SUDBURY AREA RISK ASSESSMENT

VOLUME II – CHAPTER 5: RESULTS AND DISCUSSION

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5.0 RESULTS AND DISCUSSION

This chapter provides an overview of the results of the detailed human health risk assessment, as well as a discussion of the implications of these results for residents of the GSA. The HHRA considered a variety of data, chemicals, communities, individuals, exposure pathways and assumptions including:

- Six chemicals of concern (COC) (arsenic, cobalt, copper, lead, nickel, and selenium);
- Five communities of interest (COI) (Copper Cliff, Falconbridge, Coniston, Sudbury Centre, Hanmer [as a background community], and the Typical Ontario Resident [TOR]);
- Five receptor age classes (*i.e.*, infant, preschool child, child, adolescent and adult) and composite lifetime individuals for each of the two genders;
- Consideration of the general population and a special receptor category of avid anglers and hunters (including First Nation receptors);
- Receptor characteristics characterized by: (i) average or *Central Tendency Exposure* (CTE); and, (ii) upper-bound or *Reasonable Maximum Exposure* (RME) estimates;
- Inhalation, oral and dermal exposure pathways; and,
- A large database of site-specific media concentrations characterized by average (95% upper confidence limit on the mean) or upper-bound (maximum) statistics.

Potential risk is characterized by comparing predicted exposures from all pathways with the exposure limits or toxicity reference values. For non-carcinogenic COC, this comparison is typically referred to as the *Hazard Quotient* (HQ) and is calculated by dividing the predicted exposure level by the exposure limit. If the total chemical exposure from all pathways is equal to or less than the exposure limit, then the HQ would be 1.0 or less, and no adverse health effects would be expected (refer to Chapter 4 for a more detailed discussion of this topic).

For chemicals with non-threshold-type dose responses (*i.e.,* carcinogens), the comparison is referred to as the *Incremental Lifetime Cancer Risk Level* (ILCR) or more simply a *cancer risk level* (CRL) and is defined as the incremental risk of an individual in a population of a given size developing cancer over a lifetime. The ILCR is calculated by multiplying the predicted exposure by the slope factor or unit risk value. The ILCR is expressed as the prediction that one person per ***n*** people would develop cancer, where the magnitude of ***n*** reflects the risks to that population. In the case of carcinogens, the acceptable risk level in Ontario is considered to be an incremental increase in cancer risk of one-in-one million (*i.e.*, one

additional cancer per million people). Typically, incremental lifetime cancer risks are calculated by multiplying a chemical- and route-specific cancer slope factor by facility related exposures. For the current assessment, an evaluation of all potential exposure sources and pathways was completed (regardless of source).

As discussed previously, risk assessments typically employ the 95% upper confidence limit on the arithmetic sample mean (95% UCLM) to characterize the exposure point concentration (EPC) of a given exposure unit (U.S. EPA, 2001a). The sample mean is based on a collection of samples from the exposure unit and therefore, uncertainty exists as to whether the sample mean is a true reflection of the population mean. As a result, the 95% UCLM can be thought of as an estimate of the true population mean for a given exposure unit. In this case, the exposure units were defined as the communities under assessment in the HHRA. The underlying assumption used when developing the chronic exposure scenarios was that individuals would move randomly within each community and, therefore, over time, come into contact with the average soil concentration within a given community (or exposure unit).

If the property or site of concern were a single residential lot, it would be reasonable to assume that an individual would move in a random fashion within his or her own property. In reality, individuals do not move in a random fashion within their community, but rather exhibit predictable spatial patterns in their movements. For example, many individuals will tend to spend the majority of their time between home and work or school. Therefore, the evaluation of risks on the basis of average EPCs (assuming random movement) in an area-wide risk assessment may underestimate risks for some receptors. As a result, in area-wide assessments, where data permits, it is necessary to look at upper bound concentration estimates, in addition to averages for the EPC.

For this reason, two statistics were used to characterize COC concentrations in soils within each community of interest: (i) the average soil concentration (based on the 95% UCLM); and, (ii) the maximum soil concentration. For all other media, data were not sufficient for a site-by-site evaluation, and as such, other media were only considered on an area-wide basis, using average (95% UCLM) concentrations (*i.e.*, air monitors were set-up to reflect community concentrations). As indoor dust concentrations were predicted based on outdoor soil concentrations, this medium was evaluated in a similar manner as was soil. Resultant risk estimates utilizing the average and maximum soil concentrations were referred to as the HQ_{avg} and the HQ_{max}, respectively.

In addition to a benchmark comparison, both the strength-of-evidence and weight-of-evidence must be evaluated when considering the results of an HHRA, including consideration of non-site related exposures (*i.e.*, a comparison of site conditions to background) and the consideration of additional pieces of information that may be available (*e.g.*, biological monitoring results, public health information, *etc*.).

