Water µg/L						
West Flin Flon	3	1.3	520	4.6	0.056	1.8
East Flin Flon	3	1.3	520	4.6	0.056	1.8

Table 4-29 Summary of Exposure Point Concentrations used in the HHRA

	Arsenic [a]	*Cadmium*	*Copper*	*Lead*	*Mercury* [b]	*Selenium*
Creighton	2.2	0.89	124	3.1	0.052	1.1
Channing	3	1.3	520	4.6	0.056	1.8
Typical Background	3.0	NA	41	1.9	0.01	1.6
Home Garden – Below Ground Vegetables µg/g wet weight						
All Communities	0.012	0.051	1.6	0.033	0.0025	0.3
Home Garden - Above Ground Vegetables µg/g wet weight						
All Communities	0.014	0.24	2.0	0.28	0.0082	0.3
Fish, Wild Game, and Blueberries µg/g wet weight						
Wild Game	0.00017	0.079	2	0.025	0.0068	0.37
Fish	0.0097	0.0084	0.31	0.031	0.45	1.6
Wild Blueberries	0.035	0.048	2.1	0.51	0.01	0.1
Surface Water µg/L	8.0	2.2	19	1.0	0.024	5.0
Sediment µg/g	110	150	2,600	630	2.6	100
Snow µg/L	96	113	3,000	483	1	2
Market Basket µg/g wet weight						
Dairy Products	0.0032	- [e]	0.2	0.006	0.00071	0.038
Meat, Poultry and Eggs (without organ meats)	0.00046	- [e]	1.0	0.0066	0.0011	0.17
Fish and Shellfish	0.0049	- [e]	1.3	0.0069	0.29	0.31
Bakery Goods and Cereals	0.0032	- [e]	1.4	0.012	0.00034	0.17
Root Vegetables	0.0043	- [e]	1.1	0.0073	0.00022	0.014
Other Vegetables	0.0093	- [e]	0.9	0.005	0.0059	0.016
Fruit and Fruit Juices	0.0019	- [e]	0.85	0.014	0.00024	0.0083
Fats and Oils	0.0091	- [e]	0.25	0.00038	0.00019	0.012
Nuts and Seeds	0.0073	- [e]	14	0.014	0.001	0.34
Sugar and Candies	0.0077	- [e]	1.4	0.04	0.00019	0.010
Infant Formula	0.0000072	- [e]	0.9	0.0023	0.00023	0.012

NA Not available.

[a] The arsenic exposure point concentration for all food products (*i.e.*, home garden, local produce, fish and wild game, and market basket foods) were adjusted to represent only the inorganic arsenic fraction content of the food (on which the TRV is based), as follows: all vegetable produce: 0.59, fruits and berries: 0.41, wild game: 0.01, market basket fish: 0.024, local fish: 0.068, infant formula: 0.56 (based upon whole milk), dairy: 0.47, meat and eggs: 0.03, cereals and grains: 0.21, sugars and sweets: 0.34; fats and oils: 0.34, and nuts and seeds: 0.34. Refer to Section 4.1.3 for further discussion of these factor adjustments, and Table 4-27 for the adjustment factors for each specific food grouping.

[b] Exposure point concentrations represent total mercury concentrations. For the purposes of exposure and risk calculations, total mercury measured in soil, dust, home garden produce, blueberries, wild game, sediment and all market basket foods other than fish was assumed to be 100% inorganic mercury; 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.

[c] Indoor dust concentrations were calculated based upon regression equations developed from paired soil and indoor dust data collected during the indoor dust survey except for lead. The EPC indoor dust concentration for lead is based on measured indoor dust data.

[d] Background indoor dust concentrations were not identified in the literature. The typical background soil concentrations were used as surrogate values.

[e] Insufficient literature-based data on cadmium content in individual market basket foods were available. Therefore, the total food EDI recommended by CCME was used in the HHRA (refer to Table 4-28).

4.1.5 Exposure Assessment of Carcinogens

As the health endpoint of concern for carcinogenic chemicals in the HHRA framework is considered to be the incremental lifetime cancer risk (ILCR) for an exposed population, the exposure period that is assessed is an assumed lifetime (*i.e.,* 80 years as recommended by Health Canada, 2006a). Under both the residential and outdoor worker exposure scenario, it was conservatively assumed that receptors would spend their entire lifetime living in the COI. Therefore, to assess the ILCR for an exposed population, the Lifetime Average Daily Dose (LADD) was calculated based on the predicted exposure for each individual age class (*i.e.*, infant, toddler, child, teen, adult) weighted according to the age class duration (*i.e.*, 0.5, 4.5, 7, 8, and 60 years, respectively). This is also often referred to as a "composite receptor".

4.1.6 Deterministic Versus Probabilistic Exposure Analysis

HHRA generally involves assigning numerical values to input parameters in an appropriate exposure or risk model to obtain a quantitative estimate of risk. Numerical values are required for parameters describing contaminant concentrations in environmental media, contaminant fate and transport, human exposure and toxic response. These values may be measured, assumed, prescribed or based on published literature. Variability and uncertainty in the input parameters or risk model result in variability and uncertainty in the estimate of risk. It is important that uncertainty in the model not be confused with variability. Uncertainty derives from a lack of knowledge. Alternatively, variability in the model describes differences in parameter values such as COC concentrations at different locations within the study area, or differences in body weight or food intake rates for individuals (*i.e.*, population heterogeneity).

Traditional deterministic methods of quantitative risk assessment use single, or "point estimate" values for input parameters and produce a single estimate of risk or hazard. While input parameters may be selected with some knowledge of their inherent variability or uncertainty, a deterministic analysis does not normally provide any information on the variability or uncertainty of the resulting risk estimate. For example, although input values are often selected to represent either average or reasonable maximum exposure conditions, the location of the point estimate of risk in the context of its potential range and distribution cannot be determined directly. A discrete, or deterministic, sensitivity analysis may provide some indication of the potential range of estimated risk values, but the variability of, and hence confidence in, the risk estimate remains unknown.

The outcome of a deterministic risk assessment model does not provide any information on its underlying distribution, nor does it indicate the likelihood that the risk estimates accurately represent upper percentiles or the central tendency (*e.g.,* the mean, mode, median) of the underlying risk distribution. Consequently, it can be difficult to identify instances where the deterministic risk estimate may be over- or understating the actual potential for risk.

In cases where risks to human health estimated using deterministic methods are clearly not negligible or obviously unacceptable, a probabilistic risk assessment (PRA) can be useful to better characterize risk. PRA uses probability distributions to characterize the inherent variability and uncertainty in input parameters, and produces a probability distribution of estimated exposure or risk. The exposure distribution can be directly compared to a toxicity benchmark to estimate the probability of exceedance. As such, a PRA accounts for natural variability and uncertainty to produce estimated probabilities of exceeding toxicity benchmarks or probabilities of effects of differing magnitude. Evaluating, calculating, and conveying the degree and magnitude of variability and uncertainty in each of the components of the risk

assessment process provides decision makers and the public with a strong scientific foundation for understanding risk and evaluating the believability of the final risk estimates. Prior to proceeding with a PRA, the risk assessor should consider whether a probabilistic analysis is necessary and/or appropriate, given the objectives of the assessment and the availability of data. A probabilistic analysis necessarily involves a greater commitment of resources to conduct the analysis and to report and present the results.

For the current HHRA, deterministic analyses were used to characterize the exposures experienced by Flin Flon and Creighton residents. If elevated risks are found under the central tendency scenario evaluated in the current HHRA, a probabilistic analysis may be considered. However, prior to proceeding with such an assessment, a detailed sensitivity analysis of the deterministic HHRA should be completed in an attempt to first identify those exposure pathways, assumptions and parameters that drive the resulting risk estimates.

4.1.7 Exposure Estimation Methods

The exposure assessment evaluates data related to all chemicals, receptors and exposure pathways identified during the problem formulation phase of the HHRA. The primary objective of the exposure assessment is to predict, using site-specific data and a series of conservative assumptions, the rate of exposure (*i.e.*, the quantity of chemical and the rate at which that quantity is received) of the selected receptors to the COC *via* the various exposure scenarios and pathways identified in the problem formulation step.

Point-estimate exposures were predicted for each of 5 receptor age classes (*i.e.*, infant, toddler, child, teen, and adult) as well as a life-time composite, for receptors in each of the four COI (*i.e.*, East Flin Flon, West Flin Flon, Creighton, and Channing) under a residential exposure scenario. For comparative purposes, a Typical Background scenario was evaluated. A commercial/industrial outdoor worker scenario was also evaluated in which exposure to adults was predicted assuming that 100% of their occupational duration was spent working outdoors. Receptors were assumed to move in a random fashion within each COI and, over time, come into contact with the EPC of the COC in a variety of environmental media. The EPC for any given environmental media (*e.g.*, air, soil, water, food, *etc*.) was defined as the 95% UCLM for that particular COI (with the exception of surface water in which case the maximum concentrations were used as the EPC). The rate of exposure to each COC from each exposure pathway was expressed as the amount of chemical taken in per body weight per unit time (*e.g.*, µg chemical/kg body weight/day) as a result of exposure to COC in the following media:

- Surface soil;
- Indoor dust;
- Outdoor/Indoor air
- Home grown produce (*i.e.,* above ground vegetables, root vegetables);
- Market basket foods (from non-local sources);
- Locally caught fish;
- Locally caught wild game;
- Locally harvested wild berries;
- Drinking water;
- Surface water; and,
- Sediment.

In addition, exposure *via* the inhalation pathway was also assessed using the EPC air concentration (µg/m^3) for the purpose of predicting risks associated with direct inhalation. The

following subsections briefly describe the calculations, assumptions and rationale used to estimate exposures through various exposure pathways. The following equations (algorithms) are standardized equations from U.S. EPA, CCME, and Health Canada and are commonly used in HHRA studies.

4.1.7.1 Outdoor Soil/Indoor Dust Exposure

Exposure to COC in outdoor soil and indoor dust is assumed to occur *via* incidental ingestion and dermal absorption through exposed skin.

Incidental Ingestion

Under the residential exposure scenario, it was assumed that for four months (122 days) of the year, winter snow cover would prevent direct exposure to outdoor soil. Therefore, during this time, 100% of the recommended daily soil/dust ingestion was assumed to be from ingestion of indoor dust. For the remaining eight months (243 days), 100% of the recommended daily soil/dust ingestion was assumed to be from ingestion of outdoor soil.

The effective intake of COC from soil/dust ingestion is dependent upon the amount of chemical released from the soil/dust during digestion. This is especially the case for metals. Only metals that are released in soluble form from soil/dust particles into the stomach or intestines during digestion are considered to be available for uptake. Metals not released from soil/dust are excreted in the feces and do not have the opportunity to cause adverse health effects. Therefore, in assessing exposure and potential human health risks from soil/dust ingestion, it is necessary to consider the amount of chemical that is actually released from the soil/dust into the gut and small intestine, and not just the total amount that is ingested within the soil/dust. Under ambient conditions in soil/dust, most metals are generally insoluble in water and tend to remain bound to soil/dust particles under neutral conditions (pH 6 to pH 8). However, the solubility of most metals increases under acidic conditions. Therefore, given the acidic conditions of the stomach, it is reasonable to expect that a portion of bound metals will be released and become bioaccessible. Results of the site-specific bioaccessibility study for arsenic and lead were used to determine the bioaccessibility of these COC from soil relative to the medium used to derive the toxicological criterion. For all other COC, the oral bioaccessibty was conservatively assumed to be 100% (Table 4-30). Refer to Appendix G for a discussion on the results of the bioaccessibility study.

Table 4-30 Relative Absorption Factors for Oral Exposure to Soil and Dust

COC	*RAF_{Soil}*	*Source*	*RAF_{dust}*	*Source*
Arsenic	0.33	Site-Specific Study	0.33	Assumed
Cadmium	1.0	Assumed	1.0	Assumed
Copper	1.0	Assumed	1.0	Assumed
Lead	0.58	Site-Specific Study	0.58	Assumed
Mercury	1.0	Assumed	1.0	Assumed
Selenium	1.0	Assumed	1.0	Assumed

Under the residential exposure scenario, the following equations were used to predict exposure *via* ingestion of outdoor soil and indoor dust:

Equation 1.0 Residential Ingestion of Outdoor Soil

$$EXP_{Ing\ Soil} = \frac{EF_S * C_{Soil} * SIR * RAF_{Soil}}{BW * DPY}$$

where:

$EXP_{Ing\ Soil}$	=	exposure *via* incidental ingestion of soil (µg/kg/day)
EFs	=	exposure frequency during summer (243 days/year)
C_{Soil}	=	exposure point concentration of COC in soil (µg/g)
SIR	=	soil/dust ingestion rate (g/day)
RAF_{Soil}	=	relative absorption factor for ingested soil (unitless)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

Equation 2.0 Residential Ingestion of Indoor Dust

$$EXP_{Ing\ Dust} = \frac{EF_W * C_{dust} * SIR * RAF_{dust}}{DPY * BW}$$

where:

$EXP_{Ing\ Dust}$	=	exposure *via* incidental ingestion of dust (µg/kg/day)
C_{dust}	=	exposure point concentration of COC in indoor dust (µg/g)
SIR	=	soil/dust ingestion rate (g/day)
EF_W	=	exposure frequency for winter months (122 days/year)
RAF_{dust}	=	relative absorption factor for ingested dust (unitless)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

Under the outdoor worker scenario, it was conservatively assumed that for each day spent at work (*i.e.*, 5 days per week, 48 weeks per year), an adult would spend 100% of their time (*i.e.*, 10 hours per day) outdoors and would be exposed to outdoor soil throughout the entire year (*i.e.*, 100% of the soil/dust ingestion rate was allocated to outdoor soil). The following equation was used to predict exposure *via* ingestion of outdoor soil:

Equation 3.0 Commercial/Industrial Ingestion of Outdoor Soil

$$EXP_{Ing\ Soil} = \frac{EF_{work} * C_{Soil} * TIR * RAF_{Soil}}{BW * DPY}$$

where:

$EXP_{Ing\ Soil}$	=	exposure *via* incidental ingestion of soil (µg/kg/day)
EF_{work}	=	exposure frequency at work (240 days/year)
C_{Soil}	=	exposure point concentration of COC in soil (µg/g)
TIR	=	total soil and dust ingestion rate (g/day)
RAF_{Soil}	=	relative absorption factor for ingested soil (unitless)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

Dermal Exposure

To estimate exposure *via* dermal contact with soil and dust under the residential exposure scenario, assumptions must be made regarding the time spent indoors and outdoors and the surface area of exposed skin throughout the year. It was assumed that for four months (122 days) of the year, winter snow cover would prevent direct exposure to outdoor soil. In addition, a limited amount of skin is assumed to be left exposed when spending time outdoors during this time thereby significantly limiting any potential dermal contact with soils. All dermal exposure during this time was assumed to be from contact with indoor dust. During time spent indoors on these colder months, it was assumed that arms and hands would be exposed and be available for dermal uptake of COC found in indoor dust. For the remaining eight months (243 days) of the year, dermal exposure was assumed to occur from direct contact with outdoor soil. During time spent outdoors on these warmer months, it was conservatively assumed that the hands, arms, and legs would be exposed and be available for dermal uptake of COC in outdoor soil.

On a daily basis, it is assumed that a single dermal exposure event occurs which is a function of the surface area of exposed skin and the soil/dust adherence to this skin. This will produce an estimate of the mass of COC adhered to the skin on a daily basis. Since route-specific TRVs are not available for dermal exposure, the dermal exposure must be compared to the oral TRV. The insoluble nature of most metals in soil/dust limits their potential for uptake through the skin. Available data on dermal uptake of metals indicate that uptake rates are low (Paustenbach, 2000). Since dermal absorption of the COC are low relative to oral absorption, the dermal exposure was adjusted by applying a relative dermal absorption factor (RAF_{Dermal}) to account for the relative difference in absorption between the oral and dermal routes. Therefore, the estimated mass value is multiplied by a chemical-specific RAF_{Dermal} to yield the soil/dust dermal exposure estimate in µg/kg body weight/day. The RAF_{Dermal} were those recommended by Health Canada (2008) and RAIS (2008) (Table 4-31).

Table 4-31 Relative Dermal Absorption Factors

COC	***RAF_{Dermal}***	***Reference***
Arsenic	0.03	Health Canada, 2008; RAIS, 2008
Cadmium	0.001	RAIS, 2008
Copper	0.001	Health Canada, 2008
Lead	0.006	Health Canada, 2008
Mercury	0.05	Health Canada, 2008
Selenium	0.002	Health Canada, 2008

Under the residential exposure scenario, the following equations were used to predict exposure *via* dermal contact with outdoor soil and indoor dust:

Equation 4.0 Residential Dermal Contact with Outdoor Soil

$$EXP_{Dermal\ Soil} = \frac{EF_S * EPD * C_{soil} * RAF_{Dermal} * \left[\left(SA_{hands} * AF_{hands}\right) + \left(SA_{AL} * AF_{other}\right)\right]}{BW * DPY}$$

where:

$EXP_{Dermal\ Soil}$	=	dermal exposure *via* direct contact with soil (µg/kg/day)
EF_S	=	exposure frequency for summer months (243 days/year)
EPD	=	exposure events per day (1 event/day)
C_{soil}	=	exposure point concentration of COC in soil (µg/g)
RAF_{Dermal}	=	chemical-specific relative dermal absorption factor (unitless)
SA_{hands}	=	surface area of hands (m^2)
AF_{hands}	=	soil adherence factor for hands (1 g/m^2/event)
SA_{AL}	=	surface area of arms and legs (m^2)
AF_{other}	=	soil adherence factor for area other than hands (0.1 g/m^2/event)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

Equation 5.0 Residential Dermal Contact with Indoor Dust

$$EXP_{Dermal\ Dust} = \frac{C_{dust} * RAF_{Dermal} * \left[\left(EF_w * EPD * \left(\left(SA_{hands} * AF_{hands}\right) + \left(SA_{Arms} * AF_{other}\right)\right)\right)\right]}{BW * DPY}$$

where:

$EXP_{Dermal\ Dust}$	=	dermal exposure *via* direct contact with indoor dust (µg/kg/day)
C_{dust}	=	exposure point concentration of COC in indoor dust (µg/g)
RAF_{Dermal}	=	chemical-specific relative dermal absorption factor (unitless)
EF_W	=	exposure frequency for winter months (122 days/year)
EPD	=	exposure events per day (1 event/day)
SA_{hands}	=	surface area of hands (m^2)
AF_{hands}	=	soil adherence factor for hands (1 g/m^2/event)
AF_{other}	=	soil adherence factor for area other than hands (0.1 g/m^2/event)
SA_{arms}	=	surface area of arms (m^2)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

Under the outdoor worker scenario, since it was assumed that for each day spent at work an adult would spend 100% of their time outdoors, dermal exposure was only to outdoor soil. The following equation was used to predict exposure *via* dermal contact of the hands and arms with outdoor soil:

Equation 6.0 Commercial/Industrial Dermal Contact with Outdoor Soil

$$EXP_{Dermal\ Soil} = \frac{EF_{work} * EPD * C_{soil} * RAF_{Dermal} * \left[\left(SA_{hands} * AF_{hands}\right) + \left(SA_{arms} * AF_{other}\right)\right]}{BW * DPY}$$

where:

$EXP_{Dermal\ Soil}$	=	dermal exposure *via* direct contact with soil (µg/kg/day)
EF_{work}	=	exposure frequency at work (240 days/year)
EPD	=	exposure events per day (1 event/day)
C_{soil}	=	exposure point concentration of COC in soil (µg/g)
RAF_{Dermal}	=	chemical-specific relative dermal absorption factor (unitless)
SA_{hands}	=	surface area of hands (m^2)
AF_{hands}	=	soil adherence factor for hands (1 g/m^2/event)
SA_{arms}	=	surface area of arms (m^2)
AF_{other}	=	soil adherence factor for area other than hands (0.1 g/m^2/event)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

4.1.7.2 Exposure through Inhalation of Outdoor and Indoor Air

Exposure to COC in outdoor and indoor air was assessed through the inhalation of the PM_{10} fraction of airborne particulate matter. Given that it was assumed that concentrations of COC in indoor air were equal to concentrations measured in outdoor air, the amount of time spent indoors and outdoors was not relevant to predict exposure *via* inhalation. The relative absorption factor for each COC *via* inhalation was assumed to be 100%.

Equation 7.0 Residential Inhalation of Fine Particulates in Air

$$EXP_{Inh} = \frac{C_{air} * EF * BR * RAF_{Inh}}{BW * DPY}$$

where:

EXP_{Inh}	=	exposure *via* inhalation (µg/kg/day)
C_{air}	=	exposure point concentration of COC in air ($\mu g/m^3$)
EF	=	exposure frequency (365 days/year)
BR	=	breathing rate (m^3/day)
RAF_{Inh}	=	relative absorption factor *via* inhalation (unitless)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

Under the outdoor worker scenario, since it was assumed that for each day spent at work an adult would spend 100% of their time outdoors, inhalation exposure was only to outdoor air. The following equation was used to predict exposure *via* inhalation of outdoor air:

Equation 8.0 **Commercial/Industrial Inhalation of Fine Particulates in Outdoor Air**

$$EXP_{Inh\ OA} = \frac{C_{IA} * TSO * EF * BR * RAF_{Inh}}{BW * DPY}$$

where:

$EXP_{Inh\ OA}$	=	exposure *via* inhalation of outdoor air (µg/kg/day)
C_{IA}	=	exposure point concentration of COC in outdoor air (µg/m^3)
EF	=	exposure frequency (240 days/year)
TSO	=	fraction of daily time spent outdoors at work (10/24 unitless)
BR	=	breathing rate (m^3/day)
RAF_{Inh}	=	relative absorption factor *via* inhalation (unitless)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

4.1.7.3 Exposure *via* Consumption of Home Garden Vegetables

Exposure to COC was assessed *via* the consumption of vegetables grown in home gardens in the study area under the residential exposure scenario. The results of the Manitoba Conservation home garden study were used to characterize concentrations of each COC in above ground and root vegetables. The consumption rates of home garden vegetables is based on the recommended daily intake rates for root and aboveground vegetables (Richardson, 1997) for each age class and the application of a generic fraction of total intake represented by home produced vegetables as recommended by U.S. EPA (1997a). The U.S. EPA (1997a) used the Nationwide Food Consumption Survey (NFCS) data to generate intake rate approximations of different home produced foods. At the time of the publication, the latest NFCS had been conducted from 1987 to 1988. The sample size of the NFCS in 1987 was approximately 4,300 households or 10,000 individuals.

The U.S. EPA (1997a) cautions that consumption rate data are based on short-term observations (*i.e.,* seven days) and therefore are not appropriate for use in long-term exposure assessments. This is particularly true for home produced vegetables and fruits since consumption rates would be highly correlated to season (*i.e.,* spring, summer, fall and winter). As a result, the U.S. EPA (1997a) attempted to derive a long-term distribution of the average daily intake rates of home produced foods from the short-term data available for major food groups. The approach attempted to account for variability in consumption rates from one season to the next. According to U.S. EPA (1997a), the seasonally adjusted distributions for a given region (*e.g.,* the north eastern region) were derived by averaging the intake rates for each of the four seasons (spring, summer, winter and fall).

The seasonally adjusted percentiles representing consumer-only (*i.e.*, excluding all individuals who did not consume that particular food item from statistical analyses) consumption rates of home produced vegetables in the Northeast region (which includes Connecticut, Maine, Massachusetts, New Hampshire, New Jersey, New York, Pennsylvania, Rhode Island and Vermont) were selected for use in the current assessment. Table 13-71 of U.S. EPA (1997a) indicates that for the northeast region, 6.2% of all aboveground vegetables and 1.8% of all root vegetables consumed are derived from home gardens. Given the short growing season available for residents of the Flin Flon area, use of the seasonally adjusted consumption rates for the most northern region described in U.S. EPA (1997a) was considered to provide the most accurate estimation of home garden consumption rates. The relative absorption factor for each COC *via* consumption of home-grown vegetables was assumed to be 100%.

Equation 9.0 Ingestion of Home-grown Root Vegetables

$$EXP_{RV} = \frac{C_{RV} * CR_{RV} * EF * Fr_{HGRV} * RAF_{Food}}{BW * DPY}$$

where:

EXP_{RV}	=	exposure from ingestion of home-grown root vegetables (µg/kg/day)
C_{RV}	=	exposure point concentration of COC in home-grown root vegetables (µg/g ww)
CR_{RV}	=	consumption rate of root vegetables (g/day)
EF	=	exposure frequency (365 days/year)
Fr_{HGRV}	=	fraction of root vegetables consumed derived from home gardens (0.018 unitless)
RAF_{Food}	=	chemical-specific relative absorption factor for food
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

Equation 10.0 Ingestion of Home-grown Above Ground Vegetables

$$EXP_{AGV} = \frac{C_{AGV} * CR_{AGV} * EF * Fr_{HGAGV} * RAF_{Food}}{BW * DPY}$$

where:

EXP_{AGV}	=	exposure from ingestion of home-grown above ground vegetables (µg/kg/day)
C_{AGV}	=	exposure point concentration of COC in home-grown above ground vegetables (µg/g ww)
CR_{AGV}	=	consumption rate of above ground vegetables (g/day)
EF	=	exposure frequency (365 days/year)
Fr_{HGAGV}	=	fraction of above ground vegetables consumed derived from home gardens (0.062 unitless)
RAF_{Food}	=	chemical-specific relative absorption factor for food
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

4.1.7.4 Exposure *via* Consumption of Local Wild Blueberries

Exposure to COC was assessed *via* the consumption of local wild blueberries under the residential exposure scenario. The results of the Stantec (2009) local blueberry sampling study were used to characterize concentrations of COC in blueberries collected in the study area. Since there is an abundance of blueberries in the area, rather than deriving a consumption rate that is based on a typical fruit or berry consumption rate and allocating a fraction of this to local berries, it was conservatively assumed that consumption of wild blueberries would be a separate consumption rate in addition to the consumption of market basket berries. The U.S. EPA (1997a; Table 13-49) lists an average consumer-only consumption rate of homegrown "other berries" of 0.48 g/kg/day and a standard error of 0.042. Using a consumption rate equal to the average plus the standard error (*i.e.*, 0.52 g/kg/day), this represents a local blueberry ingestion rate of 37 g/day for a 70.7 kg adult. It was conservatively assumed that receptors

would freeze blueberries and consume them one day per week throughout the entire year. Therefore, the average daily consumption rate over the course of the year is 0.074 g/kg/day, or 5.2 g/day for an adult. This equates to an annual consumption rate of 1.9 kg/year (or 4.2 pounds/year) for an adult. Receptors of all age categories were assumed to consume local berries at a rate of 0.074 g/kg/day (Table 4-32). The relative absorption factor for each COC *via* consumption of local blueberries was assumed to be 100%.