As a result, the calculation and interpretation of estimated human health risk estimates becomes a multi-step process involving the following key elements:

1. Risks estimated using both CTE and RME receptor characteristics and average (95% UCLM) concentrations for all the exposure media, to obtain a general picture of overall risk for each COI. Non-cancer HQ estimates were calculated for all individual receptor groups and presented for the female preschool child as this receptor is the most highly exposed individual (see Figure 5-1). Incremental lifetime cancer risks were also considered. CRL estimates were generated using the female lifetime or composite receptor which includes all five age categories (*i.e.,* infant, preschool child, child, adolescent and adult). The resultant values are referred to as HQ_{avg} and CRL_{avg}, and provide risk estimates for the ***average*** resident of that community (note: risk predictions presented as HQ or CRL are equivalent to HQ_{avg} and CRL_{avg}, respectively).

2. For RME receptor characteristics, risks were estimated using both the average (95% UCLM) and maximum soil concentrations. The resultant risk estimates are referred to as the HQ_{avg} and HQ_{max}, and CRL_{avg} and CRL_{max}, respectively. This provides an estimate of risk for receptors exposed to the ***maximum*** (*i.e.*, worst-case) soil concentration in each COI, characterizing exposure potential for those individuals who may reside on residential properties that have soil concentrations greater than the COI average.

3. If no unacceptable health risks were predicted using average (95% UCLM) and maximum soil concentrations, then no further assessment was considered necessary. If unacceptable risks were predicted, further evaluation of the exposure assessment was undertaken through a weight-of-evidence approach.

4. If unacceptable risks were predicted, site-specific risk management goals for soil (termed a *Soil Risk Management Level* or $SRML_{soil}$) were derived.

5. With the exception of arsenic and lead, the results of the risk assessment are presented separately for oral/dermal and inhalation exposures. Arsenic has been found to act *via* a similar toxicological mechanism following either inhalation, oral or dermal exposure. The TRV

established by the MOE for lead is for all pathways and as such risk reported for oral/dermal include inhalation as well.

6. The relative contribution of each exposure pathway (*i.e.*, soil, dust, drinking water, local foods, market basket, and air) is also presented. This is useful for identifying circumstances in which meaningful pathway-specific interventions may be undertaken, if necessary.

For demonstration purposes, discussion of non-cancer risks is limited to the female preschool child receptor under specific exposure scenarios (general population). Discussion of cancer risks is likewise limited to the female composite receptor. Brief mention of risks to other receptors is made in Section 5.1 for comparison purposes. However, detailed results for all receptors, COC, and COI are provided in Appendix O.

5.1 Overview of Results

The following sections provide a summary of the human health results, estimated deterministically. Human health results are expressed as hazard quotients (HQ values) for non-carcinogenic chemicals and as lifetime cancer risk levels (CRL) for chemicals considered carcinogenic to humans. The text is further subdivided into sections for oral/dermal exposure and inhalation exposure. A more detailed discussion of the results for each COC is provided in Section 5.2.

5.1.1 Non-Cancer Endpoints (General Population)

5.1.1.1 Oral/Dermal Exposures

Non-Cancer Lifetime vs. Individual Receptor Group Risk Estimates

For the assessment of non-cancer health risks, it is common to consider HQ (risk) estimates for the most sensitive or highly exposed life stage. Average lifetime risks can be considered when the toxicological endpoint of concern is a result of lifetime exposures (as for nickel). As demonstrated in Figure 5-1, for nickel in Sudbury Centre, consideration of the preschool child (toddler) provides the most conservative evaluation of non-cancer risks relative to other receptor age classes. Lifetime risk estimates are significantly less for the other age groups, and as such, consideration of the toddler for risk assessment and risk management purposes was considered conservative. It is noted that although both male and female receptors were evaluated separately, no significant differences in exposure estimates between

males and females were observed and, therefore, for the purposes of discussing the results, the female risk estimates will be used. Appendix O provides detailed results for all receptor age classes, COC and COI for both male and female receptors.

The following sections provide a summary of the human health risk estimates for a female preschool child (aged six months to less than five years). As indicted above, the preschool child (toddler) was determined to be the most sensitive (highly exposed) individual relative to all other age classes due to the higher potential for exposure on a bodyweight basis. As a result, non-cancer HQ estimates are summarized and presented for the female preschool child only.