Table 4-32 Rates for the Consumption of Local Blueberries

Receptor	***Consumption Rate (g/kg/day)***	***Consumption (g/day)***
Infant	0.074	0.61
Toddler	0.074	1.2
Child	0.074	2.4
Teen	0.074	4.4
Adult	0.074	5.2

Equation 11.0 Ingestion of Local Wild Blueberries

$$EXP_{WB} = \frac{C_{WB} * CR_{WB} * EF * RAF_{Food}}{BW * DPY}$$

where:

EXP_{WB}	=	exposure from ingestion of local wild blue berries (µg/kg/day)
C_{WB}	=	exposure point concentration of COC in local wild blue berries (µg/g ww)
CR_{WB}	=	wild berry consumption rate (g/day)
EF	=	exposure frequency (365 days/year)
RAF_{Food}	=	chemical-specific relative absorption factor for food
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

4.1.7.5 Exposure v*ia* Consumption of Drinking Water

Exposure to COC *via* the ingestion of municipal drinking water was assessed using EPCs derived for all communities in Flin Flon (*i.e.*, East Flin Flon, West Flin Flon, and Channing) and for Creighton based on the ongoing drinking water monitoring program and the short-term multiple location sample event completed by JW. Age-specific water intake rates are those recommend by Health Canada (2006a). The following equation was used to predict exposure under both the residential and commercial/industrial exposure scenarios. The relative absorption factor for each COC *via* consumption of drinking water was assumed to be 100%.

Equation 12.0 Ingestion of Drinking Water

$$EXP_{DW} = \frac{C_{DW} * IR_{DW} * EF * RAF_{DW}}{BW * DPY}$$

where:

EXP_{DW}	=	exposure *via* consumption of drinking water (µg/kg/day)
C_{DW}	=	exposure point concentration of COC in drinking water (µg/L)
IR_{DW}	=	intake rate of drinking water (L/day)
EF	=	exposure frequency (365 days/year)
RAF_{DW}	=	relative absorption factor for drinking water (unitless)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

4.1.7.6 Exposure *via* Consumption of Locally Caught Fish

Results from the Local Food Consumption Survey indicate that 494 of the 499, or 99% of survey households eat locally caught fish. According to the results, 901 of the 927 (97%) adults, 128 of 142 (90%) teenagers, 90 of 95 (95%) children and 72 of 83 (87%) toddlers represented by the survey eat locally caught fish from Flin Flon and surrounding areas.

Of the fish-eating households represented by the food survey, almost half (45%) eat locally caught fish all year round, 27% eat local fish in the summer, while 11%, 9% and 7% eat local fish in spring, winter and fall respectively (Figure 4-4).

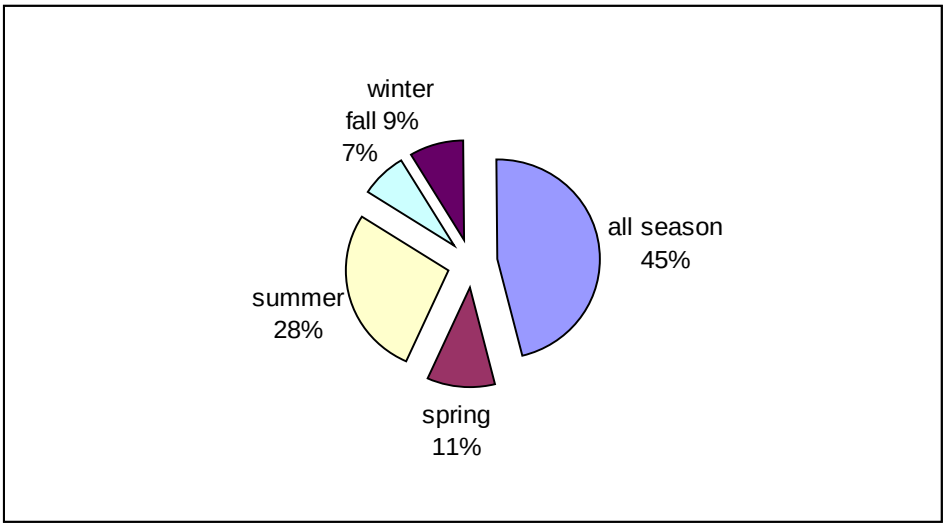

Figure 4-4 Seasonality of Local Fish Consumption by the Fish Eating Survey Respondents

The survey also asked residents to indicate the number of times per week local fish are consumed within each of the four seasons. Although this frequency varied from season to season, the most common response was 1 to 1.5 times per week. Therefore, to be conservative, for the current assessment it was assumed that adults, teens, children, and toddlers would consume local fish an average of 1.5 times per week throughout the entire year.

Since it is often difficult to accurately estimate the weight of fish consumed per meal, the survey did not request respondents to provide this information. Instead, the typical serving size of fish of 227 grams (or 8 ounces) recommended by Saskatchewan Environment (2004) was selected to represent the serving of local fish consumed by adults per meal. Combining the site-specific consumption frequency data (1.5 meals per week) with an assumed serving size of 227 grams produced an annual average daily consumption rate of 48 g/day for adults, or 0.68 g/kg/day. This rate is elevated relative to both the recommended U.S. EPA (1997a) mean and upper 95th percentile freshwater fish intake rates for recreational anglers of 8 and 25 g/day, respectively. For the general population, the U.S. EPA (1997a) provides a mean consumption rate of approximately 6.6 g/day of freshwater fish which is recommended for use in long-term exposure assessments. However, use of the average daily consumption rate of 48 g/day for adults, or 0.68 g/kg/day is considered to be a conservative and appropriate estimation for residents of Flin Flon and Creighton given the significance of the local recreational and commercial fishing activities to the community. The age-specific local fish consumption rates are provided in Table 4-33.

Table 4-33 Rates for the Consumption of Local Fish

Receptor	***Consumption Rate (g/kg/day)***	***Consumption (g/day)***
Infant	-	-
Toddler	0.68	11
Child	0.68	22
Teen	0.68	40
Adult	0.68	48

It should be noted that due to the high percentage of respondents that indicated they consumed local fish, the ingestion rates were not adjusted on a per capita basis. In addition, the consumption of local fish was considered to be a distinct dietary source in addition to the consumption of fish obtained from local supermarkets which is assessed in the market basket exposure calculations. The relative absorption factor for each COC *via* consumption of local fish was assumed to be 100%. This pathway was considered under the residential exposure scenario.

Equation 13.0 Ingestion of Local Fish

$$EXP_{LF} = \frac{CR_{LF} * C_{LF} * EF * RAF_{ORAL}}{BW * DPY}$$

where:

EXP_{LF}	=	daily exposure to COC from ingestion of local fish (µg/kg/day)
CR_{LF}	=	consumption rate of local fish (g/day)
EF	=	exposure frequency (365 days/year)
RAF_{ORAL}	=	relative absorption factor for ingestion of COC (unitless)
C_{LF}	=	exposure point concentration of COC in local fish tissue (µg/g ww)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

4.1.7.7 Exposure *via* Consumption of Local Wild Game

According to the Local Food Consumption Survey, respondents indicated that the most commonly consumed local wild game in the Flin Flon area are grouse (commonly referred to as chicken locally) (12% of respondents), deer (16%), gamebird (25%), and moose (40%).

According to the survey respondents, only 623 of the 1,247 residents represented by this survey, or approximately 50%, reported eating wild game (Figure 4-5).

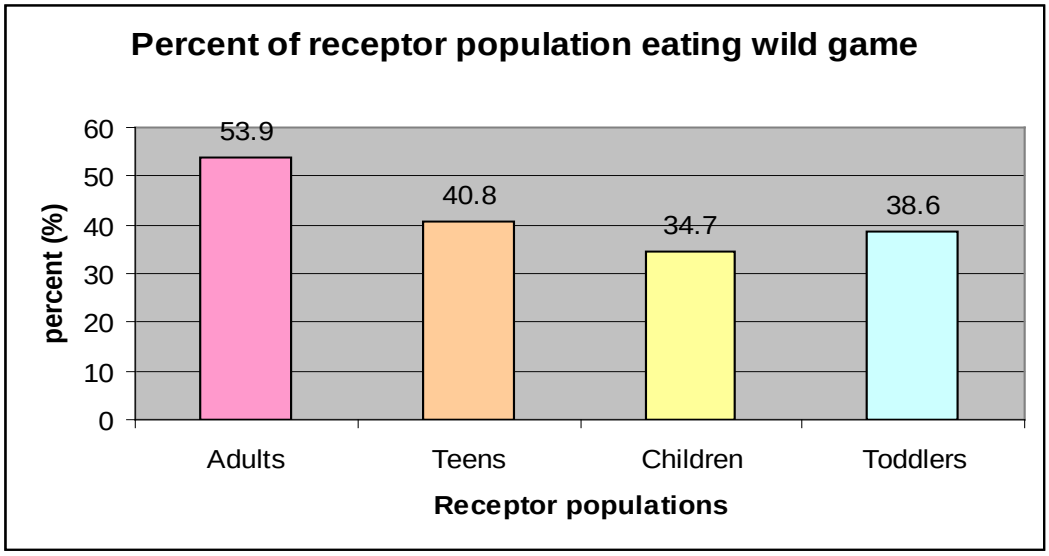

Figure 4-5 Percent of Each Receptor Population Represented by the Food Survey that Consume Wild Game

The survey also asked residents to indicate the number of times per week local game meat is consumed within each of the four seasons. Although this frequency varied from season to season, the most common response was less than once per week or 1 to 1.5 times per week. Therefore, to be conservative, for the current assessment it was assumed that adults, teens, children, and toddlers would consume local wild game meat an average of once per week throughout the entire year. Given that on average, less than 50% of the respondents indicated that they consume local wild game, and only 40% indicated that they consume wild game all year round, this is considered to be representative of an upper bound estimate of wild game consumption for the Flin Flon/Creighton community. On average, the majority of the population would have lower consumption frequencies and would have lower exposure to COC *via* this pathway.

Using the same approach used to derive rates for the consumption of local fish, site-specific consumption frequency data obtained from the local survey were combined with an assumed serving size of 227 grams (or 8 ounces) to produce an average daily consumption rate of 32 g/day for adults, or 0.46 g/kg/day (Table 4-34). This value is similar to the central tendency estimate (CTE) derived for hunting populations in the Sudbury area which was estimated to be 29.8 g/day based on a survey-based consumption frequency of 48 meals per year and an assumed serving size of 227 grams.

The fraction of total meats consumed that is local wild game was calculated as the local wild game consumption rate divided by the market basket food consumption rate for meats and eggs. Since it is unrealistic to assume that residents would be consuming local wild game meat in addition to the Health Canada recommended total meat intake represented within the market basket assessment, the consumption of market basket meats was adjusted by subtracting the fraction represented by local wild game consumption. The relative absorption factor for each COC *via* consumption of local wild game was assumed to be 100%. This pathway was considered under the residential exposure scenario.

Table 4-34 Rates for the Consumption of Local Wild Game			
Receptor	***Consumption Rate (g/kg/day)***	***Consumption (g/day)***	***Fraction of Total Meats Consumed that is Wild Game***
Infant	-	-	0
Toddler	0.46	7.6	0.10
Child	0.46	15	0.13
Teen	0.46	27	0.17
Adult	0.46	32	0.20

Equation 14.0 Ingestion of Local Wild Game

$$EXP_{LWG} = \frac{C_{LWG} * CR_{ME} * Fr_{LWG} * EF * RAF_{Food}}{BW * DPY}$$

where:

EXP_{LWG}	=	exposure from ingestion of local wild game (µg/kg/day)
C_{LWG}	=	exposure point concentration of COC in local wild game (µg/g ww)
CR_{ME}	=	consumption rate of meats and eggs (g/day)
Fr_{LWG}	=	fraction of total meats and eggs consumed that is local wild game (unitless)
EF	=	exposure frequency (365 days/year)
RAF_{Food}	=	chemical-specific relative absorption factor for food
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

4.1.7.8 Exposure while Swimming in Local Lakes

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. The method used to predict dermal absorption was that recommended by the U.S. EPA Risk Assessment Guidance for Superfund Volume I: Human Health Evaluation Manual (Part E: Supplemental Guidance for Dermal Risk Assessment) (U.S. EPA, 2004a).

Equation 15.0 Dermal Exposure While Swimming

$$EXP_{Derm\,SW} = \frac{DA_{event} * SA * EV * EF}{BW * DPY}$$

where:

$EXP_{Derm\,SW}$	=	daily dermal exposure *via* direct contact with surface water (µg/kg/day)
DA_{event}	=	absorbed dose per event (µg/cm^2-event)
SA	=	exposed surface area (cm^2)
EV	=	event frequency (event/day)
EF	=	exposure frequency (days/year)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

In equation 15.0, the DA_{event} term was calculated as follows:

$$DA_{event} = K_P * C_{SW} * CF_1 * t_{event} * CF_2$$

where:

DA_{event}	=	absorbed dose per event (µg/cm^2-event)
K_p	=	dermal permeability coefficient of COC in water (cm/hr)
C_{sw}	=	COC concentration in surface water (µg/L)
CF_1	=	conversion factor for µg/L to mg/cm^3 ($1.0x10^{-6}$)
t_{event}	=	duration of swimming event (hr event)
CF_2	=	conversion factor for mg/cm^2-event to µg/cm^2-event

The dermal permeability coefficient (Kp) for each COC was 0.001 cm/hr (U.S. EPA, 2004a).

Equation 16.0 Incidental Ingestion of Surface Water While Swimming

$$EXP_{oral\,SW} = \frac{IR_{SW} * C_{SW} * ED * EF}{BW * DPY}$$

where:

$EXP_{Oral\ SW}$	=	daily oral exposure *via* incidental ingestion of surface water (µg/kg/day)
IR_{SW}	=	incidental ingestion rate of surface water while swimming (L/hour)
C_{SW}	=	exposure point concentration in surface water (µg/L)
ED	=	event duration (hours/day)
EF	=	exposure frequency (days/year)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

To predict exposure *via* incidental ingestion of sediment while swimming, the ingestion rate for sediment was assumed to be equal to the daily soil ingestion rate. The relative absorption factors were also assumed to be equivalent to those selected for soil.

Equation 17.0 Incidental Ingestion of Sediment While Swimming

$$EXP_{oral\,Sed} = \frac{IR_{Sed} * C_{Sed} * RAF_{Sed} * EF}{BW * DPY}$$

where:

$EXP_{Oral\ Sed}$	=	daily oral exposure *via* incidental ingestion of sediment (µg/kg/day)
IR_{Sed}	=	incidental ingestion rate of sediment while swimming (µg/g)
C_{Sed}	=	exposure point concentration in sediment (µg/g)
RAF_{Sed}	=	chemical-specific relative absorption factor for sediment
EF	=	exposure frequency (days/year)
BW	=	body weight (kg)
DPY	=	days per year (365 days/year)

4.1.7.9 Market Food Basket Exposure

Market basket exposures were defined as exposures resulting from the consumption of typical supermarket foods. COC concentrations in market basket foods were considered to be representative of the typical levels observed in supermarket foods across Canada. For the current assessment, market basket exposures were classified as background exposures (*i.e.*, exposures which are independent of Flin Flon-Creighton).

Food intake rates provided by Richardson (1997; Table 5-4) were based on a 24-hour recall study collected during the 1972 to 1973 National Food Consumption Survey (NFCS). These values represent the arithmetic mean intake rates for "eaters only", meaning that those that did not report eating items within a given food group were excluded from the derivation of the mean intake rate. Use of the eater's only intake rates for all food categories can result in a significant overestimation of the total daily food intake rate because it does not account for the condition that on days when a significant amount of a few food items are consumed, there will likely be lower intake rates (or zero intake) for other food items. Therefore, the eater's only intake rates were adjusted to represent per capita intake rates by multiplying these values by the fraction of the total people surveyed that reported consuming foods in each category.

When conducting point estimate assessments, the application of successive upper confidence intervals on mean for food consumption rates (*i.e.*, the 95% UCLM) would suggest that an individual might consume every food group at an above average intake rate (observed during NFCS 24-hour recall study) for an entire year, or in the case of a carcinogenic assessment, a lifetime. As this approach is not reasonable, the arithmetic mean per capita intake rates were used to predict exposure *via* the consumption of market basket foods.

The intake rates for root vegetables, other (or above ground) vegetables, and meats and eggs were adjusted by subtracting the fraction allotted to the consumption of home garden vegetables and local wild game meat. Since the background residential scenario does not include the assessment of exposure from home garden vegetables and local wild game meat, the entire food intake for these categories (*i.e.*, home garden root vegetables, home garden above ground vegetables, and meats/eggs) were assumed to be 100% market basket foods. Although Richardson (1997) reported a single intake rate for fats, nuts and oils, the raw data from this study was obtained from Health Canada to allow for the division of these items into two separate categories (*i.e.*, Fats and oils, and Nuts and Seeds) (Table 4-35).

Table 4-35 Market Basket Food Consumption Rates (g/day)

Food Category	***Infant n=132***	***Toddler n=1,197***	***Child n=2,084***	***Teen n=2,316***	***Adult n=7,013***
Meat and Eggs					
# Eaters	38	1,074	1,949	2,151	6,696
Eater's only rate	52	86	123	170	166
Per capita rate	15	77	115	158	158
Fraction from Market Basket	1.0	0.90	0.87	0.83	0.80
Cereals and Grains					
# Eaters	123	1,192	2,080	2,297	6,928
Eater's only rate	40	168	265	282	222
Per capita rate	37	167	264	280	219
Fraction from Market Basket	1.0	1.0	1.0	1.0	1.0

Table 4-35 Market Basket Food Consumption Rates (g/day)					
Food Category	***Infant n=132***	***Toddler n=1,197***	***Child n=2,084***	***Teen n=2,316***	***Adult n=7,013***
Milk and Dairy					
# Eaters	93	1,171	2,009	2,166	6,501
Eater's only rate	664	592	613	583	286
Per capita rate	468	579	591	545	265
Fraction from Market Basket	1.0	1.0	1.0	1.0	1.0
Fish and Shellfish					
# Eaters	1	100	221	259	1,024
Eater's only rate	112	56	90	104	111
Per capita rate	0	4.7	10	12	16
Fraction from Market Basket	1.0	1.0	1.0	1.0	1.0
Fats and Oils					
# Eaters	0	1,033	1,931	2,097	6,480
Eater's only rate	0	24	40	54	48
Per capita rate	0	21	37	49	44
Fraction from Market Basket	1.0	1.0	1.0	1.0	1.0
Formula (baby)					
# Eaters	38	8	0	0	0
Eater's only rate	394	495	0	0	0
Per capita rate	113	3.3	0	0	0
Fraction from Market Basket	1.0	1.0	1.0	1.0	1.0
Fruits and Juices					
# Eaters	96	918	1,531	1,466	4,806
Eater's only rate	136	234	268	258	245
Per capita rate	99	179	197	163	168
Fraction from Market Basket	1.0	1.0	1.0	1.0	1.0
Other Vegetables					
# Eaters	41	854	1,711	1,842	5,591
Eater's only rate	72	67	98	120	137
Per capita rate	22	48	80	95	109
Fraction from Market Basket	0.94	0.94	0.94	0.94	0.94
Nuts and Seeds					
# Eaters	0	254	647	563	954
Eater's only rate	0	14	22	33	22
Per capita rate	0	3	7	8	3
Fraction from Market Basket	1.0	1.0	1.0	1.0	1.0
Root Vegetables					
# Eaters	14	901	1,711	1,889	5,725
Eater's only rate	83	105	161	227	188
Per capita rate	8.8	79	132	185	153
Fraction from Market Basket	0.98	0.98	0.98	0.98	0.98

Table 4-35 Market Basket Food Consumption Rates (g/day)					
Food Category	***Infant n=132***	***Toddler n=1,197***	***Child n=2,084***	***Teen n=2,316***	***Adult n=7,013***
Sugars and Sweets					
# Eaters	65	1,055	1,937	2,103	6,228
Eater's only rate	60	52	71	78	65
Per capita rate	30	46	66	71	58
Fraction from Market Basket	1.0	1.0	1.0	1.0	1.0

4.1.8 Development of the Risk Assessment Modelling Tool

To appropriately evaluate potential exposures to each of the COC, it is important to utilize up-to-date information and techniques for estimating exposure and risk. Exposure estimation in the current HHRA was facilitated through the use of an integrated multi-pathway environmental risk assessment spreadsheet-based (MS Excel) model. Models of this type have been used on hundreds of peer-reviewed HHRAs, including those conducted for contaminated sites, smelters, refineries, incinerators, landfills and a variety of other industrial facilities. The model incorporated the latest techniques and procedures for exposure modelling developed by various regulatory agencies (*e.g.*, Health Canada, U.S. EPA, OMOE, CCME, Cal EPA, WHO, *etc*.) and published academic and scientific literature sources.

To ensure transparency in the HHRA, and to facilitate any future regulatory and/or peer reviews of the HHRA, all assumptions, equations, and parameters used in the assessment, as well as sample calculations, were provided in the HHRA report.

In addition, for lead exposures, the IEUBK model developed by the U.S. EPA was used to predict distributions of blood lead concentrations in children and the proportion of populations that may have blood lead levels (BLLs) in excess of levels of concern. This model is typically used on U.S. Superfund sites for predicting lead exposures in children related to exposure to lead-contaminated soils. Further details regarding the IEUBK model are provided in Section 4.1.9.

4.1.9 Use of the IEUBK Model to Predict Blood Lead Levels in Children

The IEUBK computer model is a simulation model derived by the U.S. EPA to predict childhood lead exposure and retention. It has the ability to quantify the relationship between environmental lead concentrations in different media (*e.g.,* soil, water, air and food) to blood lead levels (BLLs) in children of different ages (0 to 84 months) (U.S. EPA, 1994). Estimates of a likely distribution of BLLS are centered on the geometric mean concentration and can be used to calculate the probability that BLLs in children will exceed an acceptable level. This level is typically at or below 10 µg/dL, the concentration at which health effects of concern have been identified to occur (U.S. EPA, 1994, 2002b). A general description of the IEUBK model is provided in Section 4.1.9.1. The approach in which the model was applied to predicted BLLs in children in each of the COI is described in Section 4.1.9.2.

4.1.9.1 Components of the IEUBK Model

The IEUBK model was developed to account for the unique aspects of lead exposure, bioavailability, and toxicokinetics as compared to other chemicals in the environment. The

model is comprised of four main sections or components which work together to predict BLLs from environmental media concentrations. They are:

- Exposure;
- Uptake;
- Biokinetics; and,
- Variability.

These components are described in detail below.

Exposure Component

Children may come into contact with lead in their environment in a variety of ways, depending on their daily activities and the ways in which they utilize local resources (*e.g.*, yards, playgrounds, water bodies). The path a chemical travels to reach an environmental medium (*e.g.*, air, soil, water, food, *etc.*) that a person may come into contact with is referred to as an exposure pathway. The means by which a chemical moves from the environmental medium into the body is called an exposure route. There are three major exposure routes through which chemicals can enter the body: inhalation, ingestion, and dermal absorption (*i.e.*, through the skin). The IEUBK model addresses inhalation and ingestion. The likelihood of appreciable dermal absorption of inorganic lead compounds is low, and is therefore not explicitly addressed (U.S. EPA, 1994).

The exposure component of the IEUBK model uses both receptor- and media-specific (*i.e.,* soil, dust, air, water, food) intake rates in combination with environmental media lead concentrations to estimate total daily lead intake rates of children on a µg lead/day basis. Both media concentrations and receptor intake rates are controlled by the user; therefore, "default" values and/or relationships provided by the U.S. EPA can be supplemented with site-specific data when available. The IEUBK default receptor specific media intake rates were based on a variety of studies that examined typical and/or national average intake rates for specific age classes of concern in the United States. In the absence of site-specific lead concentrations, such as indoor dust and indoor air data, the model has the ability to project indoor concentrations based on measured outdoor soil and air data. The medium of greatest concern at sites with lead-impacted soil is generally the soil itself. Children can be exposed to soil through incidental ingestion as a result of play, hand-to-mouth behaviour, or any other activity that involves oral contact with unclean objects. Furthermore, small children tend to have greater physical proximity to soil and dust.

The resulting estimated daily exposures of children in µg lead/day are used as input data for the uptake component of the model.

Uptake Component

The uptake component of the IEUBK model determines what proportion of a child's total daily lead intake will be transferred to the child's blood plasma (where it can be delivered to critical organ systems) and what portion will be eliminated from the body. Lead uptake can be defined as the amount of lead absorbed per unit time from both the gut and the lung into the systemic circulation of blood (U.S. EPA, 1994). Only a fraction of a child's total daily intake actually enters the systemic blood flow; this fraction is referred to as the *absorption fraction*.

Absorption data taken from studies in humans, primates and rats suggest a non-linear relationship between lead intake and lead absorption (U.S. EPA, 1994). Sherlock and Quinn

(1986) conducted a number of diet studies of bottle-fed infants exposed to both lead in water and formula mixed with lead-impacted water. Sherlock and Quinn were able to quantify a relationship between lead intake and lead absorption. The dose dependency of lead absorption was described by a "curvilinear" relationship (U.S. EPA, 1994). In other words, as the lead intake rate increased the rate at which lead was absorbed into the systemic blood system began to slow down. This type of non-linear absorption kinetics is addressed by the IEUBK lead model. At higher intake rates (*i.e.,* greater than 200 µg lead/day), the relationship between lead absorption and lead intake appears to be non-linear while at doses less than 100 to 200 µg/day, the relationship appears to be linear in nature (U.S. EPA, 1994). It should be noted that other factors such as the specific lead compound and particle size could affect the rate of absorption at lower doses. For example, studies conducted by Barltrop and Meek (1975) illustrated that lead in a sulfide, chromate, naphthenate or octoate form was 40 to 67% less bioavailable relative to lead in the more soluble carbonate form. Table 4-36 illustrates the default bioavailability values used by the IEUBK model for each media of concern.