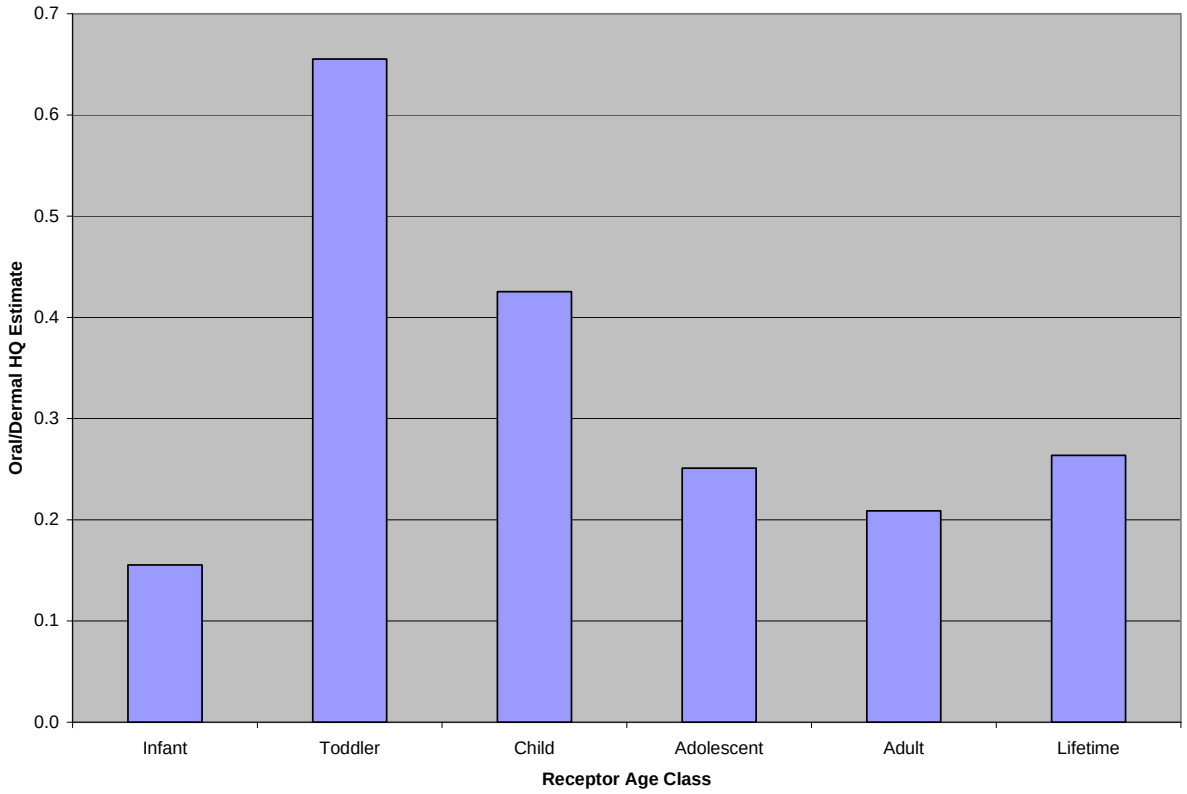

Figure 5-1 Lifetime HQ_{avg} Estimates for Nickel (RME) - Sudbury Centre

An overview of the estimated risks (HQ_{avg}) for each COC, in each COC, and for both CTE and RME exposure conditions is provided in Figure 5-2.

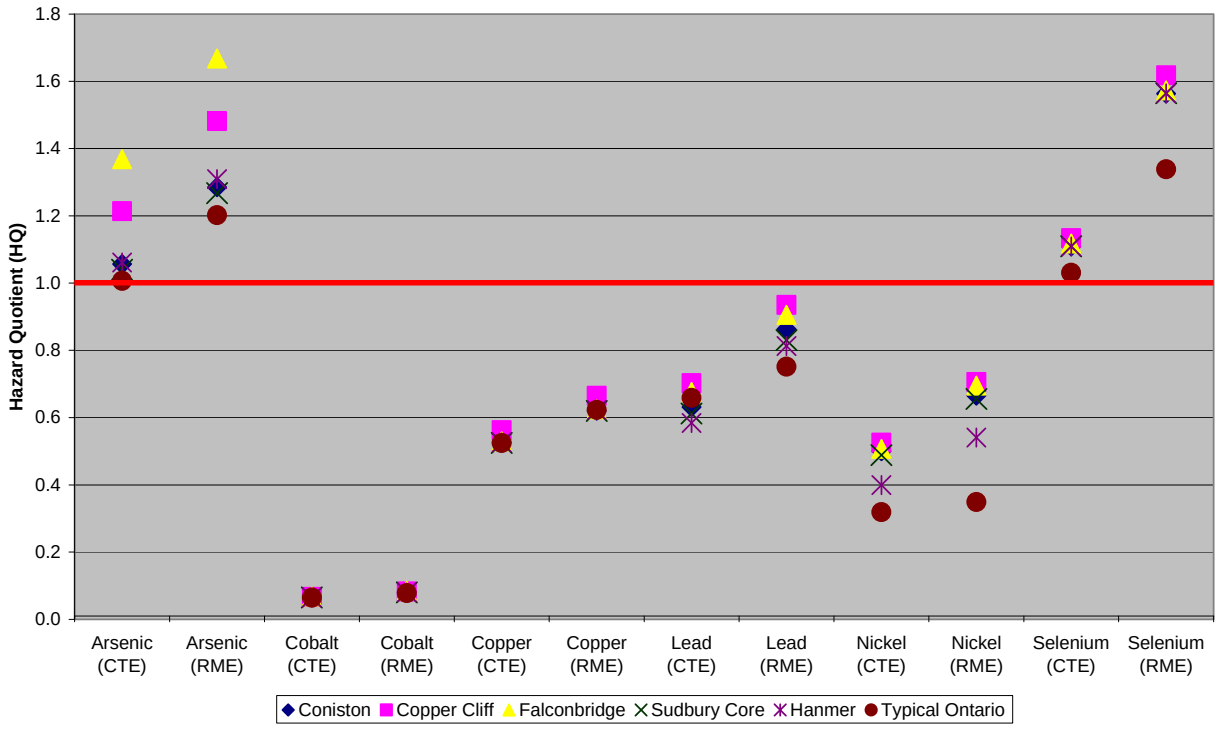

Figure 5-2 Mean Oral Hazard Quotient (HQ_{avg}) Estimates for the Female Preschool Child - General Sudbury Population

Results from Sudbury Centre are presented in Tables 5.1 and 5.2 to illustrate trends in the data. Sudbury Centre is used here as an example COI. The HQ_{avg} estimates (*via* oral and dermal pathways only) for the general Sudbury population of female preschool children exposed to cobalt, copper, lead and nickel were less than a value of 1.0 for all COI, under both central tendency (CTE) and RME exposure assumptions (See Table 5.1 and Figure 5.2). The same is also true for all other receptors (male and female infants through adults) in all other COI. Therefore, no human health risks are expected for oral/dermal exposures to cobalt, copper, lead or nickel on a community-wide basis from oral exposure (*i.e.*, the use of the 95% UCLM soil concentration for each COI). As previously indicated the HQ_{avg} estimates refer to risk estimates that have been derived using the 95% UCLM soil concentration for a specific COI.

Table 5.1 Summary of Non-Cancer Assessment Results for Female Receptors for Oral/Dermal Exposures – Sudbury Centre (Average Soil Concentrations)

COC	HQ_{avg} Estimates				
	Female Infant	Female Preschool Child	Female Child	Female Adolescent	Female Adult
CENTRAL TENDENCY ESTIMATES (CTE)					
Arsenic	0.15	1.0	0.59	0.32	0.25
Cobalt	0.03	0.13	0.08	0.04	0.026
Copper	0.42	0.52	0.31	0.17	0.12
Lead	0.16	0.61	0.29	0.15	0.11
Nickel	0.13	0.49	0.27	0.16	0.14
Selenium	0.26	**1.1**	0.77	0.41	0.27
REASONABLE MAXIMUM EXPOSURES (RME)					
Arsenic	0.18	**1.3**	0.77	0.44	0.33
Cobalt	0.04	0.16	0.1	0.056	0.036
Copper	0.54	0.62	0.40	0.22	0.15
Lead	0.19	0.83	0.51	0.28	0.23
Nickel	0.16	0.66	0.43	0.25	0.21
Selenium	0.34	**1.6**	**1.2**	0.63	0.48

Note: **Bolded** HQ values indicate predicted exposures which exceed the regulatory benchmark (*i.e.*, HQ > 1).

The HQ_{avg} estimates for the general Sudbury population of female preschool children (oral exposure) to selenium and arsenic were greater than a value of 1.0 for all COI, and for the typical Ontario scenario. HQs for the CTE female preschoolers ranged from 1.0 to 1.1 and for the RMEs from 1.1 to 1.6. The selenium HQ_{avg} estimates for female preschool children do not appear to differ significantly from one COI to another, although under the RME scenario, selenium HQ_{avg} estimates appear elevated relative to typical Ontario conditions (Figure 5.2). Selenium HQ_{avg} estimates for the female preschool child under the CTE and RME scenarios at Sudbury Centre were 1.1 and 1.6, (Table 5.1), respectively (selenium results are further discussed in Section 5.2.1). By comparison, CTE and RME HQ_{avg} estimates for the female adult in Sudbury Centre were 0.27 and 0.48, respectively.

Arsenic HQ_{avg} estimates for the general population of female preschool children were greatest at Falconbridge and Copper Cliff, with CTE and RME HQ_{avg} estimates ranging between 1.4 and 1.7 at Falconbridge and 1.2 and 1.5 at Copper Cliff (Figure5.2). Arsenic HQ_{avg} estimates for the general background population of female preschool children (*i.e.,* typical Ontario) were 1.0 and 1.2 for the CTE and RME, respectively. Arsenic results are further discussed in Section 5.2.1.

As discussed in Section 5.0, when considering community-wide risks that employs an estimate of the average (*i.e.,* the 95% UCLM) soil concentration for an entire COI, it is prudent to consider smaller, more localized areas, which may be associated with COC soil concentrations in excess of the community-wide average. To demonstrate this point, Table 5.2 provides a comparison of the RME results for female preschool children (oral/dermal exposures) in Sudbury Centre using average (HQ_{avg}), and maximum (HQ_{max}) soil concentrations. Similar trends are observed with CTE results for all COI, age groups, and receptors, including hunters/anglers.