Table 4-36 Default Bioavailability Values used within the IEUBK Model

Media of Concern	*Absorption Fraction via Gut*	*Absorption Fraction via Lungs*
Soil and Dust	30%	NA
Diet	50%	NA
Water	50%	NA
Air	NA	32% bioaccessible[a] 100% bioavailable

[a] Bioaccessible refers to the amount that reaches the alveoli of the lung.
NA Not available.

There are two mechanisms by which lead absorption is characterized, saturable (active) and non-saturable (passive) absorption. "Saturation" occurs when further absorption is limited by existing body burden. Uptake rates in the IEUBK model are both media- and age-dependent, while individual absorption fractions are presented for each media of concern (*i.e*., dietary, dust, soil and drinking water). When saturation effects are not occurring, the IEUBK model will estimate total absorbed lead intake by adding all media specific absorption values, where absorption from each media is equal to the age dependent intake rate multiplied by the media specific absorption fraction (U.S. EPA, 1994). However, to more accurately reflect absorption at higher doses, the saturable mechanism of absorption is also included. Thus, the total lead absorption is given by the sum of both the active and passive mechanisms of uptake.

Biokinetic Component

The biokinetic component of the IEUBK model calculates the mass of lead in each body compartment over time as a result of physiologic and biochemical processes. This involves a network of differential equations used by the biokinetic model to estimate the mass of lead as a function of time within each body compartment. The differential equations used within the biokinetic model form the basis of the mass balance approach.

The biokinetic model begins by calculating the volumes and weights of each compartment within a child's body as a function of age. The transfer rates between these compartments and elimination mechanisms are then estimated and an initial BLL is calculated for a newborn child (including maternal contribution). Lead masses in each body compartment and hence blood lead levels are calculated for each iteration or interval of time from birth to 84 months of age (U.S. EPA, 1994). Lead masses are estimated for several different compartments within a child's body including:

- Plasma-extracellular fluid (ECF);
- Red blood cells;
- Liver;
- Kidney;
- Trabecular bone;
- Cortical bone; and,
- Other soft tissue.

These particular body compartments were selected for a variety of reasons. The liver and kidney were selected as they are considered potential target sites of toxicity, while bone has a potential to be a major area of lead accumulation (U.S. EPA, 1994). The whole blood consists of two compartments, red blood cells and the extracellular fluid (ECF). It is assumed that the nervous system would be well perfused by blood.

The IEUBK model operates under the assumption that lead is transferred between the ECF compartment and most other compartments *via* first-order kinetics and at a rate of transport that is independent of compartment lead concentrations. The only transfer mechanism that is dependent on concentration is the transfer rate between plasma and red blood cells. The IEUBK model assumes that a lead saturation level does exist for red blood cells and therefore, this transfer coefficient helps to govern the age-dependent accumulation of lead in various compartments (U.S. EPA, 1994).

As previously mentioned, the lead mass for each body compartment of a newborn child is the starting point for the biokinetic algorithm. The masses of each compartment are calculated for each time step from birth to 84 months of age; the child's blood lead concentration is then calculated as the average monthly value over the number of time intervals in one month (U.S. EPA, 1994).

Variability Component

Variability in BLLs will exist in any given population of children even if all individuals within that population are exposed to similar levels of lead. There are many reasons why different BLLs may exist among a group of similarly exposed children. These include biological and behavioural variability, differences in food consumption rates, and variability, reproducibility and analytical errors within environmental lead measurements.

The probability distribution component of the IEUBK model addresses variability of BLLs associated with a typical child or a population of children. It should be emphasized that the IEUBK model does not address variability in BLLs as a result of substantially different intake rates among children; rather, it addresses the variability observed within a group of similarly exposed individuals.

This component of the IEUBK model uses the predicted population geometric mean BLL as estimated by the previous components of the model and calculates a lognormal probability distribution of BLLs centered on this estimate. The probability distribution is created by the application of a geometric standard deviation (GSD) that is based on empirical studies of lead-exposed children. By generating a probability distribution of BLLs, the model is able to calculate the probability that a population of exposed children's BLLs will exceed a selected level of concern (*i.e.*, generally set at a less-than 5% probability of exceeding 10 µg/dL).

The GSD recommended for use with the IEUBK model is 1.6. This value is based on a series of empirical studies of several specific sites throughout North America. A number of statistical methods were used to derive and verify this GSD and U.S. EPA recommends that the default value of 1.6 be employed unless there are site-specific empirical studies available.

The predicted distribution of BLLs can also be used to "back-calculate" a soil lead level that contributes to a benchmark "exceedance" of a particular blood lead level (given other sources of exposure).

Summary of IEUBK Model

The IEUBK model has become a standard tool for performing lead risk assessments. Variants of the IEUBK model have been developed that address uncertainty associated with model variables (*e.g.*, Lee *et al.,* 1995). However, the IEUBK model provides the most scientific generally-available method for both estimating the risk of exceeding benchmark BLLs as well as estimating environmental concentrations of lead that may result in elevated BLLs. A number of studies have confirmed the reliability of the IEUBK model, assuming appropriate inputs, as a reasonable means of estimating criteria by means of comparing the model's results with empirical blood lead data (*e.g.,* Hogan *et al.,* 1998; Zaragoza and Hogan, 1998).

Advantages of use of the IEUBK model over other methods of lead risk assessment include:

- The model is highly defensible due to a high degree of documentation and the rigorous level of peer review to which the model has been subjected;
- The model addresses a wide range of exposure pathways;
- Bioavailability of lead from different media is specifically addressed;
- The model uses a "biokinetic" component that addresses lead's complex toxicokinetics (as opposed to using a target risk-based exposure limit), and;
- Use of the model is straightforward, due to availability on the Internet and a user-friendly interface.

The only disadvantage of note is that use of the model is more complex than use of a single risk-based criterion.

4.1.9.2 IEUBK Model Application for the HHRA

In addition to the risk characterization derived using the excel-based HHRA exposure assessment and Health Canada TRV for lead, BLLs in receptors up to the age of seven years were predicted using the IEUBK Model for Lead in Children (Windows 32 Bit Version Build 264). The peer-reviewed exposure parameters and risk characterization assumptions set as default values within the IEUBK model were maintained unless scientifically defensible site-specific values were available. This allowed for the prediction of BLLs that were reflective of the unique characteristics of the distribution of lead throughout the Flin Flon-Creighton area and among the potential sources of contamination, while still relying on the widely accepted approaches used within the IEUBK model.

Use of the IEUBK model as a means of predicting the fraction of children in a population that may exceed a BLL of concern assumes that all individuals are subject to similar exposure point concentrations. Individuals within this population that may be exposed to significantly higher concentrations, particularly for prolonged periods (*i.e.*, a home with elevated levels in a backyard play area) may be subject to higher blood lead concentrations. Use of exposure point concentrations based on the 95% UCLM of data sets is intended to provide a general

representation of the geometric mean BLL in children within a population and the fraction of children which may have BLLs in excess of a level of concern.

4.1.9.3 Site-Specific Parameters Used within the IEUBK Model

Blood lead levels for child receptors in each of the four COI were assessed using the community-specific EPCs for outdoor soil, drinking water, and outdoor air. The influence of the consumption of local foods was also evaluated. EPCs were defined as the 95% UCLM of a given environmental medium and community. Parameters associated with each exposure scenario are described in detail below; and, along with a comparison to parameters used within the excel-based HHRA exposure model, are summarized in Table 4-37.

As discussed, the IEUBK model is a simulation model used to specifically predict childhood lead exposure and retention. It has the ability to quantify the relationship between environmental lead concentrations in different media (*e.g.,* soil, water, air and food) to BLLs in children of different ages (0 to 84 months) (U.S. EPA, 1994). Some of the IEUBK model default input parameters differ from those used in the excel-based exposure model (due to the use of site-specific data and assumptions). As stated previously, the peer-reviewed exposure parameters and risk characterization assumptions set as default values within the IEUBK model were maintained unless scientifically defensible site-specific values were available. Values in **bold** within Table 4-37 were used in the IEUBK model to predict BLLs for the current assessment.

Table 4-37 Comparison of Exposure Parameters Used in the Excel-Based HHRA Exposure Model to IEUBK Default Values

Exposure Parameter	*Receptor Age Categories (Years)*[a]						
	0-1[b]	*1-2*	*2-3*	*3-4*	*4-5*	*5-6*	*6-7*
Inhalation Pathway							
Ventilation Rate (m^3/day)							
HHRA Model	5.7	9.3	9.3	9.3	14.5	14.5	14.5
IEUBK Model	**2**	**3**	**5**	**5**	**5**	**7**	**7**
Time Spent Outdoors (hrs/ day)							
HHRA Model	1.5	1.5	1.5	1.5	1.5	1.5	1.5
IEUBK Model	**1**	**2**	**3**	**4**	**4**	**4**	**4**
Bioavailability (%)							
HHRA Model	100						
IEUBK Model	**32**						
Percentage of Lead from Outdoor Air in Indoor Air (%)							
HHRA Model	100						
IEUBK Model	**30**						
Body Weights (kg)							
HHRA Model	12.4	16.5	16.5	16.5	32.9	32.9	32.9
IEUBK Model	**7.4**	**11.4**	**13.4**	**15.7**	**18.2**	**20.4**	**22.3**
Drinking Water Pathway							
Consumption Rate (L/day)							
HHRA Model	0.45	0.6	0.6	0.6	0.8	0.8	0.8
IEUBK Model	**0.2**	**0.5**	**0.52**	**0.53**	**0.55**	**0.58**	**0.59**
Bioavailability (%)							
HHRA Model	100						
IEUBK Model	**50**						
Soil/ Dust Ingestion Pathway							
Soil + Dust Ingestion Rate (g/day)							
HHRA Model	0.05	0.08	0.08	0.08	0.02	0.02	0.02
IEUBK Model	**0.085**	**0.135**	**0.135**	**0.135**	**0.100**	**0.090**	**0.085**
Bioavailability in Soil (%)							
HHRA Model	**29**[c]						

Table 4-37 Comparison of Exposure Parameters Used in the Excel-Based HHRA Exposure Model to IEUBK Default Values

Exposure Parameter	*Receptor Age Categories (Years)*[a]						
	0-1[b]	*1-2*	*2-3*	*3-4*	*4-5*	*5-6*	*6-7*
IEUBK Model	30						
Bioavailability in Dust (%)							
HHRA Model	**29**[c]						
IEUBK Model	30						
Soil/Dust Weighting Factor (% Soil)							
HHRA Model	100% outdoor soil during summer; 100% indoor dust during winter						
IEUBK Model	**45**						
Food Consumption Pathway							
Dietary Lead Intake (µg Pb/day)							
HHRA Model – Market Basket	5.31	7.76	9.12	10.7	7.81	8.79	9.57
HHRA Model – Local Foods	0.80	1.4	1.6	1.9	2.0	2.2	2.4
IEUBK Model Total Diet	2.26	1.96	2.13	2.04	1.95	2.05	2.22
IEUBK Model Total Diet + HHRA Model Local Foods	**3.1**	**3.4**	**3.7**	**3.9**	**4.0**	**4.2**	**4.6**
Bioavailability (%)							
HHRA Model	100						
IEUBK Model	**50**						
Dermal Absorption Pathway							
Bioavailability (%)[d]							
HHRA Model	0.006						
IEUBK Model	**Not Assessed**						

[a] Parameters used in the HHRA model for the infant (0 to <6 months), toddler (6 months to <5 years) and child (5 to 11 years) were separated into the IEUBK age categories for comparative purposes.

[b] Ventilation, drinking water consumption, soil + dust ingestion, and dietary intake rates for the 0 to 1 age category is the average of the infant rates (representing age 0 to 6 months) and the preschool child rates (representing age 6 months to 1 year).

[c] The soil/dust bioavailability used within the HHRA model (*i.e.*, 58%) represents the relative bioavailability (RBA). This value was adjusted to represent an absolute bioavailability (ABA) (*i.e.*, 29%) for use in the IEUBK model.

[d] Bioavailability values presented for the HHRA Model represent relative accessibility factors (RAFs).

The bioavailability for lead in soil used within the IEUBK model was derived from the results of the site-specific *in vitro* determined bioaccessibility (IVBA). The 95% UCLM IVBA for all samples was 69% and was converted to an absolute bioavailability of lead in soil (ABA_{soil}) following the protocol recommended by the U.S. EPA (2007) which first involved the conversion of the IVBA to the relative bioavailability of lead in soil (RBA_{soil}) using the following formula:

$$RBA_{soil} = 0.878 \times IVBA - 0.028$$

The resulting RBA_{soil} value of 58% was used to predict exposure from soil pathways within the HHRA model. Since the IEUBK model requires the use of an ABA_{soil}, an additional step was taken to convert the RBA_{soil} value to an ABA_{soil} value. This was accomplished by multiplying the RBA_{soil} by the ABA for soluble lead ($ABA_{soluble}$) (*i.e.*, 50%) to determine the ABA_{soil} (*i.e.*, 29%) as follows:

$$ABA_{soil} = ABA_{soluble} * RBA_{soil}$$

Since an IVBA was not established for indoor dust, the ABA_{soil} value of 29% was used.

In addition to those values presented in Table 4-37, the use of the IEUBK model to predict BLLs in children from each of the 4 COI included site-specific levels of lead in outdoor soil, outdoor air, and drinking water (Table 4-38). Using the IEUBK default value representing the percentage of lead from outdoor air in indoor air, the predicted indoor air concentrations for each COI were predicted based on the measured outdoor air concentrations.

The IEUBK model includes the Multiple Source Analysis (MSA) module to predict concentrations of lead in indoor dust. This involves assigning a value to represent the mass fraction (M_{SD}) of house dust that is derived from outdoor soil. The IEUBK default value for M_{SD} is 0.70 g soil/g dust. In addition to the contribution of outdoor soil to indoor dust lead levels, the contribution of impacted outdoor air is also considered in the MSA. An additive increment of 100 µg/g of lead in indoor dust for every 1 µg/m^3 of lead in outdoor air is added to the contribution from outdoor soil. For example, for a given scenario in which the concentration of lead in outdoor soil is 200 µg/g and the concentration in outdoor air is 0.1 µg/m^3, the predicted indoor dust concentration would be 150 µg/g ((200 µg/g x 0.7)+(100 µg/g x 0.1)).

As discussed previously, relative to the measured concentrations, the MSA module underpredicted indoor dust concentrations for the communities of East Flin Flon and Creighton. Therefore, the measured indoor dust concentrations were used to predict BLLs for children in each COI. However, since the MSA module accurately predicted indoor dust concentrations for West Flin Flon, it was used in the derivation of the provisional trigger concentration (PTC). Use of the MSA module was preferred over the use of the measured 95% UCLM indoor dust concentration when deriving an PTC because it allows the model to adjust the indoor dust concentration as the outdoor soil concentration is increased or decreased by the user.

Table 4-38 Environmental Media Concentrations Used Within the IEUBK Model to Predict Blood Lead Concentrations

Environmental Medium	*West Flin Flon*	*East Flin Flon*	*Creighton*	*Channing*
Outdoor Soil (µg/g)	370	160	250	160
Indoor Dust (µg/g)	260	320	260	320
Outdoor Air (µg/m^3)	0.34	0.10	0.034	0.10
Indoor Air (µg/m^3)[a]	0.10	0.030	0.010	0.030
Drinking Water (µg/L)	4.6	4.6	3.1	4.6

[a] Predicted using the IEUBK default assumption that indoor air concentrations are equal to 30% of the measured outdoor air concentration.

Inhalation Exposure Pathway

Measured EPCs of lead in outdoor air for each of the four COI were entered into the IEUBK model to predict BLLs. Since indoor air concentrations were not measured, the IEUBK default assumption was adopted in which concentrations of lead in indoor air are assumed to be 30% of those measured in outdoor air, rather than the assumption of 100% used in the HHRA itself. All other parameters associated with the inhalation pathway, including ventilation rates, fraction absorbed by the lungs, and time spent outdoors, were set to the IEUBK default values.

Drinking Water Exposure Pathway

Measured EPCs of lead in drinking water for Flin Flon and Creighton were used to predict BLLs. IEUBK default values were used to represent the daily consumption rates and the bioavailability of lead in drinking water.

Soil/ Dust Ingestion Exposure Pathway

Concentrations of lead in outdoor soil were measured as part of the residential soil sampling study. The community-specific EPCs for lead in outdoor soil were used within the IEUBK model. Concentrations of lead in indoor dust were measured as part of an indoor dust sampling program. The site-specific ABA of lead in soil was also derived as part of the HHRA and was incorporated into the IEUBK model and applied to outdoor soil and indoor dust. The soil/ dust ingestion rates and weighting factors used were the IEUBK model default values.

Food Consumption Exposure Pathway

The IEUBK default daily dietary lead intake rates (µg/day) for market basket foods were significantly lower than those predicted by the HHRA model for all age groups (Table 4-39). In addition, as a result of factors such as reduced environmental deposition of lead and the reduction/elimination of lead solder in the canning process, concentrations of lead in supermarket food items have declined significantly over the past several years. To address this reduction in potential lead exposure, the U.S. EPA has provided an input file that can be loaded into the most recent version of the IEUBK model that contains predicted daily lead exposures for children based on the consumption of dietary items. These updated values are based the 1995 to 2003 U.S. Food and Drug Administration (FDA) total dietary study (TDS) and food consumption data collected by the Centers for Disease Control. These updated dietary lead intake values are significantly lower than the previous IEUBK default values which are based on U.S. FDA food monitoring data collected in the late 1980s.

Table 4-39 A Comparison Between the HHRA Model and the Default IEUBK Daily Market Basket Intake Rates

IEUBK Age Categories (Years)	*Body Weight Adjusted Daily Market Basket Lead Intake from HHRA Model (µg/day)*	*Default IEUBK Daily Dietary Intake Rates (µg/day)*	*Updated Default IEUBK Daily Dietary Intake Rates (µg/day)*
0 to 1	5.31	5.53	2.26
1 to 2	7.76	5.78	1.96
2 to 3	9.12	6.49	2.13
3 to 4	10.7	6.24	2.04
4 to 5	7.81	6.01	1.95
5 to 6	8.79	6.34	2.05
6 to 7	9.57	7.0	2.22

Since the updated values most accurately reflect the current exposure to lead through the consumption of market basket items, and this pathway typically represents a significant source of exposure, the updated values were considered to be the most appropriate for use in the current IEUBK assessment to address exposure *via* the consumption of market basket food items.

As part of the ongoing Flin Flon soils study, concentrations of COC were measured in local food items. This included wild blueberries (Stantec, 2009), local fish (Stantec, 2009), and home garden vegetables (Jones and Henderson, 2006). In addition, concentrations of COC in wild game tissue were predicted. These concentrations were used within the HHRA model to predict daily intake rates for each receptor through the consumption of these foods. In order to include the predicted exposure from local foods in the IEUBK assessment, the total daily dietary lead intake rates from the consumption of locals foods (µg/kg/day) predicted by the HHRA model were adjusted by the IEUBK receptor-specific body weights to derive a dietary lead intake

(µg/day) to be entered into the IEUBK model as described below.

Receptor body weights used within the IEUBK model were calculated using the following equation:

$$BW(t) = \left[\frac{8.375}{1+\exp\left\{\frac{-(t-3.80)}{3.60}\right\}}\right] + \left[\frac{17.261}{1+\left\{\frac{-(t-48.76)}{20.63}\right\}}\right]$$

where:

BW	=	body weight (kg)
t	=	receptor age (months)

For the current assessment, receptor body weights were calculated for months 0 to 84 and grouped within the IEUBK age categories. The weights among the 12 monthly values within each age category were averaged to represent a single body weight for each category.

Since the IEUBK age categories are broken down into smaller age groups than those used within the HHRA model, the intake rates for the toddler and child receptors were applied to multiple IEUBK age categories. Since the first IEUBK age group spans 0 to 12 months of age, the dietary lead intake was assumed to be equal to that predicted for the infant for the first six months, and that predicted for the toddler for the remaining six months. Table 4-40 illustrates the results of this approach.

Table 4-40 Scaling Dietary Lead Intake from Local Foods for IEUBK Age Categories and Body Weights

IEUBK Age Categories (Years)		*Average Body Weight (kg)*	*Dietary Lead Intake from Local Foods Predicted by HHRA Model (µg/kg/day)*	*Body Weight Adjusted Daily Dietary Lead Intake (µg/day)*	
0 to 1	0 to 0.5	5.75	0.086 (as per Infant)	0.49x0.5	0.80
	0.5 to 1	9.10	0.12 (as per toddler)	1.1x0.5	
1 to 2		11.42	0.12 (as per toddler)	1.4	
2 to 3		13.41	0.12 (as per toddler)	1.6	
3 to 4		15.69	0.12 (as per toddler)	1.9	
4 to 5		18.16	0.11 (as per Child)	2.0	
5 to 6		20.44	0.11 (as per Child)	2.2	
6 to 7		22.26	0.11 (as per Child)	2.4	

Therefore, the total dietary intake rates used in the IEUBK model were the 2008 U.S. EPA market basket intake rates combined with the local foods intake rates predicted by the HHRA model on a µg/kg/day basis but adjusted according to the IEUBK body weights (Table 4-41). It should be noted that due to limitations associated with the 2008 IEUBK dietary input file, the fraction of meats and vegetables that are represented by local foods could not be backed-out of the market basket consumption rates for these food categories. As a result, the consumption rates of meats and vegetables, and the subsequent predicted exposure of lead through the consumption of these foods, are over predicted.

Table 4-41 Total Dietary Lead Intake Rates used in the IEUBK Model (µg/day)							
Food Category	***0 to 1***	***1 to 2***	***2 to 3***	***3 to 4***	***4 to 5***	***5 to 6***	***6 to 7***
IEUBK Market Basket	2.26	1.96	2.13	2.04	1.95	2.05	2.22
Local Foods	0.80	1.4	1.6	1.9	2.0	2.2	2.4
Total Dietary	**3.1**	**3.4**	**3.7**	**3.9**	**4.0**	**4.2**	**4.6**

4.2 Hazard Assessment

The objectives of the hazard assessment (also termed *toxicity assessment*) are to:

- Provide the reader with an understanding of the toxicological effects that have been reported to be associated with exposure to the COC by various routes;
- Identify whether each COC is considered to cause carcinogenic (non-threshold) or non-carcinogenic (threshold) effects; and,
- Identify the most appropriate and scientifically-defensible exposure limits against which exposures can be compared to provide estimates of potential health risks.

Toxicity refers to the potential for a chemical to produce any type of damage, permanent or temporary, to the structure or functioning of any part of the body. The toxicity of a chemical depends on the amount of chemical taken into the body (referred to as the “dose”) and the duration of exposure (*i.e.,* the length of time the person is exposed to the chemical). For every chemical, there is a specific dose and duration of exposure necessary to produce a toxic effect in humans (this is referred to as the “dose-response relationship” of a chemical). The toxic potency of a chemical (*i.e.,* its ability to produce any type of damage to the structure or function of any part of the body), is dependent on the inherent properties of the chemical itself (*i.e.,* its ability to cause a biochemical or physiological response at the site of action), as well as the ability of the chemical to be absorbed into the body (*i.e.,* bioavailability), and then to reach the site of action. The dose-response principle is central to the human health risk assessment methodology.

There are two main types of dose-response relationships for chemicals:

Threshold Response Effects

For some effects, it is thought that there is a dose-response threshold below which no adverse effects or types of toxicity would be expected to occur. This relationship is true for all chemicals that do not cause cancer by altering genetic material (*e.g.,* most metals). Thresholds are generally assumed for non-carcinogens because, for these types of effects, it is generally believed that homeostatic, compensating, and adaptive mechanisms must be overcome before toxicity is manifested. Exposure limits derived for threshold-response chemicals are called RfD, ADI, TDI, PDI and are generally derived by regulatory agencies such as Health Canada and the U.S. Environmental Protection Agency (U.S. EPA). These values indicate doses of chemicals that individuals can receive on a daily basis without the occurrence of adverse health effects. Exposure limits derived for threshold-response chemicals are typically expressed as mg/kg body weight/day, and are typically based on experimentally-determined “No-Observed-Adverse-Effect Levels” (NOAELs), with the application of extrapolation factors that are often referred to as "safety factors" or "uncertainty factors" (U.S. FDA, 1982; U.S. EPA, 1989; Health Canada, 1993). The magnitude of these factors is dependent on the level of confidence in the available toxicology database, and reflects differences in species, duration of exposure, sensitivity, and overall quality of available data (*i.e.,* the weight-of-evidence of the supporting data).

Non-threshold Response Effects

For these effects or types of toxicity, it is assumed that there is no dose-response threshold. This means that any exposure greater than zero is assumed to have a non-zero probability of causing some type of response or damage. This relationship is typically used for chemicals which can cause cancer by damaging genetic material. Under a "no threshold" assumption, any exposure has some potential to cause damage, so it is necessary to define an "acceptable" level of risk associated with these types of exposures. For the purposes of evaluating exposures to chemicals in the environment, the "acceptable" level of risk is usually defined as an incremental risk above background of one-in-one hundred thousand to one-in-one million for an exposed population. These numbers can be better explained as the daily dose that may cause an additional incidence of cancer (*i.e.,* one cancer that would not be expected in the absence of the exposure) in a population of one hundred thousand (or a million) people exposed every day over their entire lifetime. The acceptable level of risk is a policy rather than a scientific decision, and is set by regulatory agencies, as opposed to risk assessors. In most jurisdictions, an incremental lifetime cancer risk (ILCR) level of less than 1-in-100,000 is considered a negligible risk level. Exposure limits derived for non-threshold chemicals that are believed to be potential carcinogens are typically expressed as "increased risk per unit of dose". These potency estimates are called cancer slope factors (SF) or cancer potency factors (*e.g.,* $[\mu g/kg \text{ body weight/day}]^{-1}$). These values are derived using a mathematical model-unit risk estimation approach with the built-in assumption that the condition of "zero increased risk of cancer" would only be observed when the dose is zero.

It must be recognized that the assumption of no dose-response threshold for carcinogens is an assumption which is difficult to directly test by experimentation. Thresholds may exist, even for assumed non-threshold chemicals and effects. The "no threshold" assumption ignores a large number of factors, such as the ability of the body to repair damage to genetic material, that are known to be important responses of people to naturally-occurring genotoxic carcinogens. Exposure to small concentrations of chemicals which have the potential to cause cancer happens on a daily basis to everyone in the world, because non-threshold chemicals (along with other chemicals which do not cause cancer) are present in soils, air, food and water, either from natural sources or as a result of human activities. The human body has many ways of handling these substances once they enter the body. In many cases, the body can repair damage that may be caused by exposures to low levels of carcinogenic chemicals; therefore, adverse effects do not necessarily occur.