Table 5.2 Comparison of Average (HQ_{avg}) and Maximum (HQ_{max}) RME Exposure Scenarios for the Female Preschool Child – Sudbury Centre

COC	Average Exposure Scenario		Maximum Exposure Scenario	
	95% UCLM Soil Concentration (µg/g)	HQ_{avg}	Maximum Soil Concentration (µg/g)	HQ_{max}
Arsenic	7.17	**1.3**	59.0	**1.4**
Cobalt	11.3	0.16	100.0	0.17
Copper	204.0	0.62	1640	0.63
Lead	35.9	0.83	309.8	**1.1**
Nickel	210.1	0.66	3260	0.79
Selenium	1.30	**1.6**	12.5	**1.6**

On the basis of these results, arsenic and selenium clearly require further consideration (see Sections 5.2.1 and 5.2.6, respectively). Although the lead HQmax estimate of 1.1 marginally exceeded a value of 1.0, due to public/regulator concerns and consideration of model uncertainties and sensitivities, lead was also carried forward into the weight-of-evidence approach.

5.1.1.2 Inhalation Exposures

With the exception of nickel in each COI, all non-cancer inhalation HQ estimates for the female preschool child were less than a value 1.0 (refer to Table 5.3 and Figure 5-3 for results in Sudbury Centre). Inhalation risks specifically for nickel are discussed in detail in Section 5.2.5.2. It should be noted that HQ values presented in Figure 5-3 and Table 5.3 are age specific, while those discussed in Section 5.2.5.2 are more generic in nature. Inhalation HQ estimates for lead are not presented in Figure 5-3 because the oral HQ estimate for this metal includes all routes of exposure. Arsenic results are also not provided since arsenic is assumed to act *via* a carcinogenic mechanism following inhalation exposure. Health risk estimates for all receptors (males and females of all age classes) can be found in Appendix O.

Table 5.3 Summary of Non-Cancer Assessment Results for Female Receptors for Inhalation Exposures – Sudbury Centre

COC	HQ Estimates				
	Female Infant	**Female Preschool Child**	**Female Child**	**Female Adolescent**	**Female Adult**
CENTRAL TENDENCY ESTIMATES (CTE)					
Arsenic	NA	NA	NA	NA	NA
Cobalt	0.016	0.034	0.027	0.016	0.015
Copper	0.14	0.30	0.23	0.14	0.13
Lead	NC	NC	NC	NC	NC
Nickel	**3.9**	**8.3**	**6.5**	**3.9**	**3.7**
Selenium	0.00038	0.00081	0.00063	0.00038	0.00036
REASONABLE MAXIMUM EXPOSURES (RME)					
Arsenic	NA	NA	NA	NA	NA
Cobalt	0.017	0.036	0.028	0.017	0.016
Copper	0.15	0.32	0.25	0.15	0.14
Lead	NC	NC	NC	NC	NC
Nickel	**4.2**	**8.9**	**6.9**	**4.1**	**3.9**
Selenium	0.00041	0.00087	0.00067	0.00040	0.00038

Note: NA – not applicable; NC – not calculated (oral exposure limit assumed all pathways including inhalation).
Bolded HQ values indicate predicted exposures which exceed the regulatory benchmark (*i.e.*, HQ > 1).