The development of toxicological criteria or exposure limits for any given chemical must consider factors which affect the potential toxicity of that chemical. These factors may be scenario-specific, such as variation in duration or levels of exposure. Where possible, it is important that exposure limits be derived from "realistic" exposure situations that are representative of those occurring under the conditions assessed in the HHRA. For many chemicals, the toxic endpoint is also dependent on the route of exposure, as exposure *via* different routes may impact different tissues, such as those at the site of entry. In such a case, different exposure limits may be identified or developed for the different routes of exposure. Toxic potency may be modified by species- or individual-specific factors such as the ability to resist, repair or adapt to the effects of chemical exposures. In these situations, separate exposure limits might be used to ensure protection of sensitive sub-populations.

Exposure limits for chemicals are based on scientific information, professional judgement and technical review by experienced scientists with expertise in a wide range of scientific disciplines. Exposure limits are derived based on the most sensitive endpoints, often referred to as the

critical effect, in individuals (*e.g.,* cancer, organ damage, neurological effects, reproductive effects). In many cases, large uncertainty factors (*i.e.,* 100-fold or greater) are used in establishing exposure limits for chemical causing effects that are expected to have thresholds. Thus, exceedance of the exposure limit does not necessarily mean that adverse effects will occur. Rather, this result would necessitate a more detailed evaluation of both exposure and the toxicity-based exposure limit to better understand the likelihood of adverse effects occurring. Exposure rates less than an exposure limit are usually considered unlikely to be associated with adverse health effects and are, therefore, less likely to be of concern. As the frequency or magnitude of exposures exceeding the exposure limit increase, the probability of adverse health effects in a human population is usually presumed to increase, subject to scientific judgement and critical evaluation of the exposure limit and the exposure estimate, as discussed above. However, it should not be categorically concluded that all exposures below an exposure limit will be unlikely to result in adverse health effects or that all exposures above such a limit are likely to result in adverse health effects.

4.2.1 Overview of Exposure Limits Selected for the HHRA

A detailed toxicological assessment was conducted for each COC, involving identification of mechanism of action and relevant toxic endpoints, and determination of receptor- and route-specific toxicological criteria (see Appendix A). These profiles were not intended to provide comprehensive reviews of the available toxicological and epidemiological literature on the various COC. Rather, the purpose of the toxicological profiles was to: i) summarize the most relevant toxicological and epidemiological information on the substances; ii) outline any recent information that may challenge previous findings; and iii), provide supporting rationale for the exposure limits selected for use in the human health risk assessment of the Flin Flon area. The toxicological reviews are based primarily on secondary sources, such as ATSDR toxicological profiles and other detailed regulatory agency reviews (*e.g*., Health Canada and U.S. EPA.) and are supplemented with recent scientific literature. For all profiles the primary literature was searched from the date of last major review to the present. The review of primary literature was mainly to determine if any recent information exists that may challenge previous findings.
A comprehensive review of the critical toxicological literature was conducted in order to put the predicted risks associated with COC into perspective. For those COC where toxicological criteria have been developed by a regulatory agency, the development of these values considered sensitive subgroups of the population, both through use of the most stringent scientific data, as well as application of uncertainty factors in the derivation of the criteria. This yielded final toxicological criteria that are considered protective of the individuals most sensitive to the toxicity of the chemical, whether due to differences in genetics, life stage, nutrition, or health status.

Exposure limits for the COC in the current HHRA have been identified from regulatory agencies such as Health Canada, U.S. EPA, U.S. Agency for Toxic Substances and Disease Registry (ATSDR), California Environmental Protection Agency Office of Environmental Health Hazard Assessment (Cal EPA OEHHA), the Ontario Ministry of the Environment (OMOE), U.S. Centers for Disease Control (CDC), the European Union, and the World Health Organization (WHO).

4.2.2 Selection of Toxicological Criteria for the HHRA

This section provides an overview of the regulatory exposure limits considered for use in the current assessment. The exposure limits (or toxicological criteria) employed in the current assessment were obtained from a review of toxicological criteria from various regulatory agencies including the OMOE, Health Canada, the CCME, the WHO, California Environmental

Protection Agency Office of Environmental Health Hazard Assessment, ATSDR, and Centers for Disease Control and the U.S. EPA. The toxicological criteria used in this assessment reflect the approach preferred by Health Canada which requires the use of toxicity assessments published by reputable regulatory agencies such as those mentioned above.

Review of the regulatory exposure limits (toxicological criteria) was supplemented by detailed toxicological assessments conducted for each COC, involving identification of mechanism of action and relevant toxic endpoints, and determination of receptor- and route-specific toxicological criteria. Together, this information was used to select toxicological criteria for each COC that are based on the best available science. In some instances, several regulatory agencies and/or authorities have recommended different exposure limit values for the same chemical. In this situation a rationale has been provided for the use of one regulatory criterion over another for use in this study.

The U.S. EPA derives exposure limits for both threshold and non-threshold effects when data are available. The RfD and RfC are based on the assumption that a threshold exists for certain toxic non-carcinogenic effects. In general, the RfD (or RfC) is an estimate (with uncertainty spanning perhaps an order of magnitude) of a daily exposure to the human population (including sensitive subgroups) that is likely to be without an appreciable risk of deleterious effects during a lifetime (U.S. EPA, 2005).

For a number of chemicals, exposure limits are not always available for all exposure routes of concern. In these circumstances, exposure limits may be extrapolated from other routes. For example, it is common in human health risk assessments to assess the risks posed by dermal absorption of a chemical based on the exposure limit established for oral exposure (U.S. EPA, 1989; 1992a). The systemic dose absorbed dermally is scaled to the “equivalent” oral dose by correcting for the bioavailability of the dermally-applied chemical relative to an orally-administered dose.

The relative absorption difference between the oral and dermal routes of exposure can be expressed as a relative absorption factor (RAFdermal). This factor, calculated as follows, is applied to dermal exposure estimates to adjust these exposures prior to comparison with oral exposure limits when route-to-route extrapolation is necessary.

$$RAF_{dermal} = \frac{AF_{dermal}}{AF_{oral}} \, x \, 100$$

where:

RAF*dermal*	=	Relative absorption factor for dermal exposure (%)
AF*dermal*	=	The fraction of the applied chemical absorbed through the skin
AF*oral*	=	The fraction of the ingested chemical absorbed into the bloodstream

It must be recognized however that route extrapolation is only appropriate where effects are systemic in nature, and not closely associated with the point of exposure.

Tables 4-42 and 4-43 summarize the toxicological criteria selected for use in the Flin Flon HHRA. Refer to Appendix A for detailed toxicological reviews of each COC in the HHRA.

Table 4-42 Summary of the Non-Carcinogenic Toxicological Criteria Chosen for the Human Health Risk Assessment

Chemical	*Route*	*Toxicological Criterion*[a]		*Endpoint*	*Study*	*Regulatory Agency*
		Limit	*Type of Limit*			
Arsenic - Inorganic						
Acute	Oral	5.0 µg/kg-day	MRL	Gastrointestinal effects and facial edema	Mizuta *et al*., 1956	ATSDR, 2007b
	Inhalation	0.3 µg/m^3	24 hrs	Irritation, sensitization, immunosuppression, teratogenesis, genotoxicity and carcinogenicity in exposed individuals	Nagymajtenyi *et al*., 1985	OMOE, 2008; Manitoba Conservation, 2005
Chronic	Oral	0.3 µg/kg-day	RfD	Hyperpigmentation, keratosis and possible vascular complications	Tseng *et al*., 1968; Tseng, 1977	U.S. EPA, 1993
	Inhalation	0.03 µg/m^3	REL	Decreased fetal weight; increased incidence of intrauterine growth retardation and skeletal malformations in mice	Nagymajtenyi *et al*., 1985	Cal EPA, 2000a
Cadmium						
Acute	Oral	4.1 µg/kg-day	MADL	Reproductive toxicity	Not provided	Cal EPA, 2006
	Inhalation	2 µg/m^3	24 hrs	Not provided	Not provided	Manitoba Conservation, 2005
Chronic	Oral	1 µg/kg-day	pTDI	Renal tubular dysfunction (proximal tubule epithelial cell damage), manifested as low molecular weight proteinura	WHO, 2001; 2004 based upon Friberg *et al*., 1971	Health Canada, 2008
Copper						
Acute	Oral	NA				
	Inhalation	50 µg/m^3	24 hrs	Health effects	Not Provided	OMOE, 2008; Manitoba Conservation, 2005
Chronic	Oral	90 (0-6 mo) 90 (7mo-4 yrs) 100 (5-11 yrs) 100 (12-19 yrs) 100 (20+, 70.7 kg) µg/kg-day	UL	Hepatoxicity, gastrointestinal effects	Pratt *et al*., 1985; O'Donohue *et al*., 1993	IOM, 2000
	Inhalation	1.0 µg/m^3	TCA	Respiratory and immunological Effects	Not Provided	RIVM, 2001
Lead						
Acute	Oral	NA	-	-		-

Table 4-42 Summary of the Non-Carcinogenic Toxicological Criteria Chosen for the Human Health Risk Assessment

Chemical	*Route*	*Toxicological Criterion*[a]		*Endpoint*	*Study*	*Regulatory Agency*
		Limit	*Type of Limit*			
	Inhalation	2 µg/m^3	24 hrs	Not Provided	Not provided	Manitoba Conservation, 2005
Chronic	Oral	3.6 µg/kg-day	pTDI	Increased blood lead concentration	Ziegler *et al*., 1978; Ryu *et al*., 1983	Health Canada, 2004a; 2008
	Inhalation	0.15 µg/m^3	AAQC (3 month averaging time)	NA	NA	U.S. EPA 2008
Mercury- Elemental						
Acute	Oral	NA	-	-	--	-
	Inhalation	NA	-	-	-	-
Chronic	Oral	NA	-	-	-	-
	Inhalation	0.06	TC (provisional)	Neurobehavioral effects	Ngim *et al*., 1992	Health Canada, 2008
Mercury- Inorganic						
Acute	Oral	7.0 µg/kg-day	MRL	Renal effects (increased absolute and relative kidney weights, increased incidence and severity of tubular necrosis)	NTP, 1993	ATSDR, 1999a
	Inhalation	2.0 µg/m^3	24 hrs	Health effects	Not Provided	OMOE, 2005b; 2008
Chronic	Oral	0.3 µg/kg-day	TDI	Kidney effects	Druet *et al*., 1978; Andres, 1984; Bernaudin *et al*., 1981	Health Canada, 2004a; 2008
	Inhalation	1.0 µg/m^3	Annual average guideline	Objective tremor, renal tubular effects (changes in plasma enzymes) and non-specific symptoms	WHO, 1991; Cardenas *et al*., 1993	WHO, 2000
Mercury- Methyl						
Acute	Oral	NA	-	-	-	-
	Inhalation	NA	-	-	-	-
Chronic	Oral	0.47 (general adult population); 0.2 (women of childbearing age, children <12 yrs) µg/kg-day	pTDI	Neurotoxicity and neurodvelopmental toxicity	Grandjean *et al*., 1997; Feeley and Lo, 1998	Health Canada, 2007a; 2008
	Inhalation	NA	-	-	-	-
Selenium						
Acute	Oral	NA	-	-	-	-

Table 4-42 Summary of the Non-Carcinogenic Toxicological Criteria Chosen for the Human Health Risk Assessment

Chemical	*Route*	*Toxicological Criterion*[a]		*Endpoint*	*Study*	*Regulatory Agency*
		Limit	*Type of Limit*			
	Inhalation	10 µg/m^3	24 hrs	Health effects	Not Provided	OMOE, 2008
Chronic	Oral	5.5(0-6 mo) 6.2(7mo-4 yrs) 6.3(5-11 yrs) 6.2(12-19 yrs) 5.7(20+, 70.7kg) µg/kg-day	UL	Selenosis	Shearer and Hadjimarkos, 1975; Yang and Zhou, 1994	Health Canada, 2008
	Inhalation	[b]20 µg/m^3	REL	Clinical selenosis	Yang *et al*., 1989b	Cal EPA, 2001

Note: For chemicals with no identified inhalation toxicological criteria, it was assumed that inhalation bioavailability and toxic potency is equivalent to that which occurs *via* the oral exposure route.

NA Not available

[a] AAQC = ambient air quality criteria; MRL = minimal risk level; RfD = reference dose; REL = reference exposure level; TCA = tolerable concentration in air; pTDI = provisional tolerable daily intake; UL = upper intake level; IOC_{POP} = intake of concern (population) – unlike an RfD or RfC (or similar benchmark), there is no established threshold or 'acceptable' or 'safe' levels for critical health effects of lead, at or below which no adverse health effects would be expected to occur.; TRV = toxicity reference value.

[b] The REL was derived from an oral RfD using a body weight of 70 kg and an inhalation rate of 20 m^3/day.

Table 4-43 Summary of the Carcinogenic Toxicological Criteria chosen for the Human Health Risk Assessment

Chemical	*Route*	*Toxicological Criterion*[a]		*Endpoint*	*Study*	*Regulatory Agency*
		Limit	*Type of Limit*			
Arsenic - inorganic	Oral	0.0015 (µg/kg-day)$^{-1}$	SF_O	Skin cancer prevalence rates	Tseng *et al*., 1968; Tseng, 1977	U.S. EPA IRIS, 1998
	Inhalation	0.0043 (µg/m^3)$^{-1}$	IUR	Lung cancer	Enterline and Marsh, 1982; Higgins, 1982; Brown and Chu, 1983a,b,c; Lee-Feldstein, 1983	U.S. EPA IRIS, 1998
Cadmium	Oral	NA	NA	NA	NA	NA
	Inhalation	0.0098 (µg/m^3)$^{-1}$	IUR	Detection of lung tumours	Takenaka *et al.*, 1983; Oldiges *et al*., 1984	Health Canada, 2004b; 2008
	Inhalation	NA	NA	NA	NA	NA

[a] SFo = oral slope factor; SFi = inhalation slope factor; IUR = inhalation unit risk; TRV = toxicity reference value.
NA Not available.

4.2.3 Summary of Toxicological Profiles

4.2.3.1 Arsenic

Arsenic occurs both naturally in the environment as a widely distributed component of the earth's crust and as a result of anthropogenic emissions (ATSDR, 2007b). It is an essential element in some animals but this has not been shown to be the case in humans. Arsenic may be found in four different oxidation states which dictate if it will bind to metals or non metals. These oxidation states are -3 (arsine), 0 (arsenic metal), +3 (arsenite) and +5 (arsenate). In differing oxidation states, arsenic is present in the environment in both the inorganic and organic forms of which are quickly absorbed by the body if ingested (Health Canada, 2006b; ATSDR, 2007b). The organic forms are rapidly eliminated from the body whereas the inorganic forms tend to accumulate more in the sulfhydryhl-rich tissues (Bertolero *et al*., 1987; Environment Canada, 1999; Health Canada, 2006b; ATSDR, 2007b). Major anthropogenic sources of arsenic include the burning of fossil fuels, waste incineration, ore refining facilities such as lead and copper smelting which release inorganic arsenic, and arsenical pesticides (Grayson, 1978; NTP, 2005a; Health Canada, 2006b; ATSDR, 2007b; HSDB, 2007). Some examples of inorganic arsenic include arsine gas (AsH_3) which is one of the most toxic arsenic compounds, as well as arsenic trioxide (As_2O_3), arsenic trisulfide (As_2S_3), sodium arsenite ($NaAsO_2$), arsenic pentoxide (As_2O_5), sodium arsenate (Na_2HAsO_4) and calcium arsenate ($Ca_3(ASO_4)_2$ (Opresko, 1992).

Exposure to arsenic within the general public is primarily through the ingestion of food such as seafood and water (NTP, 2005a). However, much of the arsenic in food items such as shellfish, fish and seaweed is present in the less toxic organic forms. Arsenic in drinking water, however, is typically in the inorganic form and, therefore, is considered to be a major potential source of exposure (Vahter *et al*., 1983; JECFA, 1988; Environment Canada, 1999; Health Canada, 2006b). Additional exposure to inorganic forms of arsenic may occur through incidental ingestion of contaminated soil and dust (Health Canada, 2006b).

The range of arsenic concentrations in naturally occurring soils (*i.e*., soil considered unimpacted by anthropogenic sources) have been reported to be 0.2 to 40 µg/g, with an average concentration of approximately 8.7 µg/g (Walsh and Keeney, 1975; Kabata-Pendias and Pendias, 1984; Adriano, 1986). Near base-metal smelters, average concentrations in Canada of arsenic in soil are elevated and typically range from 50 to 110 µg/g with a maximum reported concentration of 2,000 µg/g (Environment Canada, 1999). Although arsenic levels in soil may be elevated near smelter sites, animal and *in vitro* studies have shown that exposure to inorganic arsenic *via* soils/dust relative to more soluble forms (*e.g*., arsenic salts) is greatly reduced (Freeman *et al*., 1993; 1995; Groen *et al*., 1994; Davis *et al*., 1996; Hrudey *et al*., 1996; Ng and Moore, 1996; Ruby *et al*., 1996; Hamel *et al*., 1998; 1999; U.S. EPA, 1997b; Ng *et al*., 1998; Williams *et al*., 1998; Rodriguez *et al*., 1999; Turpeinen *et al*., 2003).

Fate and Transport

Arsenic is released into the air adsorbed onto small particles approximately 1 µm in diameter to be deposited onto an aquatic or terrestrial environment (Coles *et al*., 1979; Pacyna, 1987). Upon deposition to a water body, arsenic can undergo various processes such as oxidation-reduction, ligand exchange, precipitation and biotransformation of which are dependent on pH, metal sulfides, sulphide ion concentrations, iron concentrations, temperature, salinity, distribution of biota (biofilms) and organic matter content (U.S. EPA, 1979; Sanders *et al*., 1994; Wakao *et al*., 1988; Welch *et al*., 1988; Farago, 1997; Redman *et al*., 2002).

Partitioning to the soil or sediment from the water is dependent on the chemical form of arsenic and its interactions with other materials present (*e.g.*, biofilms, proportion of clays, iron oxides, *etc*.) (ATSDR, 2007b). In soils where arsenic is present, it is typically found as a mixture of mineral phases including co-precipitated, sorbed and dissolved species of arsenic. The amount of dissolved arsenic in soil is dependent on particle size, and the amount of arsenic distributed in the various phases. Distribution of the phases may also be indicative as to the source of the arsenic (Roberts *et al*., 2007).

In terrestrial systems, plants accumulate arsenic *via* the roots and leaves; however, relative to the environmental concentrations of arsenic, plant levels are relatively low (U.S. EPA, 1982; Gebel *et al*., 1998; Pitten *et al*., 1999). In aquatic environments, biomagnification is not significant (U.S. EPA, 1979; 1982; 1983; 2003; Mason *et al*., 2000). Bioaccumulation of arsenic depends on the environmental setting, organism, trophic status, exposure concentration and route uptake of the arsenic (Williams *et al*., 2006). Specifically, bioconcentration of arsenic in aquatic environments occurs significantly with the algae and the lower invertebrates. Bioconcentration at the predatory level is dependent on the organisms diet (ATSDR, 2007b).

Arsenic Toxicokinetics

In general, the more soluble the arsenic species the greater the potential for absorption (NAS, 1977; Hrudey *et al*., 1996). Typically, elemental arsenic (oxidation state of 0) is not absorbed by the body and is eliminated unchanged (HWC, 1989). Inorganic and organic forms of arsenic are typically well absorbed by the gastrointestinal tract, however, organic forms and arsenate (+5) are rapidly eliminated from the body. Arsenite (+3) tends to accumulate in the tissues (Bertolero *et al*., 1987). Dermal absorption of arsenic is believed to be low, evidence suggests that less than 5% of soluble arsenic compounds would be dermally absorbed and negligible amounts of particulate bound (soil or dust) arsenic is likely to cross the skin. ATSDR (2007) has indicated that dermal absorption of arsenic (organic and inorganic) is not a route that is likely to contribute towards a significant level of concern.

Arsenic absorbed into the system; binds onto haemoglobin and is distributed to various tissues and organs such as the liver, kidneys, lungs, spleen and skin within 24 hours for metabolism (Wickstroem, 1972; Axelson, 1978; Malachowski, 1990). Arsenic does not cross the blood-brain barrier, but transplacental transfer of arsenic may occur (Gibson and Gage, 1982). Metabolism of inorganic arsenic occurs primarily in the liver *via* enzymatic methylation (WHO-IPCS, 2001; Vahter, 2002). During the biotransformation process arsenate (+5) is quickly reduced to arsenite (+3) which under-goes enzymatic methylation to produce two metabolic products, including: monomethylarsinic acid (MMA) and dimethylarsinic acid (DMA) (U.S. NRC, 2001; Vahter, 2002).

Elimination of arsenic from the body occurs *via* urine within a 12 hour period after absorption, however, methylated products (MMA and DMA) can be excreted up to 2 to 3 days later (Buchet and Lauwerys, 1985; Lovel and Farmer, 1985; Malachowski, 1990). A small fraction of excreted arsenic compounds occurs in the feces, bile, sweat and breast milk as well as skin, hair and nails (ICRP, 1975; Malachowski, 1990; Hrudey *et al*., 1996; Kurttio *et al*., 1999; ATSDR, 2007b).

Biomonitoring

Exposure to total arsenic can be determined from urine, blood, hair, and nail samples (ATSDR, 2007b). Determination of recent exposure to arsenic is most readily and reliably obtained through urine samples (Millham and Strong, 1974; Pinto *et al*., 1977; Enterline *et al*., 1987;

Polissar *et al*., 1990). Blood samples are commonly used to evaluate the levels of an individual who has had an acute exposure to arsenic (Heydorn, 1970; Valentine *et al*., 1979, 1981; Driesback, 1980; Hindmarsh and McCurdy, 1986). Lastly, to determine exposure to arsenic from as recently as one month to up to 10 months, hair and nail samples may be analyzed, however external contamination with these samples is a high concern (Milham and Strong, 1974; U.S. EPA, 1977; Valentine *et al*., 1979; 1981; Bencko *et al*., 1986; Choucair and Ajax, 1988; Yamauchi *et al*., 1989; Agahian *et al*., 1990).

Arsenic Health Effects

Inhalation of inorganic arsenic commonly causes irritation to the throat and lungs (RAIS, 1997). Concentrations of 100 µg/m^3 or more, may lead to damaged mucous membranes and perforations of the nasal septum causing rhinitis, pharyngitis and laryngitis (U.S. EPA, 1984; RAIS, 1997; ATSDR, 2007b). Gastrointestinal effects are not typically associated with arsenic poisoning by inhalation but can occur if mucocillary transport of arsenic dust from the lungs to the gut occurs (Pinto and McGill, 1953; ATSDR, 2007b). Acute arsenic poisoning by inhalation in an occupational setting has been reported with workers who reported nausea, vomiting and diarrhoea (Pinto and McGill, 1953; Beckett *et al*., 1986; Bolla-Wilson and Bleecker, 1987; Ide and Bullough, 1988; Morton and Caron, 1989). Mortality from acute inhalation exposure to arsenic has not been reported and is not a concern (ATSDR, 2007b). Adverse health effects including circulatory and peripheral nervous disorders have been reported as a result of chronic inhalation exposure to arsenic (RAIS, 1997).

Acute oral exposure to concentrations of 1.2 and 21 mg/L of arsenic in well water has been found to lead to symptoms of abdominal pain, vomiting, diarrhoea, pain to the extremities and muscles and well as weakness with flushing of the skin as well as the appearance of a papular erythematous rash, hyperkeratosis of the palms and soles and deterioration of motor and sensory responses (Feinglass, 1973; Wagner *et al*., 1979; Fennel and Stacy, 1981; Murphy *et al*., 1981; Wesbey and Kunis, 1981).

Chronic oral exposure to inorganic arsenic in contaminated drinking water may result in dermal lesions such as hyperpigmentation, warts, hyperkeratosis of the palms and soles in adults exposed to 700 µg/day over a 5 to 15 year period or 2,800 µg/day over a 6 month to 3 year period (U.S. EPA, 2001a). One of the largest epidemiology studies conducted was by Tseng *et al*. (1968) and Tseng (1977) who observed an increased frequency in dermal lesions ("blackfoot disease") which is a peripheral vascular disorder in addition to an increased skin cancer incidence in a Taiwanese farming community who were exposed to varying levels of arsenic (≥0.60 mg/L, 0.3 to 0.59 mg/L, 0.01 to 0.29 mg/L) in their drinking water. Adverse effects to the skin, respiratory, cardiovascular and digestive systems have also been observed in children (<16 years old) who were exposed to mean arsenic concentrations of 0.6 mg/L in drinking water. The rate of effects decreased after levels were decreased to 0.08 mg/L but were still greater than the control group (Zaldivar and Ghai, 1980; Zaldivar, 1980). Infants who were exposed to an estimated amount of 3 mg/day of arsenic for two months through milk experienced effects to their central nervous system (CNS) which including hearing loss, eye damage, epilepsy, *etc*. as well as other effects such as melanoma and hyperkeratosis (Hammamoto, 1955; U.S. EPA, 1984).

Carcinogenicity

Arsenic has been classified as carcinogenic to humans by the U.S. EPA IRIS (1998). This is based on evidence that exposure to inorganic arsenic through the inhalation or oral route increases the risk of lung cancer in humans as well as observations of increased internal organ

cancers (liver, kidney, lung and bladder) and increased incidence of skin cancers (U.S. EPA IRIS, 1998; ATSDR, 2007b).

U.S. EPA IRIS (1998) derived an inhalation unit risk of 0.0043 $(\mu g/m^3)^{-1}$ for an increased incidence of lung cancer in occupationally exposed males employed in smelters (Enterline and Marsh, 1982; Higgins, 1982; Brown and Chu, 1983a,b,c; Lee-Feldstein, 1983). The unit risk is the upper-bound (95% confidence limit) lifetime cancer risk that is estimated to result from continuous exposure to a carcinogen (U.S. EPA, 1997b). An absolute-risk linear model was used to derive the unit risk of each study. The geometric mean of the unit risk was calculated for each data set that used a distinct exposed population (*e.g.*, Anaconda smelter and ASARCO smelter). The final estimate of the unit risk was calculated using the geometric mean of the unit risk from the Anaconda and ASARCO smelter data sets. The increase in age-specific mortality rate of lung cancer in these populations was attributed to cumulative exposure. The U.S. EPA value was adopted as the inhalation unit risk to evaluate the long-term human health effects of arsenic exposure in the current assessment.