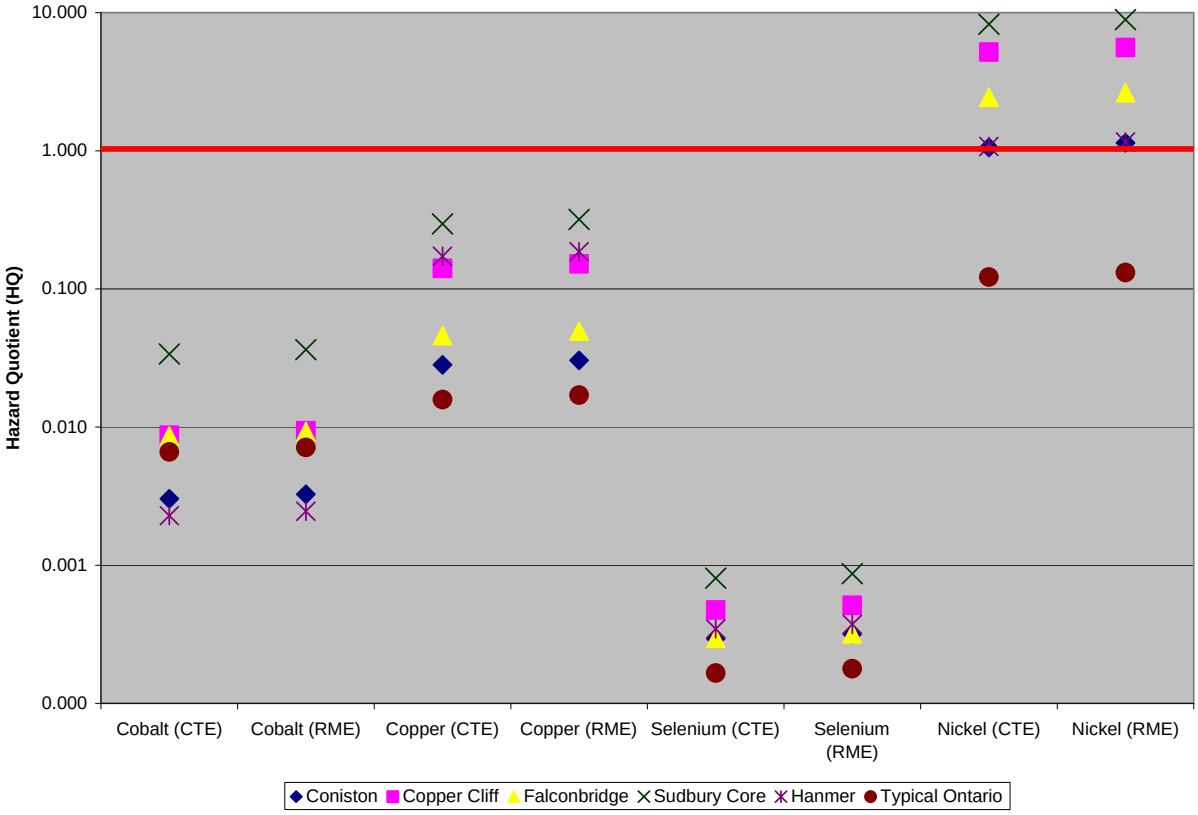

Figure 5-3 Inhalation Hazard Quotient (HQ) Estimates for the Female Preschool Child - General Sudbury Population

5.1.2 Carcinogenic Endpoints

Arsenic cancer risks represent lifetime cancer risk levels (CRL) for a female composite (or lifetime) receptor resulting from exposure to inorganic arsenic from all Sudbury specific pathways of exposure and are presented in Figure 5-4 (all COI). It should be noted that potential carcinogenic effects related to nickel inhalation exposure are addressed in Section 5.2.5.2.

Under the RME receptor exposure scenario, arsenic lifetime CRL estimates range from 1.3×10^{-4} (or 1.3 in 10,000) in Coniston to 2.5×10^{-4} (or 2.5 in 10,000) in Falconbridge. Lifetime CRL estimates for female composite receptors under typical Ontario conditions were between 5.5×10^{-5} to 6.3×10^{-5} (*i.e.,* 5.5 and 6.3 in one hundred thousand for CTE and RME scenarios, respectively). Arsenic results are further discussed in Section 5.2.1.