The cancer potency for arsenic *via* oral consumption has been defined by an oral slope factor of 0.0015 $(\mu g /kg\text{-}day)^{-1}$ by the U.S. EPA IRIS (1998) which represents the upper-bound estimate (95% confidence limit) of the slope of the dose-response curve in the low dose region for carcinogens (U.S. EPA, 1997b). The dose-response curve is based on the data from the Tseng *et al*. (1968) and Tseng (1977) studies in which a Taiwanese farming community (population approximately 40,000) were exposed to differing levels of arsenic (high levels (≥0.60 mg/L), medium (0.3 to 0.59 mg/L) and low (0.01 to 0.29 mg/L)) in their drinking water originating from artesan wells and elevated incidences of skin cancer were detected in comparison to the control group. It was assumed that a constant exposure was experienced from birth, and that the drinking water consumption rate for males and females was 3.5 and 2.0 L/day, respectively. The doses were converted to equivalent doses for U.S. males and females and it was assumed that the skin cancer risk in the U.S. population would be similar to that in the Taiwanese population. Dose-specific and age-specific skin cancer prevalence rates were calculated using the multistage model with time. This value was adopted as the oral slope factor to evaluate long-term human health effects of exposure to arsenic.

Toxicological Reference Values (TRVs)

In addition to the carcinogenic TRVs selected, non-carcinogenic TRVs were also selected for both inhalation and oral exposure.

To identify the non-carcinogenic risks due to the inhalation of arsenic, acute and chronic TRVs were selected. An acute reference exposure level (REL) (4 hour exposure time) of 0.19 $\mu g/m^3$ was derived for arsenic by Cal EPA (1999a) for decreased fetal weight in mice during a developmental study (Nagymajtényi *et al*., 1985). Mice were exposed to arsenious acid *via* inhalation for 4 hours on gestation days 9 through 11. An uncertainty factor of 1,000 was applied to the study LOAEL of 190 $\mu g\ As/m^3$ for interspecies variation (10), the use of a LOAEL (10), and intraspecies variation (10). A chronic REL value of 0.03 $\mu g/m^3$ was set by Cal EPA (2000a) which represents the value below at which an adverse effect will occur (Collins *et al*., 2005). This value was derived from a study that examined maternal inhalation exposure in CFLP mice to 200 $\mu g/m^3$ of arsenic or 260 $\mu g/m^3$ of As_2O_3 for a 4 hour period per day during days of gestation. Reduction in fetal weight was observed with increased dose (Nagymajtenyi *et al*., 1985). The REL value derived in this study is supported with data from other animal studies (Kamil'dzhanov, 1982; Aranyi *et al.*, 1985). However, limitations of this REL include lack of human inhalation data, no observations at any concentration at which no observed effect was noted, as well as lack of comprehensive, long term, and multiple species studies. Also at lower

doses, results from this study may not apply (Cal EPA, 2000a). OMOE (2008) derived an AAQC (24 hour averaging time) for arsenic of 0.3 µg/m^3. This value was based on human health effects including irritation, sensitization, immunosuppression, teratogenesis, genotoxicity and carcinogenicity in exposed individuals. Manitoba Conservation (2005) has adopted this value as their 24-hour air guideline for Arsenic. No further information regarding the derivation of this exposure limit was available; however, it was selected as the 24 hour limit for arsenic for the current assessment. Cal EPA (2000a) utilized the same study in order to derive a chronic REL of 0.03 µg/m^3. This value was selected for the current assessment.

The acute oral minimal risk level (MRL) of 5 µg/kg-day derived by ATSDR (2007) was selected for the current HHRA risk assessment. The MRL is based on a LOAEL of 50 µg/kg-day for gastrointestinal effects and facial edema of a group of Japanese people who ingested arsenic contaminated soy sauce for a period of 2 to 3 weeks (Mizuta *et al*., 1956). An uncertainty factor of 10 was chosen based on a factor of 10 for the use of the LOAEL and 1 for human variability. The U.S. EPA (1993) determined an oral reference dose concentration of 0.3 µg/kg-day which estimates the daily oral consumption of inorganic arsenic that is not likely to cause adverse effects throughout a lifetime. The value selected is based on the epidemiology study of the Taiwanese farming community (Tseng *et al*., 1968; Tseng, 1977). Observations showed an increased incidence of hyperpigmentation, keratosis as well as vascular complications such as blackfoot disease with increasing age and dose.

4.2.3.2 Cadmium

Cadmium is a metal that exists in association with other elements, predominantly occurring as cadmium oxide (CdO), cadmium chloride ($CdCl_2$), cadmium sulphate ($CdSO_4$), and cadmium sulphide (CdS) (OMOE, 2006). Cadmium and its compounds are relatively stable in the environment, and are not readily degraded. Releases of cadmium into the environment can occur through zinc-sulphide-ore processing, cement production, fossil fuel combustion, sewage sludge and domestic waste disposal (OMOE, 2006). Fumes produced from the roasting of zinc ores and concentrates as well as from the precipitates obtained during the purification of zinc sulphate contain by-products of cadmium which may be recovered (ATSDR, 1999b; CCME, 1999a). Cadmium compounds are used in the manufacture of industrial and consumer products. Primary uses include electrodes in batteries, pigments in plastic products, heat stabilizers for polyvinyl chloride, anti-corrosive metal coatings, and alloy components (ATSDR, 1999b; OMOE, 2006).

Human exposure to cadmium occurs through the intake of food, water or accidental ingestion of contaminated dust or soil (Health Canada, 1986; NTP, 2005b). Additional exposure may also occur through the inhalation of cigarette smoke, occupational sources, and ambient air (ATSDR, 1999b; NTP, 2005b). For non-smokers, the principal source of cadmium is food. In the U.S. and Canada, food items have been found to contain approximately 2 to 40 ng/g cadmium (Dabeka and Mckenzie, 1992, 1995; NTP, 2005b). For smokers, cigarettes can be the principal source of cadmium (Friberg *et al*., 1986; NTP, 2005b). Exposure to cadmium through drinking water and ambient air tends to be quite low (ATSDR, 1999b). Cadmium concentrations in drinking water generally range from 0.01-1 µg/L and in more polluted areas, can be elevated up to 25 µg/L (Lauwerys *et al*., 1990; WHO, 1992; 2000). In air, concentrations of cadmium were found to range between 0.1 and 20 ng/m^3 for remote and urban areas in Europe, respectively (WHO, 2000). In soils, background concentrations in Canada range from non detectable to 8.1 µg/g (Whitby *et al*., 1978; Frank *et al.*, 1986). In areas near lead-zinc smelters such as in British Columbia, in the Columbia River Valley near the Comino smelter, average concentrations within a 10 km radius were 17.1 µg/g and in Flin Flon, Manitoba near the copper smelter, concentrations in garden soils were 3.2 to 13 µg/g (John, 1975; Pip, 1991). The Rouyn-

Noranda smelter in Quebec, had cadmium levels within a 1 to 3.7 km radius of 54 to 66 µg/g in the top 15 cm (CCME, 1999a). Cadmium is easily mobilized and assimilated into plants, and therefore, the consumption of food crops is typically an exposure pathway of concern (CCME, 1999a).

Fate and Transport

Cadmium is present in the atmosphere as suspended particulate matter which can travel large distances (*e.g.*, >1,000 km) over a period of 1 to 10 days with no susceptibility to photochemical reactions (Keitz, 1980; Elinder, 1985a). Cadmium may be transferred from the atmosphere to the terrestrial and aquatic environments through wet or dry deposition (WHO, 2000). Cadmium is unlikely to undergo photolysis or biomethylation in the aquatic environment (Callahan *et al*., 1979; U.S. EPA, 1983). In the water column, cadmium will complex predominantly with organic matter due to a strong affinity for humic acids (Callahan *et al*., 1979; McComish and Ong, 1988; NTP, 1991). Partitioning to the sediment is dependent on the precipitation and sorption of cadmium to mineral surfaces, hydrous metal oxides and organic materials, and is pH dependent (Callahan *et al*., 1979; Eisler, 1985; Feijtel *et al*., 1988; Muntau and Baudo, 1992). Partitioning of cadmium from water to sediments is also influenced by sediment bacteria (Burke and Pfister, 1988). Soil pH, oxidation-reduction and the formation of complexes are key factors affecting the mobility of cadmium in soils (McComish and Ong, 1988; Bermond and Bourgeois, 1992; Herrero and Martin, 1993). Typically, 90% of cadmium found in soils lies in the top 15 cm (Anonymous, 1994 (Cited ATSDR, 1999b)).

Aquatic and terrestrial biota bioaccumulate cadmium (Health Canada, 1986; ATSDR, 1999b). In crops, bioaccumulation varies from plant to plant (Kristensen *et al*., 1996) due to differences in the uptake of cadmium from the soil by the root system and differences in direct foliar uptake of surface deposited cadmium (ATSDR, 1999b). Cadmium tends to accumulate primarily in the leaves of plants (Alloway *et al*., 1990; He and Singh, 1994). In the food chain, animals and humans will preferentially bioaccumulate cadmium in the liver and kidneys compared to the muscle tissues (ATSDR, 1999b). The presence of acidic soil, in areas where acid rain is a concern, has been associated with enhanced cadmium uptake by plants and thus increased potential for bioaccumulation; potential leaching of cadmium into a water source is also enhanced at low pH levels (Callahan *et al*., 1979; Elinder, 1985a; Elinder, 1992).

Cadmium Toxicokinetics

Absorption of cadmium through inhalation occurs in the alveoli if the particles are <0.1 µm. Therefore, cadmium from cigarette smoke has a greater potential for absorption than from aerosols due to the smaller particle size. On average, 50% of particles <0.1 µm will be deposited at the alveoli and 50 to 100% of these particles will be absorbed (Nordberg *et al*., 1985). Absorption does not appear to be dependent on solubility but rather dependent on the different forms of cadmium (Glaser *et al*., 1986; Rusch *et al.*, 1986). Ingested cadmium, largely passes through the gastrointestinal tract without being absorbed except for some of the cadmium which is trapped in the intestinal mucosa (Kjellstrom *et al*., 1978; Foulkes, 1984). Dermal absorption was studied in one *in vitro* study using human skin (Wester *et al.*, 1992) and does not appear to be a concern unless the skin is in contact for several hours with the cadmium compound (ATSDR, 1999b).

Once absorbed, cadmium is distributed to all tissues however the liver and the kidney contain the greatest concentrations; approximately 40 to 80% of the entire body burden (Sumino *et al.*, 1975; Chung *et al.,* 1986; WHO, 2000). In the liver cadmium concentrations rise to 1 to 2 µg/g

(wet weight) in adults of 20 to 25 years old and then slightly increase thereafter (Hammer *et al.,* 1973; Sumino *et al.*, 1975; Chung *et al.,* 1986; Lauwerys *et al.,* 1984). In the kidneys, concentrations of 40 to 50 µg/g (wet weight) are found in adults of 50 to 60 years old and concentrations either plateau or decline thereafter (Hammer *et al.,* 1973; Lauwerys *et al.,* 1984; Chung *et al.,* 1986). The placenta acts as a partial barrier to fetal exposure to cadmium; although levels of exposure have been found to vary (Kuhnert *et al.,* 1982; Truska *et al.,* 1989). Cadmium levels in cord blood are half of what is present in the maternal blood (Lauwerys *et al.,* 1978; Kuhnert *et al.,* 1982; Truska *et al.,* 1989). Human milk contains 5 to 10% of the cadmium levels that are in the blood (Radisch *et al.,* 1987).

In terms of metabolism, cadmium does not undergo oxidation, reduction or alkylation (ATSDR, 1999b). Cadmium (+2) does bind to proteins such as albumin and metallothionein to circulate in the plasma (Nordberg *et al.,* 1985; Foulkes and Blanck, 1990). Cadmium blood levels in the general population are generally <0.5 µg/100 mL (WHO, 2000)

Much of the cadmium that enters the body is excreted in the feces or urine (Kjellstrom and Nordberg, 1978).

Biomonitoring

Exposure to cadmium can be measured in the blood, urine, feces, liver, kidney and hair (ATSDR, 1999b). Recent cadmium exposure can be determined by measuring blood cadmium levels (Ghezzi *et al*., 1985; Jarup *et al*., 1988; Roels *et al*., 1989; Lauwerys *et al*., 1994). To detect chronic exposure to cadmium, urinary cadmium levels may be measured (Bernard and Lauwerys, 1986). Once cadmium-induced renal damage occurs however, urinary cadmium levels may increase sharply in response to intrarenal cadmium release and a decrease in renal reabsorption of cadmium (Roels *et al*., 1981; Lauwerys *et al*., 1994). Under these circumstances, urinary cadmium can also be reflective of recent exposure (Lauwerys *et al*., 1994). Fecal matter can be measured for dietary exposure to cadmium since cadmium is poorly absorbed by the gastrointestinal tract (Kjellstrom *et al*., 1978; Adamsson *et al*., 1979). Liver and kidney tissues can also be measured to determine cadmium exposure since these tissues preferentially bioaccumulate cadmium (ATSDR, 1999b). Generally, cadmium concentrations measured from the kidney cortex peak around 50 to 60 years of age in an individual, whereas, liver cadmium concentrations continue to increase over time (Elinder, 1985b; ATSDR, 1999b). Cadmium concentrations measured from hair samples is controversial due to the potential for contamination; therefore this type of measurement is better used when there has been high exposure to cadmium rather than low exposure (Huel *et al*., 1984; Shaikh and Smith, 1984; Wilhelm *et al*., 1990; Frery *et al*., 1993; Lauwerys *et al*., 1994).

Cadmium Health Effects

Chronic effects resulting from inhalation exposure including bronchitis, emphysema and renal effects including proteinuria (kidney disease) have been seen in workers exposed to cadmium. To minimize risk, WHO (2000) suggested a critical concentration of 0.005 mg/m^3 for occupational exposure (8 hours) to cadmium and 0.0003 mg/m^3 for chronic inhalation exposure. Chronic oral exposure has resulted in renal damage from rice produced in a contaminated region of Japan (Nogawa *et al.,* 1989). Renal effects were also documented in a study by Shiwen *et al.* (1990), which found tubule interstitial lesions at 78 µg/kg/day resulting from environmental exposure estimated over 25 years. At this concentration, muscular, skeletal, and haematological effects were not found. The highest renal level of cadmium not associated with significant kidney disease was reported as 200 µg Cd/g (wet weight) of human renal cortex by

the U.S. EPA (1985a). Other effects resulting from chronic oral exposure to cadmium and its compounds have not been clearly demonstrated in scientific studies.

Carcinogenicity

The U.S. EPA (1985b) listed cadmium and its compounds in Group B1, denoted as "probable human carcinogens " due to sufficient evidence of lung cancer in laboratory animals (Takenaka *et al.,* 1983), but limited evidence of lung cancer in humans resulting from occupational exposure (Thun *et al.,* 1985). For the current assessment cadmium was assessed as a human carcinogen *via* inhalation only since there are not sufficient studies of oral consumption of cadmium to assess the carcinogenic risk (U.S. EPA, IRIS, 1992).

An inhalation unit risk of 0.0098 $(\mu g/m^3)^{-1}$ was selected for the current assessment (Health Canada, 2004b; 2008). This value was based upon the detection of lung tumours in a chronic study (Takenaka *et al*., 1983; Oldiges *et al*., 1984) whereby rats were exposed to cadmium chloride aerosols over a total period of 18 months with dosing concentrations of 12.5, 25 and 50 $\mu g/m^3$ for 23 hours/day, 7 days/week (Health Canada, 2004b; 2008).

Toxicological Reference Doses (TRVs)

Non-carcinogenic TRVs were selected to assess risk for both short-term inhalation and oral exposure, as well as chronic oral exposure. Chronic inhalation exposures were not assessed in this manner.

The OMOE (2006) has proposed a 24 hour ambient air quality criteria (AAQC) for cadmium of 0.025 $\mu g/m^3$. This value was based upon the proposed annual average AAQC adjusted by a factor of 5. This value comes into effect in Ontario after February 2013. Until February 2013, the upper risk threshold value of 0.25 ug/m^3 has effectively been adopted as the AAQC. The Manitoba Conservation (2005) air guideline value of 2 ug/m^3 was adopted as the 24 hour exposure limit for cadmium in the current assessment. In addition, OMOE (2006) proposed a chronic AAQC (annual averaging time) of 0.005 $\mu g/m^3$ which was based on the scientific approach used by the European Commission (2000). The value was derived by applying an uncertainty factor of 50 (5 for use of a LOAEL and 10 for interspecies variability) to an adjusted LOAEL of 0.27 $\mu g/m^3$ for proteinuria associated with proximal tubular dysfunction and lung cancer as a result of workplace exposure (Thun *et al*., 1991). This value, which was consistent with the chronic inhalation RfC for cadmium recommended by the European Commission (2000) and WHO (2000), was not selected as the chronic inhalation exposure limit in the current assessment since it was based on a cancer endpoint , which is addressed through the Health Canada unit risk value.

Cal EPA (2006) has derived a maximum allowable daily level (MADL) of 4.1 µg/day based on the oral intake of cadmium. The MADL was calculated by dividing the NOEL by 1,000. This value was chosen as the exposure limit for the acute oral pathway in the current assessment. The U.S. EPA IRIS (1994) derived a chronic oral RfD for diet of 1 µg/kg-day. The choice of NOAEL used to develop the chronic RfD does not reflect the information from a single study, but rather data from many studies on the toxicity of cadmium in both humans and animals. These data also permit calculation of pharmacokinetic parameters of cadmium absorption, distribution, metabolism and elimination, therefore, high confidence is given for the oral RfD and database. Health Canada (2008) have recommended a similar value for their pTDI. This value, which was consistent with the chronic inhalation RfC for cadmium utilized by WHO (2005), was selected as the chronic oral exposure limit in the current assessment.

4.2.3.3 Copper

Copper is an essential element in living organisms. It is a reddish-brown, odourless metal that occurs naturally in the environment in its metallic form or in minerals (ATSDR, 2004). In general, copper binds readily to organic and inorganic matter in soil, water and sediments, and is rarely present in its free form. Due to its durability, malleability, ductility, and thermal and electrical conductivity, copper is frequently used in its metallic form or as an alloy in construction, electrical products and systems, transportation and industrial equipment. In addition, copper compounds may also be used as fungicides, algicides, and repellents. Copper sulphate is the mostly widely produced copper compound, and is utilized in various industrial and agricultural applications (ATSDR, 2004).

Copper is released into the environment as a result of mining activities (copper and other metals), combustion of fuels and wastes, wood production, fertilizer use and production, and landfills. The major release of copper to land is from tailings and overburdens from mines and sewage sludge. Natural sources of copper result from volcanoes, fires, decomposition of organic material, and sea spray (ATSDR, 2004). Mean copper concentrations in soils across the U.S. are dependent on the soil type and the land use and can range from 14 to 41 mg/kg (dry weight) (Fuhrer, 1986; Chen *et al*., 1999). In areas near smelter operations, where copper emissions are elevated, copper concentrations have been found to be approximately 2,480 ppm in the first 5 cm of top soil (Davis and Bennett, 1985). In the vicinity of the Sudbury smelter, maximum wetland soil/sediment copper concentrations were 6,912 ppm (Taylor and Crowder, 1983). In all cases near smelter operations, copper concentrations have been found to decrease with distance to the main stack (Taylor and Crowder, 1983; ATSDR, 2004).

Human exposure occurs primarily through regular consumption of food and water and through inhalation as well as dermal contact with air, water and soil (ATSDR, 2004). However, increased copper exposure tends to occur through drinking water. In the U.S., typical copper concentrations in drinking water are a few ppbs to 10 ppm. Increased exposure may also occur in smelter/mining areas where there are copper emissions through the inhalation of dust containing elevated levels of copper (ATSDR, 2004).

Fate and Transport

Copper is released to the atmosphere adsorbed to particulate matter (PM) and can remain bound to PM in the troposphere for approximately seven to thirty days (Perwak *et al*., 1980). Removal from the atmosphere occurs *via* gravitational settling as well as wet and dry deposition. The rate of removal is dependent on various factors including particle size, turbulence, and wind velocity (ATSDR, 2004). Once deposited in surface waters, copper will primarily be adsorbed by forming stable ligands to organic matter and iron oxides within the first hour (Davies-Colley *et al*., 1984; Harrison and Bishop, 1984; Kust, 1978; U.S. EPA, 1979). Formation of these ligands will affect future adsorption, precipitation and oxidation-reduction reactions (U.S. EPA, 1979).

In sediment and soils, copper can adsorb to organic matter, carbonate minerals, clay minerals and/or hydrous iron and manganese oxides (Janssen *et al*., 1997; Petruzzelli, 1997; U.S. EPA, 1979; Tyler and McBride, 1982; Fuhrer, 1986). At pH levels greater than five, copper has been found to adsorb to soils but at pH levels less than five copper remains in porewater and is mobile in the soil; therefore, increasing the occurrence of leaching into groundwater (Perwak *et al*., 1980; Luncan-Bouche *et al*., 1997). At sites where acid rain is a concern due to the SO_x

emissions (*e.g.*, smelter sites), soil pH levels may be lower therefore increasing the leachability of copper (Amrhein *et al*., 1992).

Copper has not been found to biomagnify at higher levels in the food chain, although there is some concern of bioconcentration at lower levels (*i.e.*, within mollusks) (Perwak *et al*., 1980; Bradley and Morris, 1986). In plants, copper is an essential nutrient and its uptake is dependent on the concentration and bioavailability of copper in the soil. Other factors that will influence copper uptake in plants includes root surface area, plant genotype, stage of plant growth, weather conditions, and interaction with other nutrients (Clemens, 2001; Gupta, 1979).

Copper Toxicokinetics

To date, no studies have been located which determine the rate and extent of absorption following inhalation exposure of copper to humans (ATSDR, 2004). Oral consumption of copper, leads to absorption into the system from the stomach and the small intestine. The site of maximum copper absorption for humans is unknown, however, it is suspected to be in the stomach and the duodenum (Bearn and Kunkel, 1955). Copper may be absorbed as ionic copper or bound to amino acids and a small concentration of copper is transported from the mucosal side of the intestine to the serosal side (Gitlin *et al*., 1960; Crampton *et al*., 1965). A greater portion of copper is absorbed *via* the absorptive surface and mucosal uptake followed by binding to metallothionein or another binding protein in the intestine (Evans and LeBlanc, 1976). Factors affecting copper absorption include, the amount of copper in the diet, competition with other metals and age (Farrer and Mistilis, 1967; Hall *et al*., 1979; Haschke *et al*., 1986; Hooegenraad *et al*., 1979; Prasad *et al*., 1978; Strickland *et al*., 1972; Davies and Campbell, 1977; Turnlund *et al*., 1988; 1989; Varada *et al*., 1993). Generally, copper metabolism involves transfer to and from different organic ligands such as sulfhydryl and imidazole groups on amino acids and proteins. Specific binding proteins for uptake, storage and release have been identified for copper. In the liver, copper is bound to metallothionein and amino acids and can induce metallothionein synthesis as well as ceruloplasmin biosynthesis (Evans *et al*., 1970; Haywood and Comerford, 1980; Mercer *et al*., 1981; Wake and Mercer, 1985). From this point, copper can be slowly released from metallothionein to the bloodstream thereby eliminated *via* the bile or excreted when the mucosal cell is degraded (ATSDR, 2004). Dermal exposure to copper does not result in a great amount of exposure due to poor absorption of the copper through the skin as seen with *in vivo* and *in vitro* data (ATSDR, 2004; Pirot *et al*., 1996a,b).

Biomonitoring

Exposure to levels of copper can be measured in the blood serum, urine, hair, toenails and liver (ATSDR, 2004). Chuttani *et al*. (1965) found increased serum levels in individuals exposed to a single dose of copper sulphate compared to non-exposed individuals. However, increased serum levels may only be indicative of recent exposure (ATSDR, 2004). Exposure may also be assessed through the examination of hair and toenails. Due to differing growth rates of hair and toenails, the first 2 cm of a hair strand (closest to the scalp) can be analyzed to measure copper exposure within the past two months and toenails can be used to determine exposure over the past 12 to 18 months (Hopps, 1977; Fleckman, 1985). In cases of excess copper intake, the liver has been a primary target and there have been reports of changes in the serum enzymes resulting from liver damage (Chuttani *et al*., 1965; Haywood, 1980; Haywood and Comerford, 1980; Epstein *et al*., 1982; NTP, 1993; Sugawara *et al*., 1995; Muller *et al*., 1998). As well, increased levels of bilirubin have also been detected however, these particular changes may not necessarily be uniquely related to excess copper intake (ATSDR, 2004)

Copper Health Effects

Occupational exposure to copper has resulted in "metal fume fever"; an acute illness characterized by fever, metallic taste in the mouth, aching muscles, headache, and dryness of the mouth and throat (ATSDR, 2004). Gleason (1968) observed symptoms of metal fume fever in workers exposed to 30 to 120 µg/m^3 copper dust for a number of weeks. A "sweet taste in the mouth" (consistent with symptoms of metal fume fever) was observed in workers upon exposure to 1,000 to 3,000 µg/m^3 copper fume for unspecified short periods of time (Whitman, 1957). Upper respiratory irritation occurred in addition to metal fume fever in workers exposed to copper cutting brass pipe with an electric cutting tool for 1 to 10 hours (Armstrong *et al.,* 1983). Ocular irritation was observed in factory workers exposed to copper dust for an unspecified duration (Askergen and Mellgren, 1975). In the majority of studies identified, co-exposure to other metals in addition to copper occurred (Cal EPA, 1999b; ATSDR, 2004).

Copper was found to irritate the respiratory system in workers spraying with an agent containing 1 to 2.5% copper sulphate (neutralized with lime) (Pimentel and Marques, 1969). Further case studies reported symptoms of intra-aveolar desquamation of macrophages, formation of histiocytic and noncaseating granulomas with inclusions of copper; however, concentration-specific information is not available (Pimentel and Marques, 1969; Plamenac *et al.,* 1985). Suciu *et al.* (1981) reported pulmonary fibrosis and nodulation in workers exposed to concentrations of copper ranging from 111 to 434 mg/m^3 over a period of 1 to 3 years. Askergren and Mellgren (1975) observed vascularity and superficial epistatic vessels in the nasal mucosa of sheet metal workers exposed to unquantified concentrations of patina dust (a mixture of copper-containing compounds).