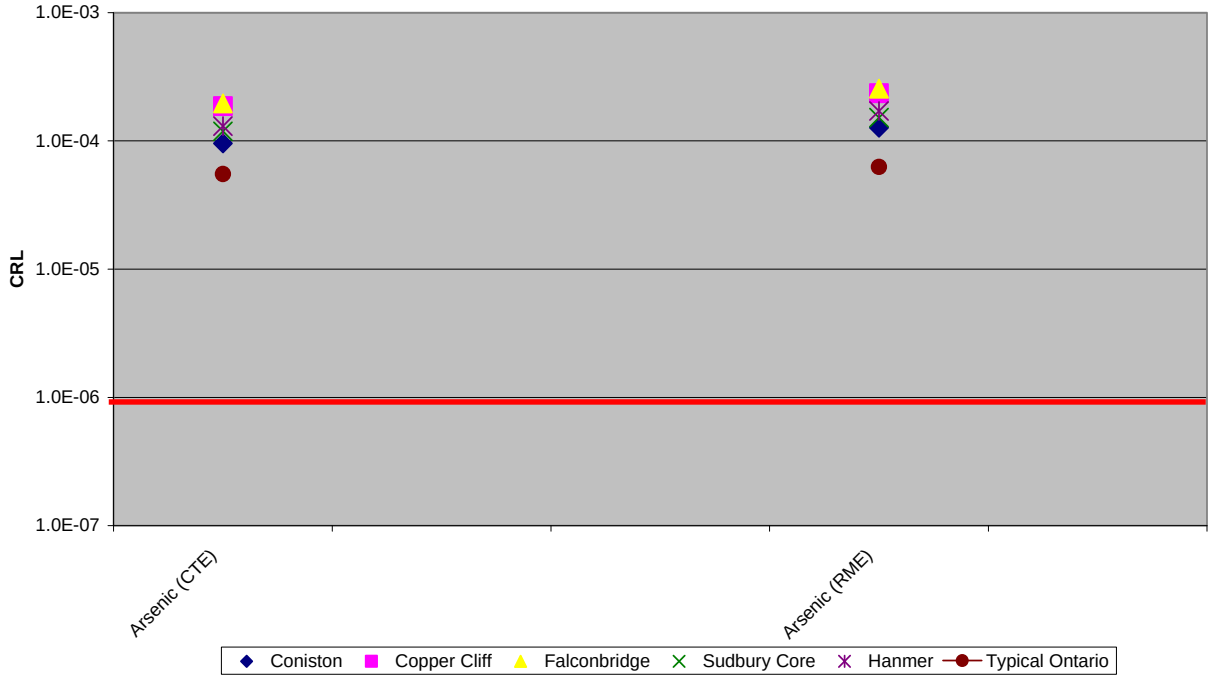

Figure 5-4 Female Lifetime Cancer Risk Level Estimates - General Sudbury Population

5.1.3 Hunting and Fishing Populations within the GSA

In addition to the general Sudbury population, individuals within the GSA who participate in hunting and fishing activities were considered separately when predicting HQ and ICRL estimates. Members of the general GSA population were assumed to consume local wild game and fish; however, members of the hunting/fishing sub-population (including First Nation members) were considered to consume significantly more local wild game and fish relative to those in the general population. Refer to Chapter 2 for a discussion regarding wild-game and fish intake rates. Predicted HQ (female preschool child) and CRL (female composite receptor) values of the hunting and angling sub-populations of the GSA were compared to those of the general Sudbury population. Detailed and summary results for all receptors (males and females of all age classes) can be found in Appendix O.

Figure 5-5 provides a comparison of HQ_{avg} estimates for the general GSA population of female preschool children *versus* those of female preschool children in an avid hunting/fishing sub-population.

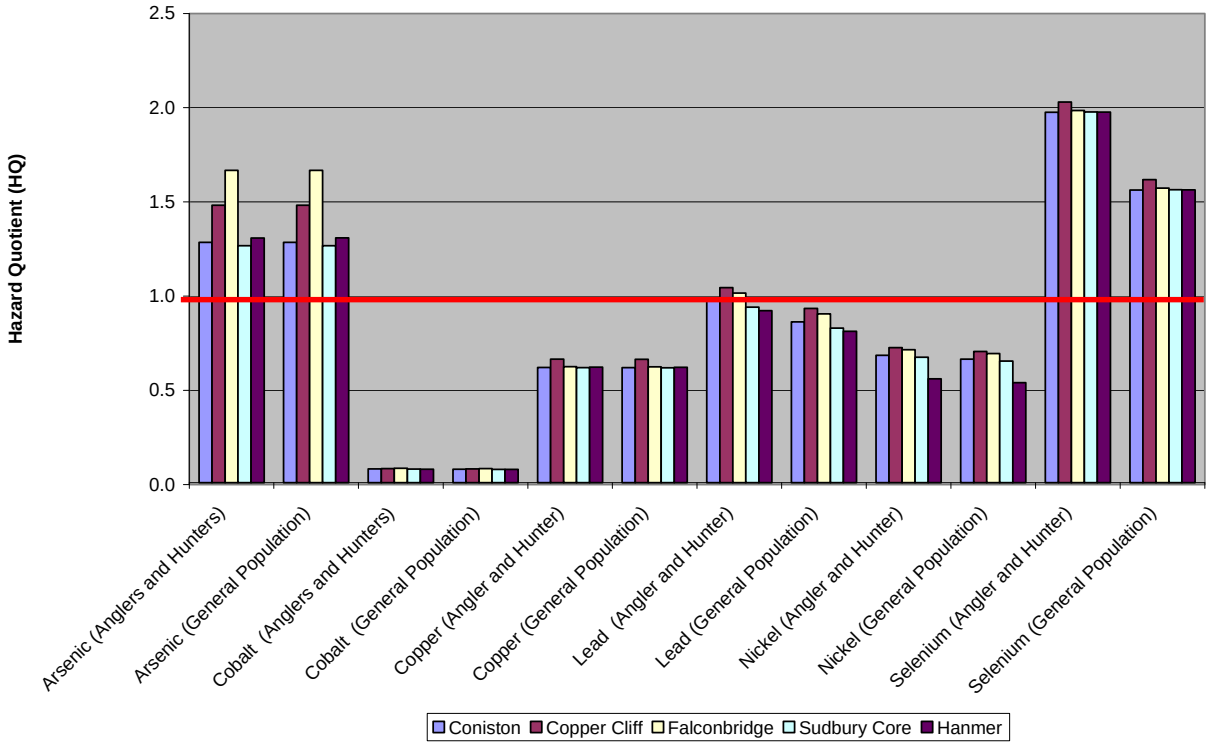

Figure 5-5 RME Oral Hazard Quotient Estimates – Female Preschool Child - General Sudbury Population *versus* Anglers and Hunters

Little difference, if any, was observed in HQ_{avg} estimates (under a RME scenario) between female preschool children from the general GSA population and those from an avid hunting and angling population. The greatest differences in HQ_{avg} estimates between the two populations were observed for selenium and lead. Selenium HQ estimates of 1.6 and 2.0 were predicted for the female preschool child of the general and hunting/angling sub-populations living in Sudbury Centre, respectively. Slight differences in HQ_{avg} estimates predicted for lead were also observed between the two populations.