Hepatic, neurological, and reproductive effects have been reported in workers exposed to copper dust concentrations ranging from 111 to 434 mg/m^3 as a result of sieving copper for 1 to 3 years (Suciu *et al.,* 1981). Gastrointestinal effects were also observed with copper intoxication, but these may have been attributed to oral exposure. No reproductive or developmental effects in humans were reported to result from chronic inhalation exposure (Cal EPA, 1999b; ATSDR, 2004). Haematological effects such as decreased haemoglobin and erythrocyte levels were reported in workers exposed to copper concentrations of 640 µg/m^3 for an unspecified period of time (Finelli *et al*., 1981). However, workers were exposed to other metals and this was not taken into consideration in the study.

A limited number of human studies were located that identified adverse effects of chronic oral exposure to copper; hepatotoxicity and gastrointestinal effects have been noted in individuals consumping copper gluconate capsules.

Carcinogenicity

IARC, NTP, U.S. EPA, Cal EPA and OSHA have not classified the carcinogenicity copper. For the purposes of this risk assessment copper was assessed as a non-carcinogen.

Toxicological Reference Doses (TRVs)

Cal EPA (1999b) derived an acute REL for a 1 hour exposure of 100 µg/m^3 for copper. The critical effect was metal fume fever which was observed in workers occupationally exposed to copper for an unknown duration (Whitman, 1957; 1962; Gleason, 1968; ACGIH, 1991). An uncertainty factor of 10 for intraspacies variation was applied to the NOAEL of 1,000 µg/m^3. OMOE (2008) lists an AAQC (24 hour average) of 50 µg/m^3 for copper, based on the protection

of human health effects. Manitoba Conservation (2005) has also adopted this value as their 24-hour air guideline value. No further information regarding the basis of this exposure limit was available; however, it was selected as the 24 hour TRV for copper. RIVM (2001) have derived a Tolerable Concentration in Air (TCA) of 1.0 ug/m^3 for copper. This value has been adopted as the chronic TRV for the current assessment.

ATSDR (2004) derived an acute oral MRL of 10 µg/kg-day and an intermediate oral MRL of 10 µg/kg-day to be protective of health effects from copper levels in drinking water. The acute MRL is based on a study where gastrointestinal disturbances were experienced in women who were ingesting 73.1 µg/kg-day of copper in drinking water over a 2 week period. A NOAEL of 27.2 µg/kg-day was derived and an MRL was calculated using an uncertainty factor of 3 to account for human variability. An intermediate MRL was derived based on the study where gastrointestinal disturbances in men and women were detected who were consuming 91 µg/kg-day of copper in drinking water over a two month period. A NOAEL of 42 µg/kg-day was determined and an MRL was calculated using an uncertainty factor of 3 for human variability. This value was not choosed in the the current assessment since it is 10-fold less than the chronic TRV derived by Health Canada (see below).

IOM (2000) derived a series of tolerable daily intake levels, *i.e*., Upper Levels (ULs), for different age groups. The ULs range from 1,000 for 1 to 3 year olds to 10,000 µg/day for adults (male weight: 76 kg; female weight: 61 kg). The ULs were determined using human studies by Pratt *et al*. (1985) and O'Donohue *et al*. (1993) where hepatotoxicity and gastrointestinal effects where noted in individuals consumping copper gluconate capsules for a given duration. Health Canada has indicated that the agency will officially adopt upper intake level (ULs) as toxicity reference values for all essential elements (Roest and Petrovic, 2005 pers. comm.) for contaminated sites human health risk assessments. The UL values adopted by Health Canada are equal to those of IOM but have been adjusted for the duration of the life stage and body weight (Health Canada, 2008). These values were selected as the chronic oral exposure limit for the current assessment.

4.2.3.4 Lead

Lead is a naturally occurring metallic element that occurs in a variety of minerals, often in close association with zinc (CCME, 1999b). Most of the lead found in the environment is the result of mining and smelting operations as well as combustion sources (Corrin and Natusch, 1977; U.S. EPA, 1986; Environment Canada, 1996; Spear *et al*., 1998). Due to several desirable properties, including the ability to resist corrosion, lead has historically been used in a wide variety of products in both industrial applications and residential uses, particularly lead-storage batteries for vehicles and general industry (*e.g*., piping, cable covering, bearing metals for machinery, and sheet lead, *etc*.) (ATSDR, 2007a). Organic forms of lead have traditionally been used as additives in vehicle fuels (ATSDR, 2007a).

The general population may be exposed to lead *via* food items, cigarette smoke, contaminated dust and/or soil present in food items as well as exposure to ink, paint and plaster which may contain lead (CCME, 1999b; ATSDR, 2007a). Absorption of lead into the body from these items can occur through inhalation, ingestion, dermal contact and for foetuses, exposure through the placenta. In Ontario, Canada, lead levels in drinking water ranged from 1.1 to 30.7 µg/L with a median of 4.8 µg/L. This contributes to an average lead consumption from 1.5 L of drinking water of 7.2 µg for adults and 2.9 µg for children. Lead can be present in food items inadvertently through lead-soldered cans or through fall out of lead emissions onto fruits and vegetables. For children, lead in soils and household dust can be a significant contributor

towards exposure (U.S. EPA, 1986). In Canadian soils, background lead levels have been reported to range from 12 to 25 µg/g.

Specifically, in the cordilleran region, lead levels have been detected at 16 µg/g whereas in the Appalachian and Canadian shield levels have been at 21 µg/g. Similarly, background lead levels for soils in the U.S. have been measured to range from <10 to 30 µg/g (ATSDR, 2007a). In Missouri, soils next to the smelter had greater concentrations of lead which were approximately 60,000 µg/g (Palmer and Kucera, 1980). Soils which also contained elevated levels of lead were those beside roadways which have been found to be 30 to 2,000 µg/g higher than natural levels but which decrease significantly the further away from the roadway (U.S. EPA, 1986). In Minneapoli/St. Paul, lead levels in the soils were 60 times higher (423 µg/g) than those in rural Minnesota (6.7 µg/g). Elevated lead levels near roadways have been attributed to the combustion of leaded gasoline (ATSDR, 2007a). Other sources of lead include lead paint which results in lead levels greater than 10,000 µg/g in soils next to homes with exterior lead pain (U.S. EPA, 1986).

Fate and Transport

Particulate-bound lead emitted from mining operations, smelters, and combustion sources occurs primarily in the form of lead-sulfur compounds; however, in the atmosphere, lead exists primarily in the form of particulate-bound lead sulphate and lead carbonate and has a residence time of approximately 10 days (Corrin and Natusch, 1977; NAS, 1980; U.S. EPA, 1986; Spear *et al*., 1998; OMOE, 2006; ATSDR, 2007a). Once in the environment, lead may transform, but does not degrade and cannot be destroyed (ATSDR, 2007a).

Soil is a major sink for lead deposited from the atmosphere. Generally, lead is immobile in soil due to the complexes it forms with organic matter; however pH, mineral composition, microbial activity, ion exchange capacity, and presence of inorganic colloids and iron oxides also influence transport and bioavailability (WHO, 1995; CCME, 1999b; U.S EPA, 2006; ATSDR, 2007a). In addition to the complexes formed and the pH of the soil, the mobility of lead in the soil also varies with the different forms of lead released into the environment (CCME, 1999b). Soil properties also affect the speciation of lead within the soil. In aerobic soils, lead compounds undergo weathering and may become more stable over time (CCME, 1999b). In anaerobic soils, much of the sulphate is reduced to sulphide which is highly stable, insoluble and a relatively non-reactive lead species. Generally, lead does not bioaccumulate in terrestrial or aquatic food chains (ATSDR, 2007a).

Lead enters aquatic ecosystems through atmospheric deposition, runoff, and industrial wastewater. It exists in the form dissolved lead, suspended particulate, or sediment (ATSDR, 2007a). Lead partitions rapidly between sediment and water, depending on pH, salt content, sulphur content and presence of organic matter (WHO, 1995). Generally, lead does not volatilize readily into the atmosphere from water. Once present in the sediment, lead is stable and has a long residence time, and this medium is major sink for lead (U.S. EPA, 2006). Lead does not biomagnify in aquatic ecosystems. In the aquatic food chain, lead concentrations are the greatest in benthic organisms and algae in comparison to top predators (ATSDR, 2007a).

Lead Toxicokinetics

Absorption of lead *via* inhalation is dependent on the particle size, solubility and location of the deposited lead particles within the respiratory tract. Larger particles (>2.5 µm) are deposited in the ciliated airways and then transferred to the esophagus by mucociliary transport to be

eventually swallowed. Smaller particles (<1 µm) are deposited into the alveolar region and absorbed after extracellular dissolution or after ingestion by phagocytic cells (ATSDR, 2007a). If ingested, the degree of absorption of lead will be dependent on factors/status such as age, diet, and pregnancy as well as particle size, solubility and species of lead. In addition, the amount of lead absorbed may vary with the amount of lead ingested (ATSDR, 2007a). Specifically, studies have shown that absorption of lead is greater in children than in adults (Alexander *et al*., 1974; Ziegler *et al*., 1978; Rabinowitz *et al*., 1980; Heard and Chamberlain, 1982; James *et al*., 1985; Watson *et al*., 1986). In terms of dietary status, it was shown that absorption of lead is decreased significantly in individuals who have consumed a meal with the ingested lead (Rabinowitz *et al*., 1980; Heard and Chamberlain, 1982; Blake and Mann, 1983; Blake *et al*., 1983; James *et al*., 1985; Maddaloni *et al*., 1998). Children and adults who were iron and calcium deficient showed higher blood lead levels (Ziegler *et al*., 1978; Heard and Chamberlain, 1982; Blake and Mann, 1983; Mahaffey and Annest, 1986; Mahaffey *et al*., 1986; Marcus and Schwartz, 1987;). Although increased blood lead levels have been observed in pregnant women, there is no direct evidence that it is a result of increased absorption but rather a factor of increased bone lead mobilization (Rothenberg *et al*., 1994; Lagerkvist *et al*., 1996; Schuhmacher *et al*., 1996; Gulson *et al*., 1997; 1998b; 2004).

The distribution of absorbed lead was found to be the same regardless of the route of entry into the body, in both children and adults (Chamberlain *et al*., 1978; Kehoe, 1987). In blood, most of the circulating lead (99%) is found in red blood cells, primarly bound to plasma proteins. Lead that is not protein-bound is found in complexes with sulfhydryl compounds and other ligands (Ong and Lee, 1980; Al-Modhefer *et al*., 1991; Schutz *et al*., 1996; Bergdahl *et al*., 1997; 1999; Hernandez-Avila *et al.*, 1998; Manton *et al*., 2001; Smith *et al*., 2002). The half-life of lead in blood is approximately 30 days (Griffin *et al*.,1975; Rabinowitz *et al*., 1976; Chamberlain *et al*., 1978), therefore blood lead levels do not accurately reflect exposure that occurred more than six weeks prior to testing (Patrick, 2006). Lead in the body continues to circulate until it is either excreted or deposited in soft tissues including the liver, renal cortex, aorta, brain, lungs, and spleen, as well as the bones where the total body burden of lead is mainly stored (Hu *et al*., 1996).

The half-life of lead in human bone is estimated to be up to 30 years (Ibrahim *et al*., 2006). In adults, approximately 80 to 90% of retained lead is stored in bone. For children, less (~70%) is stored in bone, indicating that significantly more lead burden in children is in the soft tissues (Patrick, 2006). During infancy and childhood, deposition of lead occurs in trabecular bone (high turnover); while in adulthood, both trabecular and cortical (tibia) bones are sites of deposition (Patrick, 2006). Lead stored in the bone generally increases in concentration with age and can be released back into blood over time (Schroeder and Tipton, 1968; Barry, 1975; 1981; Gross *et al*., 1975). Conditions that increase bone turnover (pregnancy, lactation, postmenopausal osteoporosis, hyperthyroidism and *cis*-platin chemotherapy) can mobilize stores of lead into circulation (Patrick, 2006). Bone lead can be transferred to a fetus during production of the fetal skeleton (Franklin *et al*., 1997; Gulson *et al*., 1997; 1999; 2003). Maternal lead can also be transferred to the fetus *via* cord blood or through breastfeeding (Goyer, 1990; Graziano *et al*., 1990; Carbone *et al*., 1998; Gulson *et al*., 1998a; Ettinger *et al*., 2006).

Metabolism of inorganic lead occurs *via* the formation complexes with both protein and non protein ligands such as albumen. Lead will form complexes with ALAD; an intracellular ligand in red blood cells. Lead may also form complexes with proteins in the cell nucleus and cytosol (ATSDR, 2007a). The complexes that lead forms often cause interference with numerous physiological processes including haeme biosynthesis by inhibition of δ-aminolevulinic acid

dehydratase (δ-ALAD), probably through its high affinity for the zinc-binding site in the enzyme. Although lead displaces zinc more readily in one of the alloenzymes of the protein, the relationship between the δ-ALAD genotype and sensitivity to lead at different blood lead concentrations remains unclear. Lead also causes an increase in zinc protoporphyrin, by a mechanism which is not fully established. Lead inhibits pyrimidine-5′-nucleotidase, resulting in accumulation of nucleotides, and subsequent haemolysis and anemia (IARC, 2004).

Final excretion of lead from the body occurs primarily through urine and feces but may also be released in minor amounts through sweat, saliva, hair, nails and breast milk (Hursh and Suomela, 1968; Hursh *et al*., 1969; Griffin *et al*., 1975; Rabinowitz *et al*., 1976; Chamberlain *et al*., 1978; Kehoe, 1987; Stauber *et al*., 1994).

Biomonitoring

Human blood, hair, teeth, bone and urine are used to determine lead exposure. Blood is the most widely used to determine short-term exposures because of its short half-life, which is approximately 36 days (ATSDR, 2007a). Average blood lead levels in the general population, as reported in the National Health and Nutritional Examination Survey (NHANES IV), were 1.7 µg/dL and 1.46 µg/dL in children (1 to 5 years) and adults (≥20 years), respectively in 2001 to 2002 (CDC, 2005). The mean blood lead level from occupational exposure is 2.42 µg/dL which is slightly greater than in the general population (Yassin *et al.,* 2004).

Bone is a good biomarker of cumulative lead exposure because lead will accumulate in the bone over a lifetime, with a half-life ranging from 12 to 27 years (U.S. EPA, 2006). A high proportion of the total body burden of lead occurs in bone, ranging from 90% in adults to 70% in children. Like bones, teeth may also be used in determining long-term lead exposure. Tooth enamel is indicative of *in utero* exposure, while dentin lead reflects exposure postnatal exposure up until the time that the tooth is shed (U.S. EPA, 2006). Unlike bone, lead in teeth does not significantly contribute to lead body burden. Due to the high variability in lead levels from tooth analysis as well as moderate correlations between blood and tooth lead, teeth are not used as a biomarker for regulatory policy of lead exposure or toxicity (Mushak, 1991; U.S. EPA, 2006).

Urinary lead has been used as in indicator of current lead exposure. It is not regarded as a reliable biomarker due to low and fluctuating levels of lead (WHO, 1995). However, after administration of a chelating agent ($CaNa_2$-ETDA), urinary lead is considered to be an excellent measure of the toxic fraction of the total body burden of lead. Chelatable lead that is excreted in urine is representative of lead removal from soft tissues, blood, and bone (WHO, 1995). It has been used to confirm elevated lead body burden (ATSDR, 2007a).

Levels of lead in hair have been used as an indictor of exposure in children and also in some epidemiological studies (WHO, 1995; U.S. EPA, 2006). However, lead in hair is not considered to be a useful biomarker of lead exposure. This is primarily due to variations caused by different hair colors, textures, growth phases, and chemical treatments (WHO, 1995).

Lead Health Effects

The toxic effects of lead in humans are widely believed to be the same regardless of the route of entry, and are correlated to blood lead in the vast majority of studies (ATSDR, 2007a). The effects from chronic exposure to lead on humans are primarily neurological, renal, hematological, reproductive, and developmental (CDC, 1991; ATSDR, 2007a). Well-characterized human health effects include neurotoxicity and renal toxicity, which can be severe

at blood lead levels greater than 120 µg/dL (U.S. EPA, 1986). Severe lead exposure in children (blood lead levels above 380 µg/dL) can cause coma, convulsions, and even death.

Clinical signs of lead toxicity, generally manifested as neurotoxicity and anaemia, are evident at blood lead levels of 70 µg/dL and greater (IARC, 2004). The most commonly reported and well-studied effects of environmental lead exposure are (1) adverse effects on neurological function and neurobehavioural development in children, and (2) reduced growth rate. However, it remains unclear if lead causes such effects in adults (U.S. EPA IRIS, 2004). The effects in children often manifest as decreased IQ and memory, decreased gestation period, and retarded growth rate. While the debate as to whether or not a threshold exists for the cognitive effects of lead in children continues, there is consistent information from the available lead health effects literature indicating that childhood blood lead levels >10 µg/dL are linked to decreased intelligence and impaired neurobehavioral development (WHO, 1995; CDC, 2009; Lanphear *et al*., 2005; U.S. EPA IRIS, 2004; ATSDR, 2007a).

Anaemia and altered blood enzyme levels or activity have been commonly correlated to elevated blood lead concentrations in the scientific literature. However, haematological effects are less sensitive endpoints for lead toxicity than neurological, neurodevelopmental, and neurobehavioural effects. The severity of anaemia resulting from lead exposure is linearly related to blood lead concentrations (Landrigan, 1989). Anaemia, resulting from reduced hemoglobin production and damage to erythrocytes, has been observed in adults with blood lead levels of 80 µg/dL and in children at blood lead levels of greater than 70 µg/dL (Goyer, 1989; U.S. EPA, 1986).

A number of epidemiology studies indicate an association between lead exposure and adverse renal effects. Some studies have found evidence of renal tubular dysfunction in children living in the vicinity of lead smelters (Bernard *et al.,* 1995; Verberk *et al.,* 1996). Renal tubular dysfunction is also seen in males with osteoporosis who have been occupationally-exposed to lead (Sun *et al*., 2008). Renal damage and osteoporosis was demonstrated by the stable relationship between concentrations of various enzymes serving as biomarkers that increased concomitantly with a decrease in bone density. Evidence of osteoporosis was initially associated with blood lead levels >8 µg/dL and an elevation in serum concentrations of alkaline phosphatase. Changes in other enzyme functions specifically associated with increased risk of osteoporosis and increased bone metabolism were associated with blood lead levels in excess of 10 µg/dL in the population (n=211) (Sun *et al.*, 2008).

Pre-natal exposure to low levels of lead includes effects such as reduced birth weight, reduced gestational age and neurobehavioral deficits, or delays. The evidence for an association between blood lead levels and reduced birth weight and gestational age is inconsistent, and therefore equivocal. Evidence in support of neurobehavioral deficits or delays has been much more consistent, with most studies indicating a positive association between low level lead exposure and developmental neurobehavioral effects (ATSDR, 2007a). It has also been suggested that bone resorption during pregnancy and lactation could result in increased blood lead levels and neonatal exposure during lactation (Ettinger *et al.*, 2006). However, a recent examination found that while there was a clear connection between bone loss and lactation, there was no evidence of a statistically significant association of bone loss with change in maternal blood lead concentration that would support a hypothesis of risk from lead to neonates from bone turnover (Sowers *et al.*, 2002). In fact, women who showed evidence of higher bone turnover had lower blood lead levels in breast milk (Sowers *et al.*, 2002).

There is currently scientific debate as to whether there is a causal relationship between blood lead and adverse cardiovascular outcomes such as hypertension (ATSDR, 2007a). Occupationally exposed populations with blood lead levels of between 30 to 120 µg/dL experienced hypertension, increased heart rate and increased blood pressure (Pollock and Ibels, 1986; Weiss *et al*., 1986, 1988; de Kort *et al*., 1987; Marino *et al*., 1989). However, these studies all had small cohort sizes (n<100) and failed to control for at least one confounding factor (ATSDR, 2007a). A number of other studies have failed to find a strong correlation between blood lead and cardiovascular effects (WHO, 1995; ATSDR, 2007a). Other studies that have investigated cardiovascular endpoints associated with lead (Jain *et al*., 2007) have been recently reviewed (Navas-Acien *et al*., 2007). Despite the uncertainties, these authors inferred a causal relationship between lead exposure and hypertension; however, the shape of the concentration-response relationship for lead and blood pressure is incompletely characterized. The lowest level of lead exposure not associated with alteration of blood pressure is unknown, although in the available studies there seems to be no evidence of a threshold effect (Hertz-Picciotto and Croft 1993; Schwartz *et al*., 2001; Navas-Acien *et al*., 2007).

Investigation of the association between bone lead levels and adverse cardiovascular outcomes has been examined in efforts to determine whether lead-associated elevations in blood pressure reflect acute responses to blood concentrations or rather chronic effects associated with cumulative dose (Glenn *et al.*, 2006). Contrasting results have been found. Glenn *et al.*, (2006) investigated changes in systolic blood pressure associated with lead in blood and bone in a lead-exposed occupation cohort (n=575) and found that while both cross-sectional and longitudinal blood lead variables predicted a relatively small increase in systolic blood pressure (1 mm Hg per 10 µg/dL increase in blood lead) associated with this acute pathway, average annual change in blood pressure was not predicted by tibia lead dose (the chronic accumulation of lead in the cortical bone). Navas-Acien *et al.*, (2008) however, concluded from a meta-analysis of data from eight studies examining the relation between lead detected in tibia and patella bone and changes in blood pressure, that systolic blood pressure and hypertension risk were associated with lead levels in tibia bone, although the magnitude of the summary estimates was small. The relative contribution of bone and blood lead levels was not examined, nor was the evaluation of non-linear dose-response relationships, as the study was conducted using published literature (Navas-Acien *et al.*, 2008).

Carcinogenicity

Currently Cal EPA is the only public regulatory agency known to have derived regulatory exposure limits for lead based on carcinogenic effects. The U.S. EPA Carcinogen Assessment Group has determined that an estimate of carcinogenic risk from oral exposure (such as a slope factor) using standard methods would not adequately describe the potential risk for lead compounds given the current lack of understanding on various toxicological and toxicokinetic characteristics of lead. Health Canada has not formally classified lead compounds with respect to their carcinogenic potential.

It is well established that the most sensitive endpoint for lead (and the greatest potential risk) is the development of adverse neurological, neurodevelopmental and neurobehavioural effects in young children. As a result, virtually all regulatory agencies around the world have focused on these health outcomes of lead exposure, and not on the potential carcinogenic risks. This regulatory focus also reflects the fact that the concern for neurological, neurodevelopmental, and neurobehavioural effects is associated with a less than lifetime exposure duration (*i.e*, toddlers, infants, young children), whereas carcinogenic effects are virtually always associated

with much longer exposure durations over a lifetime. In addition, it is important to recognize that the human carcinogenicity potential of lead compounds is largely inconclusive.

Toxicological Reference Doses (TRVs)

OMOE (2007; 2008) proposed an acute 24 hour AAQC of 0.5 µg/m^3 based on neurological effects in children. The U.S. EPA in 1978 developed an acute national ambient air quality standard (NAAQs) (3 months averaging time) of 1.5 µg/m^3 based on keeping 99.5% of children from exceeding a blood lead level of 40 µg/dL. However, as of October 15th, 2008, the U.S. EPA has revised the NAAQs to a value of 0.15 µg/m^3 to be protective of children and other at-risk populations. The U.S. EPA considered the available literature since the previous standard was released and has noted that there are no known safe levels of lead in the body and have seen that children may be greatly affected by this *via* neurological effects such as neurocognitive and neurobehavioral. Manatoba Conservation (2005) has recently adopted an air guideline value of 2 µg/m^3 for lead.

The OMOE (1994) recommended an intake of concern for populations (IOC_{pop}) of 1.85 µg/kg/day in order to minimize the predicted number of children with individual blood lead levels of concern. Subclinical neurobehavioural and developmental effects were the critical effects appearing at the lowest levels of exposure (OMOE, 1994). The intake of concern for individuals (IOC_{ind}) was based on a Lowest Observed Adverse Effect Level (LOAEL) in infants and young children of 10 µg/dL PbB divided by an intake/PbB slope factor of 0.21 µg Pb per DL PbB per µg/day. This resulted in an IOC_{ind} of 3.7 µg/kg/day for a 13 kg child (0.5 to 4 yrs). To derive the IOC_{pop} an uncertainty factor of 2 was applied to the IOC_{ind}, which resulted in a daily intake of 1.85 µg/kg/day (OMOE, 1994). This value is based on the same research as the other agencies limits (Ryu *et al*., 1983 and Ziegler *et al*., 1978). As it is based on an internal PbB concentration, this IOC_{pop} applies to lead exposure received from all sources, *via* all routes.

Health Canada (1992; 2004b; 2008) has selected a pTDI of 3.6 µg/kg-day which corresponds to a intake of lead by infants and children that was not associated with an increase in blood lead levels. This is consistent with the pTDI derived by JECFA (1987), based on the weight of evidence and the studies by Ryu *et al*. (1983) and Ziegler *et al*. (1978). The toxic effects of lead in humans are widely believed to be the same regardless of the route of entry, and are correlated to blood lead in the vast majority of studies. As a result, the Health Canada pTDI has been selected and is used to evaluate both oral and inhalation exposure pathways. This value corresponds well to the lead intake level that OMOE (1994) estimated would result in a blood lead level of 10 ug/dL.

In 1994, the Federal-Provincial Committee on Environmental and Occupational Health (CEOH, 1994) established a Lead Working Group to review the medical and scientific evidence on the health effects of lead. The stated objective of the Lead Working Group was to revisit the blood lead intervention level set in 1987. The recommendation of the CEOH 1994 report was to reduce the 1987 blood lead action level for intervention for individuals from 20 to 25 µg/dL to 10 µg/dL. Since the release of that report, the scientific and medical literature suggests that health effects may be occurring below blood lead levels of 10 µg/dL and Health Canada is currently reviewing this action level. CEOH (1994) also established community intervention levels as follows:

- Mean blood lead levels in children exceed the mean plus 3 standard deviations from the mean general population; or,

- when % of children with blood lead above 0.5 µmol/l (10 µg/dl) is double that of the general population'

Current regulatory TRVs for lead, including those derived by OMOE and Health Canada are generally protective of a blood lead level of 10 µg lead/dL blood. 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; Bellinger, 2008). Many regulatory agencies consider there to be no known safe levels of lead in the body. 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. (CDC, 2009) and NHMRC in Australia (NHMRC, 2009a,b), 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. Therefore, major health agencies (*e.g.*, Health Canada, Centre of Disease Control, NHMRC and the U.S. EPA) continue to use the 10 µg/dL limit, despite the new and emerging science which is indicating effects may be occurring at lower levels.

4.2.3.5 Mercury

Mercury occurs naturally in the environment in rocks and soil as mercuric sulphide, however, anthropogenic activity accounts for approximately 33 to 66% of total mercury releases (ATSDR, 1999a; RIVM, 2001). Mercury occurs in three oxidation states in nature: metallic/elemental (Hg^0), mercurous (Hg^+), and mercuric (Hg^{2+}). Generally, mercuric cations can form various organic and inorganic mercury compounds (ATSDR, 1999a).

Elemental mercury is mined and refined from its natural form. Inorganic forms of mercury, also known as mercury salts, are the result of the combination of elemental mercury with other elements (*e.g.,* chlorine, sulphur, oxygen). The mixture of mercury and carbon produces various forms of organic mercury including methylmercury, dimethylmercury, and phenyl mercury (ATSDR, 1999a). Methylmercury is primarily produced by microorganisms, which can convert inorganic mercury to an organic form. In the past, organic forms of mercury were used as fungicides, but these uses are presently banned in North America and Europe due to evidence of adverse human health effects. Today, dimethylmercury is used in the preparation of resonance and mass spectrometry standards (ATSDR, 1999a). Organic forms of mercury are generally volatile, and solubility differs greatly for the different forms (WHO, 1990). Methylmercury is soluble and highly bioaccumulative (ATSDR, 1999a). Research has shown that methylmercury will biomagnify in aquatic food chains, and has been identified as the predominant form of mercury in fish.

Mercury is predominantly used in chemical and mining applications, such as in the extraction of gold, and in the production of polymers, chlorine gas, and caustic soda. Electrical applications include use in batteries, wires and switches, and thermostats, though these uses are being phased out. Mercury is used for medical applications, such as dental amalgams and

thermometers. Phenyl mercuric acetate is used in ink and adhesive preparations, and as a fungicide in paints (ATSDR, 1999a). Mercury salts have been used in skin-lightening creams and medications (*e.g.,* laxatives, teething powders), but have since been replaced with safer compounds (OEHHA, 1999).

Mercury is released into the atmosphere from mining and smelting, fossil fuel combustion, and waste incineration. Releases into aquatic and terrestrial ecosystems result from municipal waste (*e.g.,* used batteries), fertilizers, fungicides, and industrial wastewater (WHO, 1991). Average soil concentrations of mercury in areas of relatively minimal anthropogenic activity range from 0.01 to 0.4 mg/kg (Jonasson and Boyle, 1972; Gracey and Stewart, 1974; McKeague and Kloosterman, 1974; Environment Canada, 1979; OMEE, 1994). Frank *et al*. (1976) found that in agricultural soils, concentrations were more elevated ranging from 0.01 to 1.14 mg/kg. In Canadian lakes located near smelters, surface sediments ranged from 0.03 to 9.22 µg/g. Concentrations were greatest near the smelter, however, it was found that mercury concentrations in fish could not be correlated with the mercury content in sediments (Harrison and Klaverkamp, 1990).

Dental amalgams are the largest non-occupational source of metallic mercury vapour to humans (*via* inhalation and ingestion); however, the risk is deemed minimal due to the small amounts of mercury present in amalgams (ATSDR, 1999a). Inhalation of metallic mercury vapour from the workplace is an additional route of human exposure. The major sources of exposure to inorganic mercury (mercuric chloride) are dermal (skin-lightening creams, soaps) and oral (medicines, such as calomel) (WHO, 1991). Human dietary intake is the most important route of exposure for methylmercury (Health Canada, 2007a).

Fate and Transport

Approximately 95% of the total mercury found in the atmosphere is in the form of elemental/metallic mercury vapor (Hg^0) which has a high vapor pressure and low water solubility (ATSDR, 1999a). Its chemical properties allow it to have a long residence time in the atmosphere, therefore, allowing for wide range transport (U.S. EPA, 1997c; ATSDR, 1999a). This form of mercury is subject to oxidation/reduction reactions which will convert it into an inorganic form; therefore allowing for it to be deposited into other environmental media.

Inorganic mercury exists in both aquatic and terrestrial ecosystems (ATSDR, 1999a; CCME, 1999b). In soil and sediments inorganic mercury has limited mobility since it readily adsorbs onto organic matter in soil and sediments; therefore not readily leaching into ground or surface waters (CCME, 1999b; OEHHA, 1999). Since inorganic mercury does not easily desorb from soil or sediment, it enters aquatic systems through runoff. Inorganic forms of mercury in both terrestrial and aquatic systems are naturally transformed to organic methylmercury by aerobic and anaerobic bacteria *via* the methylation process (ATSDR, 1999a). Similarly, some bacteria can demethylate methylmercury into metallic mercury which readily volatilizes into the environment (ATSDR, 1999a). Methylmercury is mobile and bioavailable, therefore binding tighly to proteins in fish tissue, resulting in bioaccumulation in food chains (Health Canada, 2007a). Carnivorous fish have been found to biogmagnify to levels that are 10,000 to 100,000 times of that in the water (OEHHA, 1999). Most plants grown in areas with elevated mercury in soil do not accumulate the chemical, although studies have show that edible mushrooms accumulate mercury to a great extent (ATSDR, 1999a). Inorganic mercury bioaccumulates to a much lesser extent (Health Canada, 2007a). Bioavailability of mercury in aquatic systems can decrease with the presence of selenium which forms complexes with inorganic and organic

forms of mercury (ATSDR, 1999a; WHO, 1991). Other organic forms of mercury, such as dimethylmercury, are rarely found in the environment.

Mercury Toxicokinetics

Absorption of metallic mercury after inhalation is quite high since it is highly lipophillic and can rapidly diffuse across alveolar membranes of the lungs into the blood (Hursh *et al*., 1976; Teisinger and Fiserova-Bergerova, 1965). No studies could be found indicating absorption of mercury after inhalation to phenyl or methyl mercury, although there is indirect evidence that organic mercury can be absorbed through the lungs (Ostlund, 1969). Similarly, few studies could be found regarding the absorption of metallic or inorganic mercury after oral exposure (ATSDR, 1999a). Some studies have shown that absorption is negligible (Sue, 1994; Wright *et al*., 1980). Conversely, organic mercury is much more readily absorbed, up to 95% as one study showed (Aberg *et al*., 1969). Other studies have also shown absorption to be quite high, although the amount absorbed was not expressed quantitatively (Al-Shahristani *et al*., 1976; Mieltinen, 1973). Absorption following dermal exposure to mercury vapour is minimal; however, applications of ointments containing inorganic mercury have shown to have considerable absorption (Bourgeois *et al*., 1986; DeBont *et al*., 1986; Hursh *et al*., 1989). Minimal information regarding absorption after dermal exposure to organic mercury could be found however, dermal exposure to dialkylmercurials has been shown to have high absorption (Blayney *et al*., 1997; Nirenberg *et al*., 1988).

Since metallic mercury is quite lipophillic it is readily distributed throughout the body and can cross blood-brain and placental barriers after inhalation (Clarkson, 1989; Hursh *et al*., 1976). In the blood, metallic mercury is oxidized to the divalent form which can exist in both diffusible or non diffusible forms, however, the non diffusible form is predominant and binds to proteins such as albumin and globulin (Berlin and Gibson, 1963; Cember *et al*., 1968; Clarkson *et al*., 1961). As well, the kidney is the major organ for mercury deposition since it contains metallothionein which is stimulated by mercury (Cherian and Clarkson, 1976; Piotrowski *et al*., 1973; Rothstein and Hayes, 1964). Distribution of organic mercury is similar to that of metallic or inorganic mercury (ATSDR, 1999a). Metabolism of mercury involves an oxidation-reduction cycle, whereby the mercury is oxidized to the divalent inorganic cation in the red blood cells and lungs. It can be reduced to the metallic or monovalent form and released as exhaled metallic mercury vapour (ATSDR, 1999a). Ultimately, mercury can be excreted *via* the urine, feces or expired air as well it can be excreted through breast milk (ATSDR, 1999a).

Biomonitoring

Human blood, hair, breast milk, and urine are generally used in measuring mercury exposure (ATSDR, 1999a). In the blood, mercury has a short half-life thereby making it a useful medium for determining short-term exposures (WHO, 2003). However, long-term consumption of fish containing elevated methylmercury levels can be determined through blood analysis. Mercury analysis of hair is useful in that it reveals dietary exposure to methylmercury (WHO, 1990). Mercury vapours also accumulate in hair, however, this has not been regarded as a suitable indicator for inorganic mercury exposure (WHO, 1991). Urine samples are most appropriately used to indicate long-term, low level exposure to inorganic forms of mercury (ATSDR, 1999a; WHO, 2003; Yoshida, 1985). Mercury concentrations in the blood or urine do not reflect body burden concentrations of mercury (WHO, 1991).

Mercury Health Effects

Elemental/Metallic Mercury

Acute Effects (Inhalation)

Following acute inhalation of metallic mercury vapours, reports have indicated cases of effects on the gastrointestinal, cardiovascular, hemotological, renal and endocrine systems. In addition, effects on the central nervous system have also been reported (McFarland and Reigel, 1978; ATSDR, 1999a; Cal EPA, 1999c). Dermal effects such as skin rashes have also been reported in numerous studies following acute inhalation of mercury vapours (Nakayama *et al*., 1983; ATSDR, 1999a).

Chronic Effects (Inhalation)

Both the central nervous system (Fawer *et al.,* 1983; Piikivi, 1989; Piikivi and Hanninen, 1989; Piikivi and Tolonen, 1989; Ngim *et al*., 1992; Liang *et al*., 1993) and the kidney (Kazantzis *et al.,* 1962; Bernard *et al.,* 1987; Barregard *et al.,* 1988; Piikivi and Ruokonen, 1989; Cardenas *et al*., 1993) are sensitive target areas for metallic mercury vapours. Other studies have documented increased heart and blood pressure with inhalation exposure (Tubbs *et al.,* 1982; Barregard *et al.,* 1990; Fagala and Wigg, 1992; Taueg *et al.,* 1992). In addition, gastrointestinal (Smith *et al*., 1970; Vroom and Greer, 1972; Buckell *et al*., 1993) and endocrine effects have also been reported (Barregard *et al.,* 1994).

Inorganic Mercury

Limited information is available pertaining to the chronic effects inorganic mercury in humans (WHO, 1991; ATSDR, 1999a; OEHHA, 1999; RIVM, 2001). In general, the kidney is a major target organ of chronic inorganic mercury exposure, and autoimmune glomerulonephritis has been documented as has renal failure (Davis *et al*., 1974; U.S. EPA, 1992b; OEHHA, 1999). Dermal effects including rash, swelling, itching and excessive perspiration as well as thirst, increased blood pressure and tachycardia have been reported in children from subchronic exposure to mercurous chloride in medications (tablets, powders) (Warkany and Hubbard, 1953).

Organic Mercury

Chronic Effects (Oral)

Several epidemiological studies of neurotoxicity caused by chronic foetal exposure provide the basis for chronic oral exposure levels for methylmercury for agencies such as ATSDR (1999a), U.S. EPA IRIS (2001), RIVM (2001), WHO (2004), and Health Canada (2007b). Approximately 95% of the methylmercury ingested is absorbed, and peak methylmercury levels in the blood are reached within 6 hours. Methylmercury is readily able to cross plasma membranes as well as the blood-brain barrier and the placenta as animal studies have shown since the blood-brain barrier in the foetus is not fully developed. Once in the brain, methylmercury accumulates and is slowly converted to inorganic mercury (Maycock and Benford, 2007). Developmental neurotoxicity is considered to be the most sensitive chronic endpoint of methylmercury (ATSDR, 1999a). There is clear evidence from the concentrations of mercury in human milk and in the blood of infants that, compared with exposure in *utero*, postnatal exposure to methylmercury is considerably lower in infants who are breastfed

(JECFA, 2007). Postnatally, both neuronal myelination and remodelling of the cortex of the brain occur and have a protracted time course, continuing through adolescence until about 17 years of age (Rice and Barone, 2000; JECFA, 2007).

Davidson *et al.* (1998) tested neurodevelopmental capabilities in children (5.5 years old) that were exposed *in utero* (maternal fish ingestion) and through breast milk in 779 mother-infant pairs in fish-eating populations in the Republic of Seychelles. Mercury exposure was determined from maternal and child hair for which determinations of mean concentrations were 6.9 and 6.5 ppm, respectively. Fish in the maternal diet were contaminated with methylmercury and fish consumption was comparable to that in the U.S. In addition, the location of the Seychelles was considered pristine, suggesting the health of the population was not influenced by other contaminants. A NOAEL (1.3 µg/kg-day) was established for developmental effects in this study. Similar epidemiological studies by Myers *et al.* (1997) and Crump *et al.* (1998) reported NOAELs (0.5 and 0.62 µg/kg-day, respectively) that indicated effects were observed at lower levels of contamination. Grandjean *et al.* (1997) reported neurotoxicity associated with *in utero* exposure in children at 7 years of age in a study of 900 mother-infant pairs in the Faroe Islands. Tests showed mercury-related abnormalities with neuropsychological indicators such as language, attention, and memory. The mean cord blood concentration of mercury was 22.8 µg/L, and the average concentration of methylmercury found in children's hair was 11.68 ppm. In Northern New Zealand, dose-related effects for psychological endpoints occurred in 6 year olds resulting from pre-natal exposure (Kjellstrom *et al.,* 1989). Maternal hair mercury levels of 13 ppm (during pregnancy) were associated with the effects in this study population. Data from these studies were used to derive benchmarks for chronic oral exposure limits (U.S. EPA IRIS, 2001; WHO, 2004). No studies in regards to reproductive effects of chronic exposure to mercury in humans were located (ATSDR, 1999a; Cal EPA, 2005b).

Carcinogenicity

Metallic mercury has been determined to not be classifiable as a human carcinogen (Cal EPA, 2006; IARC, 2007; U.S. EPA IRIS, 1995a; 2001). Inorganic mercury has also been deemed to not be classifiable as a human carcinogen (Cal EPA, 2006; IARC, 2007). One form of inorganic mercury (mercuric chloride) was classified as a possible human carcinogen (U.S. EPA IRIS, 2001). Methylmercury has been classified as a carcinogen (Cal EPA, 2006) or possible carcinogen (U.S. EPA IRIS, 2001; IARC, 2007)

Toxicological Reference Doses (TRVs*)*

Elemental/Metallic Mercury

U.S. EPA IRIS (1995a) derived a chronic inhalation RfC of 0.3 µg/m^3 for elemental mercury vapour based on neurobehavioral effects (hand tremors, increases in memory disturbance, and slight subjective and objective evidence of autonomic dysfunction) in occupationally-exposed subjects (Fawer *et al.,* 1983; Piikivi and Tolonen, 1989; Piikivi and Hanninen, 1989; Piikivi, 1989; Ngim *et al.,* 1992; Liang *et al.,* 1993). U.S. EPA IRIS (1995a) assigned a medium level of confidence in the RfC (0.3 µg/m^3). This value was adopted as the inhalation chronic exposure limit for elemental mercury in the current assessment.

Health Canada (2008) has selected a provisional tolerable concentration (TC) value of 0.06 µg/m^3 for chronic inhalation of elemental mercury vapour. This value is based on an epidemiology study by Ngim *et al*. (1992) of an occupational cohort who was exposed to mercury vapours over an average duration of 5.5 years, whereby, neurobehavioral effects were

noted. This value was chosen as the chronic inhalation exposure limit for elemental mercury in the current assessment.

Inorganic Mercury

Cal EPA (1999c) derived an acute inhalation REL of 1.8 µg/m^3 for a 1 hour exposure time based on behavioural deficits in rats exposed to metallic mercury vapour *in utero* (Danielsson *et al.,* 1993). OMOE (2008) lists an AAQC (24 hour average) of 2 µg/m^3 for elemental mercury. This value was adopted as the 24 hour acute exposure limit for the current assessment. An annual average guideline of 1 µg/m^3 was established by WHO (2000) for objective tremors, renal tubular effects (changes is plasma enzymes) and non-specific symptoms in workers subjected to long term mercury vapour exposure (WHO, 1991; Cardenas *et al.,* 1993). This value was adopted as the chronic inhalation exposure limit for inorganic mercury for the current assessment. ATSDR (1999) have established a short-term MRL for inorganic mercury of 7.0 ug/kg-day based on Renal effects (increased absolute and relative kidney weights, increased incidence and severity of tubular necrosis). This value has been selected as the short-term oral TRV for the current assessment.

A TDI of 0.3 µg/kg-day for the chronic oral intake of inorganic (ionic) mercury has been derived by Health Canada (2004b). This value was back-calculated from a drinking water equivalent level (DWEL) of 10 µg/L and adjusted for daily water consumption and body weight (RfD = 10 µg/L x 2L/day/kg bw). The U.S. EPA identified an oral RfD for mercuric chloride of 0.3 µg/kg-day (U.S. EPA, IRIS, 1995b). The Health Canada (2004b) TDI was adopted as the chronic oral exposure limit for inorganic mercury for the current assessment.

Organic Mercury

The U.S. EPA IRIS (2001) has recommended an oral RfD of 0.1 µg/kg/day for methyl mercury. This value is based on benchmark daily intake dose of 1 µg/kg/day found in human epidemiological studies by Grandjean *et al*. (1997) and Budtz-J∅rgensen *et al*. (1999). Due to the variability of the studies, the U.S. EPA IRIS (2001) calculated the RfD of 0.1 µg/kg/day by applying an uncertainty factor of 10 (3 for pharmacokinetic variability and uncertainty in dose estimation and 3 for pharmacodynamic variability and uncertainty) to the benchmark dose.

WHO (2004) derived a pTWI of 1.6 µg/kg/week for methylmercury based on the association between maternal exposure and developmental effects in children (Grandjean *et al*., 1997; Davidson *et al.,* 1998; Budtz-Jorgensen *et al.*, 1999). A steady state of 1.5 µg/kg/day was estimated to represent the level of exposure not expected to have any appreciable adverse effects on children. An uncertainty factor of 2 was applied to the TDI to account for interindividual variation of the maternal hair:blood. In addition, a combined uncertainty factor of 3.2 was applied to account for total human interindividual variation for dose reconstruction in converting maternal blood concentration to a steady-state TDI (WHO, 1990). Therefore, an uncertainty factor of 6.4 (2x3.2) was applied to the calculated TDI of 1.5 µg/kg/day to result in a TDI of 0.23 µg/kg/day, which equates to a pTWI of 1.6 µg/kg/week (1.5 µg/kg/day x 7 days/week ÷ 6.4). The calculated pTWI is for the protection of developing fetuses, which are the most sensitive subpopulation. This revised pTWI was considered protective of all effects, including neurodevelopmental effects (Maycock and Benford, 2007).

Health Canada has determined a TDI of 0.47 ug/kg-day for the general adult population and a TDI of 0.2 ug/kg-day for women of childbearing age and children <12 years for lifetime exposure to methylmercury. The general population TDI was based on the World Health Organization

(WHO, 2004) provisional tolerable daily intake (pTDI) for total mercury in adults (based on a 60 kg body weight) of 0.71 µg per kg body weight per day. Two-thirds, or 0.47 µg/kg bw/day, was allocated to methylmercury based on the average ratio of total mercury to methylmercury in food. The Bureau of Chemical Safety (Health Canada) concurred with this assessment and has employed the 0.47 µg/kg bw/day value for the general population (Health Canada, 2007a; 2008). This value was based on epidemiological studies including: epidemic accidental poisoning and chronic low-level exposure in populations with a high consumption of fish.

In 1997, the Bureau of Chemical Safety derived a pTDI for methylmercury of 0.20 µg/kg bw/day for women of child-bearing age and young children (<12 years) (Health Canada, 2007a; 2008). This value was derived in recognition that developing fetuses and young children have increased susceptibility to the effects of methylmercury, based on epidemiological prospective studies on neurodevelopmental effects. The pTDI was derived based on a 10 ppm maternal hair methylmercury level as the approximate threshold (Grandjean *et al*., 1997) for neuropsychological dysfunctions. This value was first converted to a corresponding blood methylmercury level and then to a dietary methylmercury intake level using an equation used by U.S. EPA IRIS (2001). A 5-fold uncertainty factor was applied to this intake level to obtain a pTDI for methylmercury, protective of women of child-bearing age and young children (<12 years) of 0.20 µg/kg bw/day (Feeley and Lo, 1998).

The Health Canada pTDI for women of child-bearing age and young children (<12 years) was recently re-affirmed and is consistent with the more recently derived WHO pTWI. Both values are intended to be protective of the most sensitive subpopulations and endpoints, including neurodevelopmental effects. This value is considered defensible and most appropriate for use in this assessment and has been selected.

4.2.3.6 Selenium

Selenium is an essential micronutrient required by humans. It is a naturally occurring element which is widely but unevenly distributed within the earth's crust (ATSDR, 2003). Selenium can exist in a variety of chemical forms in the environment including various salts, oxides, hydrides, sulphides, and metal selenides. In the ambient environment, selenate and selenite are the primary forms of selenium. Selenium compounds are used in the glass industry as decolourizing agents and in the rubber industry as vulcanizing agents (Cal EPA, 2001). Selenium-containing compounds can also be found in photographic toning baths, insecticides and photoelectric cells (Cal EPA, 2001). The most widely used selenium compound in industry is selenium dioxide which catalyzes reactions of organic compounds (Cal EPA, 2001). The greatest anthropogenic sources of selenium in the ambient environment are from the combustion of fossil fuels and the production and refinement of copper (Cal EPA, 2001).

Exposure to low levels of selenium occurs on a daily basis *via* food, water, and air (ATSDR, 2003). However, typically, the greatest percentage of exposure to selenium comes from the dietary ingestion of organic (selenomethionine and selencysteine) and inorganic (selenate and selenite) forms of selenium, although, the organic forms are more readily absorbed as compared to the inorganic forms (ATSDR, 2003). In the U.S., dietary intake of selenium is approximately 1 to 2 µg/kg/day but can be greater depending on the area. In foods, meat products have the highest levels of selenium, whereas fruits and vegetables have the lowest levels (ATSDR, 2003). Brazil nuts have elevated selenium levels since they are grown in soils containing high levels of selenium (Secor and Lisk, 1989). Worldwide, selenium concentrations in the soil are on average between 0.05 to 0.09 mg/kg and in the U.S. most soils have concentrations are between 0.01 to 0.2 mg/kg (Sindeeva, 1964). In seleniferous soils,

concentrations in the U.S. range from <2 to <100 mg/kg (Rosenfeld and Beath, 1964). Atmospheric depositions of selenium from mining and smelting operations will also contribute to these levels (Glooschenko and Arafat, 1988). Normally, humans are not exposed to large amounts of selenium in the air, unless selenium dust or volatile selenium compounds are formed in the workplace (ATSDR, 2003).

Fate and Transport

Selenium in air exists in the forms of selenium dioxide, hydrogen selenide, dimethyl selenide and dimethyl diselenide. Elemental selenium can also be formed by the rapid oxidation of hydrogen selenide and the reduction of selenium dioxide released to the air from fossil fuel combustion (ATSDR, 2003). These airborne compounds may be removed by wet or dry deposition to soil or surface water. In water, the predominant selenium species are the salts of selenic (selenates) and selenious (selenites) acids (CCME, 2007). Because of its high solubility and inability to adsorb to soil particles, sodium selenate is one of the more mobile selenium compounds, which are more available for uptake by biological systems (ATSDR, 2003). Selenium is bioaccumulated by aquatic organisms such as algae, insect larvae, molluscs, and crustaceans (ATSDR, 2003; CCME, 2007).

In soils, natural levels of selenium are mostly the product of weathering and leaching, with some input from wet and dry deposition from the atmosphere (CCME, 2007). The pH and pE in soils are important factors in the transport of selenium. In soils of low pH and strong reducing conditions (high organic content), metal selenides, selenium sulfides and selenites predominate (CCME, 2007). In neutral conditions, selenites are the more common species (ATSDR, 2003), but can form insoluble complexes with iron oxides/hydroxides and clays (CCME, 2007). Insoluble elemental selenium is formed under reducing conditions, but its bioavailability is low due to its low solubility (CCME, 2007). Terrestrial plants that take up soluble selenates and selenites convert these species into organic selenium compounds, which are then released to the soil when the plants die (ATSDR, 2003). In reducing soil conditions, elemental selenium is formed, which (along with inorganic selenium compounds) can be methylated by microorganisms and then volatilized into the atmosphere (CCME, 2007).

Selenium Toxicokinetics

Absorption of selenium *via* inhalation has shown inconclusive results in occupational studies (Sanchez-Ocampo *et al*., 1996; Glover, 1970). When ingested orally, selenium is readily absorbed from the gastrointestinal tract. Both sodium selenite and selenomethionine exceeded 80% absorption after consumption of both small and large doses (Thomson, 1974; Thomson and Stewart, 1974; Griffiths *et al*., 1976; Thomson *et al*., 1977). Other studies have demonstrated that the extent of adsorption is dependent on the chemical form of selenium; particularly selenomethionine was absorbed more efficiently in the body than sodium selenite (Robinson *et al*., 1978; Swanson *et al*., 1991; Moser-Veillon *et al*., 1992). Those with selenium deficient diets did not show evidence of increased absorption (Martin *et al*., 1989). No evidence could be found correlating selenium absorption due to dermal exposure (Cummins and Kimura, 1971; Burke *et al*., 1992; Kalivas, 1993).

After oral absorption of selenium, it is distributed into plasma proteins including selenoprotein-P, gluthathione peroxidases and albumin (Ducros *et al*., 2000). Since 3% of the plasma selenium is bound to lipoproteins, then selenomethionine can replace methionine during protein synthesis and/or be bound to cysteine residues by Se-S bonds (ATSDR, 2004). As well, selenoprotein-P is an extracellular protein in the plasma, therefore it is involved in the transport of selenium and

as an antioxidant (Hill and Burk, 1989; Yang *et al*., 1989a; Burk and Hill, 2000). Generally, selenium from selenomethionine is in greater concentrations in tissues and for a greater period of time as compared to inorganic selenium because of slower elimination rates as a result of its incorporation into body proteins (Stadtman, 1983; 1987; 1990; Ip and Hayes, 1989; Butler *et al*., 1990; Gronbaek and Thorlacius-Ussig, 1992; Salbe and Levander, 1990;).

In brief, inorganic selenium is reduced to hydrogen selenide which is incorporated into selenoproteins in the form of covalent carbon-selenium bonds after transformation to selenophosphate and selenocysteinyl tRNA or excreted from the body after transformation to the methylated metabolites of selenide (Holmgren and Kumar, 1989; Lobinski *et al*., 2000). Selenomethionine, is also metabolized, but if not immediately, it can be stored in the skeletal system, muscle, liver, stomach, gastrointestinal muscosa and erythrocytes until it is metabolized (Schrauzer, 2000). Excretion and elimination of selenium from the body may be dose dependent and occurs mainly *via* the urine, feces and expired air; minor amounts may also be excreted *via* released sweat (McConnell and Roth, 1966; Lathrop *et al*., 1972; Thomson and Stewart, 1974; Griffiths *et al*., 1976; Levander *et al*., 1987; Hawkes *et al*., 1992; 1994; ATSDR, 2003). The proportion of selenium released by each is dependent on the level and time since exposure and the level of exercise by the individual (Olson *et al*., 1963; McConnell and Roth, 1966; ATSDR, 2003).

Biomonitoring

Human blood, urine, toenails, fingernails, breast milk, semen, and various body tissues are commonly analyzed for selenium levels as indicators of selenium exposure (Yang, 1989b; Roy, 1990; Longnecker *et al.*, 1991; Brätter *et al.*, 1996). Other indicators of exposure which are being researched include measurement of selenium concentration in umbilical blood and fetal tissues, as well as the post-mortem analysis of livers, lungs, and spleens of infants (ATSDR, 2003).

Selenium Health Effects

The main organ affected by acute inhalation exposure to selenium dusts or fumes is the lung (ATSDR, 2003). Cardiovascular, hepatic, nervous, and renal effects can also be observed upon acute selenium exposure *via* the inhalation route (ATSDR, 2003). Workers exposed to high concentrations of elemental selenium dust have reported stomach pain and headaches, whereas workers exposed to high levels of selenium dioxide dust experienced a variety of respiratory symptoms including pulmonary oedema, bronchial spasms, symptoms of asphyxiation and persistent bronchitis (ATSDR, 2003). Chronic exposure to selenium dioxide or elemental selenium present in dust, resulted in irritation of the nose, respiratory tract, and lungs which led to bronchial spasms, and coughing (ATSDR, 2003).
Signs of toxicity in humans orally exposed to elevated levels of selenium in foods and soils include: loss of hair, thickened or brittle nails, skin lesions, tooth decay, irregularities of the nervous system, garlicky breath, reduced haemoglobin, and altered thyroid hormone levels in blood (Yang *et al*., 1983; Yang *et al*., 1989a,b; Contempre *et al*.,1991; Longnecker *et al*., 1991; Brätter *et al*., 1996; Hagmar *et al*., 1998; Duffield *et al*., 1999). Dietary levels (high, unspecified) of selenium compounds have been reported to cause gastrointestinal disturbances in chronically exposed humans as well (Smith *et al*., 1936). Dilation of the stomach, small intestine (Carter, 1966) and erosive changes in the gastrointestinal tract are some of the effects that have been documented after ingestion of selenium compounds (Koppel *et al*., 1986). The reported gastrointestinal disturbances may not to be specific to selenium intoxication (ATSDR,

2003). Chronic selenium intake also causes haematological effects such as decreased blood clotting as determined by increased prothrombin time (Yang *et al*., 1989a).

Selenium intake or use over a long period also contributes to enhanced immune functions (Peretz *et al*., 1991; Kiremidjian-Schumacher *et al*., 1992; Baum *et al*., 1997). Reports have described enhanced lymphocyte response in the elderly after taking a selenium-enriched yeast supplement (0.0014 mg/kg/day for six months) (ATSDR, 2003). Significant changes were also observed in phagocytosis, chemotactic factor generation, and antibody or leukocyte migration inhibitory factor production by lymphocytes. Low levels of selenium intake were also observed to enhance proliferative responses to the T-cell mitogens phytohemagglutinin or concanavalin A (ATSDR, 2003). Adverse neurological effects can also be caused by chronic selenosis (ATSDR, 2003). Chronic oral selenium intake at 4.99 mg/day was reported to cause peripheral anesthesia, pain of the limbs, exaggerated tendon reflexes, convulsions, paralysis and hemiplegia (Yang *et al*., 1983).

Carcinogenicity

The IARC (1987) and U.S. EPA IRIS (1991) have currently evaluated the carcinogenicity of selenium compounds as not classifiable as to human carcinogenicity. An exception is selenium sulphide, which the U.S. EPA IRIS (1991) has classified as a probable human carcinogen. The Eleventh Annual Report on Carcinogens (ROC) classed selenium sulphide as reasonably anticipated to be a human carcinogen (NTP, 2005c). For the purposes of this risk assessment selenium was assessed as a non-carcinogen.

Toxicological Reference Values (TRVs)

The OMOE (2008) derived an AAQC (24 hour averaging time) of 10 µg/m^3 for selenium which was selected as the 24 hour acute exposure limit for selenium. The basis of this value was not provided. Cal EPA (2001) utilized the U.S. EPA IRIS (1991) RfD to derive a chronic REL of 20 µg/m^3 for selenium using a body weight of 70 kg and an inhalation rate of 20 m^3/day. The specific study used to derive the RfD is described below. This value was selected to evaluate human health effects of long-term selenium exposure *via* inhalation.

Selenium was retained for evaluation in the multi-pathway exposure model due to its persistence; therefore, an oral RfD was required. An oral RfD of 5.0 µg/kg-day was established by the U.S. EPA IRIS (1991) based on clinical selenosis in human epidemiological studies (Yang *et al*., 1989a,b). An uncertainty factor of 3 for intraspecies variation was applied to the study NOAEL of 15 µg/kg-day. A full uncertainty factor of 10 for intraspecies variation was not considered necessary as similar NOAELs were identified in two additional moderately-sized human populations exposed to selenium levels in excess of the RDA throughout a lifetime without apparent clinical signs of selenosis (Yang and Zhou, 1994). A chronic oral exposure limit of 50 µg/kg-day was also derived by Cal EPA (2001) and ATSDR (2003) using similar epidemiological studies. IOM (2000) derived a set of tolerable upper intake levels (UL) for adults based on the study by Yang and Zhou (1994) which was based on a human epidemiological diet study. A NOAEL of 800 µg/day was calculated for adults and this value was divided by the uncertainty factor to calculate upper intake levels. Similarly, IOM calculated UL for infants and children based on a diet study by Shearer and Hadjimarkos (1975) and calculated a NOAEL of 7 µg/kg-day for infants and children. The UL values adopted by Health Canada are equal to those of IOM but have been adjusted for the duration of the life stage and body weight. These values were selected as the chronic exposure limit for the current assessment.

4.2.4 Bioavailability/Bioaccessibility

One of the most important factors in determining exposure of target tissues to a substance, and the body's ultimate response, is bioavailability. Bioavailability is the fraction of the total amount of a substance to which an organism has been exposed that successfully enters the blood stream. The bioavailability of a substance is dependent on the chemical form, the environmental medium, the route of exposure, physiological characteristics of the organism at time of exposure (*e.g*., ingested substances may be absorbed to different extents depending on whether the stomach is full or empty) as well as the tissues/organs with which the substance must interact as it passes from the point of entry to target tissues.

When applying exposure limits, it is necessary to consider the bioavailability of each substance in the particular study from which the exposure limit is derived, to obtain reasonable estimates of the quantity of the chemical entering the body of study animals or subjects. This allows for the normalization of exposures with respect to exposure route, and comparison of the bioavailable doses to humans with the exposure limits determined from animal studies or human epidemiological data. It is inappropriate to convert exposure estimates to absorbed doses if toxicity values are based on administered doses. However, if an exposure estimate is adjusted for bioavailability then it must be compared to an exposure limit which is based on an absorbed, rather than an administered dose. Otherwise, the estimation of potential impacts would be incorrect and may underestimate exposure and risk depending on the particular circumstances. Since most exposure limits are based on administered doses, it is not appropriate to consider absolute bioavailability (fraction or percentage of an external dose which reaches the systemic circulation) in the assessment of exposures in most instances. A better measure may be that of relative bioavailability which can be determined by comparing the extent of absorption among several routes of exposure, forms of the same substance, or vehicles of administration (such as food, soil, and water). Systemic absorption of substances will differ according to whether the dose was received *via* dermal contact, ingestion or by inhalation. Also, systemic absorption will differ depending on whether the substance is delivered in a solvent vehicle (water, soil, food, *etc*.).

As discussed previously, for some substances, exposure limits are not available for all exposure routes of concern. In cases when (1) an exposure limit is available for some exposure routes but not for the exposure route of concern; and, (2) no other data (such as pharmacokinetics) are available, it may be necessary to extrapolate an exposure limit from one route to another. For example, it is common in human health risk assessment to assess the risks posed by dermal absorption of a substance based on the exposure limit established for oral exposure. The systemic dose absorbed dermally is scaled to the "equivalent" oral dose by correcting for the bioavailability of the dermally-applied chemical relative to an orally-administered dose.
The oral bioavailability of a substance is typically determined from absorption or excretion studies. The bioavailability, expressed as a percentage, is generally assumed to be 100% minus the percent of the ingested chemical excreted unchanged in the feces. In cases where only the fraction of chemical in the urine is reported, this fraction is selected as the minimum oral bioavailability with the maximum being 100%. In the absence of relevant data, this approach is considered to be reasonable, and to reflect the uncertainty in the oral bioavailability of the chemical.

The relative absorption difference between the oral and dermal routes of exposure can be expressed as a relative absorption factor (RAFdermal), which has been described previously.

Typically, adjustments of exposure limits for bioavailability are considered for systemic effects (*i.e*., following entry into and distribution by the bloodstream, as opposed to occurring at the site of entry [*e.g*., lungs, skin, gut]) when:

- The exposure limit is based on a different route of exposure (*i.e*., when the criterion is based on ingestion and the exposure routes of interest are inhalation or dermal exposure);
- The medium of administration in the study used to develop the exposure limit results in a different bioavailability than the exposure medium of interest (*e.g*., ingestion in drinking water *versus* ingestion in soil); or,
- If the bioavailability of the chemical, based on the particular study animal/receptor, is different from that of the receptor upon which the exposure limit is based (*e.g*., the exposure limit is based on a study using mice, the species of interest is human, and there are reported bioavailabilities for both mice and humans).

In these cases, adjustment for bioavailability may be important in determining appropriate toxicological criteria for use in comparing to route-specific exposures, as well as ensuring that comparisons are made either for internal ("bioavailability-adjusted") doses and limits relevant to the species or population being assessed, or route-specific doses and limits. It allows for normalization of exposures with respect to exposure route, the calculation of total exposures through all routes, and allows the bioavailable doses to humans to be compared with bioavailable doses determined from animal studies. In cases where the bioavailabilities for the route of the estimated exposure and the route considered in the toxicological criterion development are the same, the bioavailability adjustment is, in effect, cancelled out by use on both sides of the risk characterization equation.

When evaluating the health risks related to exposures to metals, such as the COC being evaluated in the Flin Flon HHRA, an important aspect of a substance's bioavailability is the bioaccessibility exhibited by that substance. Bioaccessibility is the mass fraction of a substance that is converted to a soluble form under conditions of the external part of the membrane of interest. If one is evaluating bioaccessibility *via* the oral route, it is the fraction of substance that becomes solubilized within the gastrointestinal tract (*i.e*., stomach and small intestine). In the case of dermal exposures, it is the fraction solubilized on the outside of the skin (*i.e*., in sweat). To better characterize this fraction, a detailed site-specific *in vitro* bioaccessibility study was conducted to estimate the bioaccessibility of each of the COC present in soil and indoor dust media collected as part of the overall study. These site-specific oral bioaccessibility studies were used to help address the differences in oral bioavailability observed in these media *versus* the medium used in the study from which the toxicological criterion was derived.

4.2.5 Speciation

The geological history of the Flin Flon/Creighton area, and the highly mineralized nature of the area, will likely have significant implications on the form in which many of the COC will be available for potential exposure. The occurrence of metals in the environment related to natural deposits *versus* those arising from smelting and processing activities are likely to be different in structure which may require specific methodologies to address each particular form (*e.g*., soluble forms, sulfides, subsulfides, and oxides). The process of determining the actual form of metals present within a specific soil sample is typically referred to as speciation. This is relevant to the assessment of risks because the form of a metal in soil can have an important impact on its bioavailability and toxicity to human health. Due to their chemical structure, insoluble forms

of metals are less available for uptake into biological systems than more soluble forms which may be readily absorbed.

It may be beneficial to collect samples from a variety of different soil types and geographical locations to provide a complete speciation breakdown representative of soils in the study area. These data can be used in the HHRA to provide a more accurate reflection of the bioavailability and toxicity of specific metals to humans. Samples can be obtained through field sampling or from existing archived soil samples. For example, the speciation of lead can sometimes help to determine whether lead present in house-dust has originated from paint chips containing lead or some other outdoor source. As another example, it is widely accepted that different forms of arsenic have different toxicological significance to humans. Organic forms of arsenic typically found in biological samples are much less toxic relative to inorganic forms found in soil. It is noted that data are readily available to determine the relative speciation of arsenic in these types of media. As a result, speciation of arsenic in environmental media is generally not required. It is noted that arsenic speciation is a critical component to urinary arsenic studies which are currently beyond the scope of the project.

There is a paucity of toxicological information pertaining to species-related differences in toxicological potency for all COCs. As a result COC speciation was not considered further and each COC was evaluated as a single entitity. In most cases, toxicology studies utilized soluble species/forms of the COCs to ensure that chemicals will reach the target tissues in the toxicological study. The species/forms of the COCs in the environment are typically less soluble and therefore less available for uptake and toxicity.

4.3 Risk Characterization

The risk characterization step integrates the exposure and hazard assessments to provide a conservative estimate of human health risk for the receptors assessed in the various exposure scenarios. Potential risk were characterized through a comparison of the estimated or predicted chronic exposures from all pathways (from the Exposure Assessment) with the identified exposure limits (from the Hazard Assessment) for all COC.

Risk characterization for chemicals with a threshold-type dose-response consists of a comparison between the toxicological criteria (*i.e.*, the rate of exposure that would not produce adverse effects) against the total estimated exposure. This comparison is expressed as a Hazard Quotient (HQ) for oral and dermal exposures and a Concentration Ratio (CR) for inhalation exposures. These ratios are calculated by dividing the predicted exposure by the toxicological criterion, as indicated in the following equations:

$$Hazard\ Quotient = \frac{Estimated\ Exposure\ \left(\mu g / kg / day\right)}{Exposure\ Limit\ \left(\mu g / kg / day\right)}$$

Or

$$Concentration\ Ratio = \frac{Air\ Concentration\ \left(\mu g / m^3\right)}{Exposure\ Limit\ \left(\mu g / m^3\right)}$$

Risk characterization for COC with a non-threshold-type dose response (*i.e.,* carcinogens) consists of a calculation of the Incremental Lifetime Cancer Risk (ILCR), which is defined as the upper bound predicted risk of an individual in a population of a given size developing cancer

over a lifetime. The ILCR is expressed as the prediction that 1 additional person per ***n*** people might develop cancer, where the magnitude of ***n*** reflects the risks to that population; for example, if the ILCR is 1 person per 10, the predicted risks of any individual developing cancer would be higher than if the ILCR is 1 per 1,000. The following equation provides the method whereby the ILCR is calculated:

$$ILCR = Estimated\ Exposure\,(ug/kg/day)\,x\,q_1^*\,(ug/kg/day)^{-1}$$

where q_1* represents the cancer slope factor.

or

$$ILCR = Air\ Concentration\ \left(\mu g/m^3\right)x\ Unit\ Risk\ \left(\mu g/m^3\right)^{-1}$$

The resulting estimated cancer risk can then be compared to an acceptable upper bound risk level of cancer to determine if exposures to the assessed chemical pose an unacceptable health risk. Health Canada (2006a) indicates that given the conservatism associated with the derivation of cancer slope factors and unit risk values, and that the lifetime probability of developing cancer in North America from background sources is approximately 40% , an incremental lifetime cancer risk level of 1-in-100,000 is consisered to be acceptable for the purpose of assessing cancer risks associated with contaminated sites.

In addition to the assessment of risks resulting from chronic, multi-media exposures, acute or short-term risks associated with the inhalation of COC in ambient air were assessed through comparison with short-term (*i.e.*, 1 hour or 24 hours) air quality guidelines.

4.3.1 Evaluation and Interpretation of Hazard Quotients and Incremental Lifetime Cancer Risk

The evaluation and interpretation of HQs and ILCRs can be applied with greatest confidence to situations where comparisons are made between the HQs/ILCRs of two or more independent exposure scenarios. From such comparisons, the incremental difference in the potential for occurrence of adverse health effects between the two or more different scenarios (*e.g.*, study area *versus* typical background) can be assessed with reasonable confidence since the same exposure and hazard assessment methodologies are used in addressing each situation. Most of the uncertainties in such comparative assessments are related to the ability to accurately estimate COC concentrations in the various environmental media that determine the different exposure pathways, and in the estimation of the toxicological criteria that exposure estimates are compared against.

Hazard Quotients (HQ)

Once HQ values have been determined for threshold chemicals (non-carcinogens), they are compared to a benchmark indicator of “safety”, which is sometimes called the Critical Hazard Quotient (CHQ). In general, 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. Therefore, the benchmark of safety would be 1.0, assuming that estimates of exposure from all relevant exposure pathways are included.

However, for threshold chemicals, the exposure limits (or toxicological criterion) represent the level of total exposure which would not result in adverse health effects, regardless of the source or pathways of exposure. As most risk assessments generally evaluate single or few sources of contamination and a limited number of exposure pathways, the selection of a CHQ value of 1.0 for threshold chemicals is not always appropriate. In an attempt to address this issue, the CCME (1996b) considers that a substance has the potential to be present in all media, and assumes an allocation of 20% of the residual tolerable daily intake for each of the five major media (*i.e.,* air, water, soil, food, consumer products). Similarly, the OMOE recommends apportioning 20% of the total exposure to any one pathway (OMOE, 1987), in the absence of information to the contrary. This means that the overall CHQ (*i.e.,* 1.0) must also be apportioned for the single source (*e.g.,* a contaminated site) under consideration. This yields a value of 0.20, which can be considered as the CHQ representing a situation in which no adverse health effects are likely to be associated with the estimated level of exposure for a given pathway. Therefore, if threshold chemicals are determined to have HQ values equal to or less than 0.20, exposure rates are considered to be less than 20% of the exposure limit (toxicological criterion), and no adverse health effects would be expected to occur in the receptors and scenarios evaluated in the risk assessment. If HQ values are greater than 0.20, the estimated exposure rates are considered to exceed 20% of the exposure limits, indicating the potential for adverse effects in sensitive individuals or in some of the exposure scenarios considered.

It should be noted however, that if the risk assessment includes the estimation of exposures to COC associated with all significant background sources (*e.g.*, market basket foods, consumer products), the total exposure of each receptor is being adequately accounted for. In the current assessment, all significant sources of exposure were accounted for, therefore, the use of a CHQ value of 1.0 was considered to be appropriate to represent an “acceptable level” of exposure. It should be recognized there is some uncertainty with respect to the potential contribution of consumer products to an individual’s EDI.

Incremental Lifetime Cancer Risk (ILCR)

For non-threshold chemicals (*i.e.,* chemicals believed to act as carcinogens), the risk characterization is based on limiting ILCR to some level considered “negligible” or “acceptable”. As previously discussed, the ILCR represents a predicted upper bound incremental risk of cancer over a lifetime to an individual member of a population of a given size, and is expressed as a risk level (*e.g.,* one person per ***n***). Calculated ILCRs are compared to an upper bound benchmark risk level that is considered to be acceptable by the responsible regulatory agency in a given jurisdiction. In Manitoba, the Province follows the Health Canada recommendation for acceptable ILCR of one-in-one hundred thousand ($1x10^{-5}$). In other jurisdictions, negligible or *de minimis* cancer risk levels are generally considered to be in the range $1x10^{-4}$ to $1x10^{-6}$ (Health Canada, 2004c). An ILCR refers to the contribution that a facility or site makes to the total risk excluding background sources.

Where estimated risks (ILCRs for non-threshold acting chemicals) or risk indicators (HQs, for threshold-acting chemicals) are at or less than the acceptable level, it can be concluded that no observable adverse health effects would be expected to occur including sensitive subpopulations or groups, within the exposure scenarios considered in the HHRA. Risk estimates that are substantially less than the acceptable level are not considered to require further evaluation. In situations where risks are predicted to be within the same order of magnitude as the acceptable level, re-evaluation of certain model parameters (*e.g.,* chemical concentration estimates, exposure parameters, and toxicological criteria) is conducted before

the potential risks to health are fully characterized. In these situations, consideration must be given to the possibility of adverse health effects, but a slight exceedance (or lack of exceedance) of the acceptable risk benchmarks do not typically indicate a high potential for risk.

The methods and assumptions used in this HHRA are designed to be conservative (*i.e.,* health protective), and have a built-in tendency to overestimate, rather than underestimate, potential health risks. Thus, risk estimates that are within an order of magnitude of the acceptable risk benchmarks may reflect overestimation through the use of conservative assumptions and parameters. In these cases, interpretation of the risk estimates may indicate that given the conservatism of the assessment, no adverse health effects would be expected despite the exceedance of the acceptable risk level or, that further assessment (*i.e.,* progression to a more detailed and specific risk assessment that could involve further data collection or probabilistic exposure analysis), or mitigative measures are warranted.

When predicted risks are substantially greater than the acceptable level (*i.e.,* more than 10-fold), the potential for adverse effects in sensitive individuals or in some of the exposure scenarios is suggested. Again, however, the re-evaluation of such HQs/ILCRs is extremely important since both the exposure estimation procedures and the toxicological criteria are based on a series of conservative assumptions that tend to overestimate exposures and risks. Often, a sensitivity analysis is conducted which facilitates the re-evaluation by focusing on the proportional contribution of various parameters to the final HQ/ILCR value. Once the major contributing model parameters have been identified, they can be re-evaluated to determine their impact on the resulting risk estimates and whether health risks have been under-estimated or over-estimated. Most often, the sensitivity analysis indicates that exposures and risks were overestimated. This occurs because a certain amount of over-estimation of risk is inherently built into the risk assessment process. For example, in cases where there is considerable uncertainty in the data such as the determination of toxicological criteria for cancer causing chemicals (*e.g.,* arsenic), a conservative dose-response extrapolation model is used to derive the toxicological criterion to ensure the protection of human health. In probabilistic analyses, the estimates of potential adverse effects on human health at the upper end of the distribution forecast (*e.g.,* the 95th percentile upper confidence limit on the mean) represent the combination of numerical parameter values that occur infrequently based on the frequency distribution functions used for the various parameter values. Re-evaluation of the basis for these values at the upper end of the frequency distribution forecast must be conducted prior to recommending any remedial or other mitigative actions that would be based on these risk estimates. The outcome of this re-evaluation may include recommendations towards progressing to additional probabilistic analyses, additional data collection, or remedial action. Probabilistic analyses also allows risk managers to predict the effectiveness of different risk management activities by reducing exposure and risk profiles.

4.3.2 Consideration of Chemical Mixtures

Concurrent exposures to more than one chemical may result in interactions among toxicological effects; this may result in a combined toxicity which is equal to the sum of toxicities of the individual chemicals (additivity or independence), greater than the sum (synergism or potentiation) or less than the sum (antagonism). In general, toxicological interactions depend on the chemicals present, the levels of exposure to each, their mode of action and their concentrations. Most non-additive interactions can only be demonstrated at relatively high exposures, where clear adverse effects are observed. Such interactions have not been observed or quantified at the relatively low rates of exposure typical of those associated with most environmental situations (NAS, 1983; Krewski and Thomas, 1992). Based on this, a

summary of the scientific literature will be provided on possible effects of mixtures for the COC which are considered in the HHRA (Chapter 6, Section 6.4).

4.3.3 Sensitivity Analysis and Identification of Uncertainties and Limitations

A sensitivity analysis of the HHRA pathways was conducted to determine the relative contribution to total exposure from the various exposure pathways. This information will be critical for decision-making related to the need for and possible effectiveness of risk management, if deemed necessary. In addition, key uncertainties and limitations have been identified in the HHRA report, and their effect on the HHRA results were discussed in the report.

4.4 Risk Management Recommendations

If, after careful review and consideration of the factors described previously, the results of the risk characterization indicate that there may be unacceptable risks posed to some receptors of concern, then preliminary recommendations towards mitigation of those risks can be made. Risk management recommendations may suggest possible ways in which exposure pathways contributing significantly to overall exposure and risk can be limited or eliminated. For example, if contact with surface soils is driving risk, depending on the current and future uses of the land, it may be appropriate to simply put a layer of asphalt or clean fill over the contaminated soil, thereby preventing soil contact and mitigating the risk. Soil amendments, such as liming, can also be used to mitigate risks, in that they can modify the availability of chemicals in the soil. In some cases it may be necessary to remove contaminated media to mitigate risk. In cases where it is determined that risk management is necessary, risk management criteria (RMC) are used to guide potential remediation activities.

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, 2001b). 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 further consideration may be warranted.

The PTCs 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.
The need to recommend risk management criteria (RMC) is based on a number of key considerations including:

1. The nature, extent and duration of the risk and the uncertainties in how risks are estimated;
2. Evidence or lack of evidence of actual harm to health in the community; and,
3. Outcomes of risk assessments in other communities with similar or higher levels of exposure.

There may also be legal, financial, political, and community concern-based issues that play a role in the establishment of suitable RMC and subsequent action that may be taken.

For the current assessment, the risk predictions for the residential exposure scenario (*i.e.*, central tendency estimates) were used to generate the PTCs (see Chapter 5 for a further discussion). Ecological risks, as well as other factors (*i.e.*, legal, financial, political and community concerns) should be considered prior to finalizing the RMC and choosing a risk management option.

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