No difference in arsenic lifetime CRL estimates for female composite receptor was observed between those who participate in hunting/fishing activities and members of the general GSA population (see Figure 5-6).

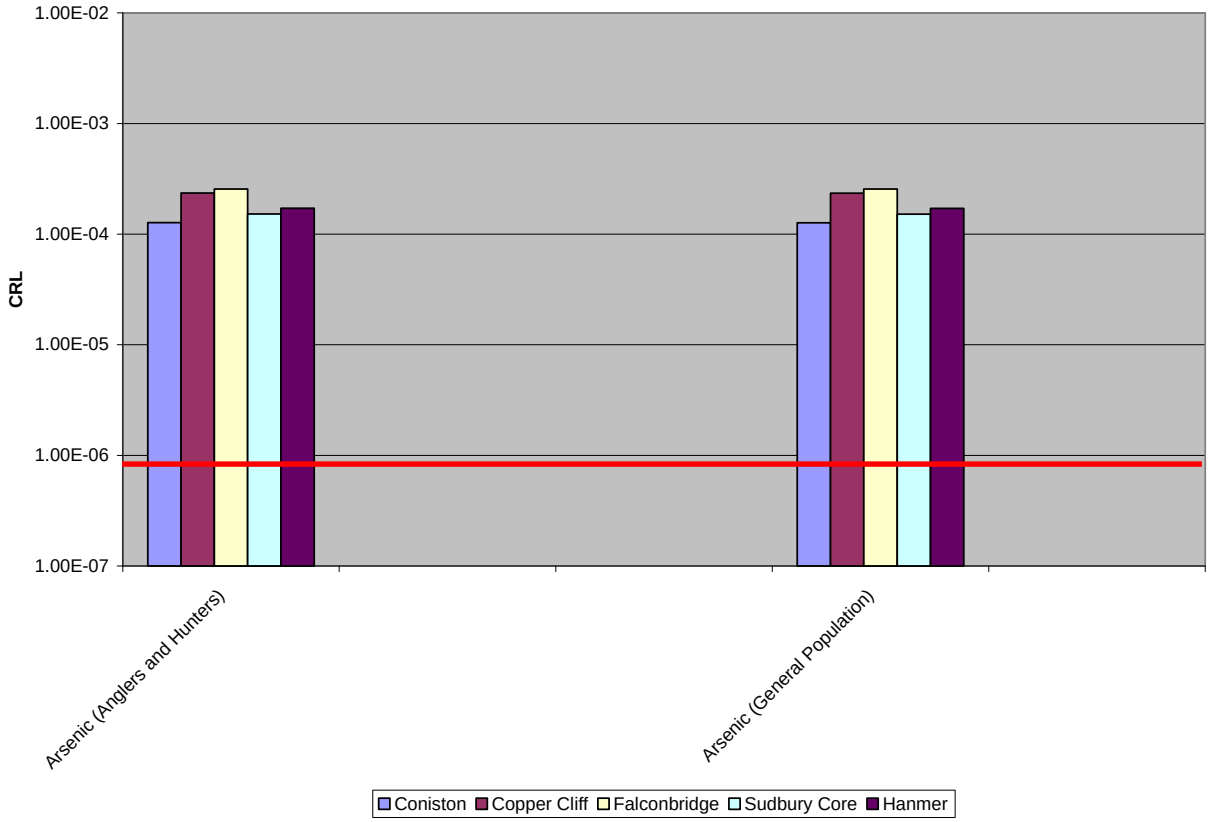

Figure 5-6 Female Lifetime Cancer Risk Estimates - General Sudbury Population *versus* Anglers and Hunters

5.2 Detailed Discussion of Results

The following sections provide additional analysis and discussion regarding the results presented earlier. Note that the results presented in Tables 5.3 and 5.4 for female receptors are provided as examples of the results of the assessment. Similar tables for all COI, COC, and receptors are provided in Appendix O.

5.2.1 Arsenic

Health-based arsenic soil standards in Ontario (*i.e.,* soil concentrations resulting in incremental lifetime cancer risks (ILCRs) of less than one-in-one million) result in the derivation of impractical soil remediation standards that are typically lower than the levels of arsenic found to occur naturally in many soil environments.

According to the *Guideline for Use at Contaminated Sites in Ontario* (MOE, 1996) and the Risk Assessment Procedures document (MOE, 2005), site-specific health-based soil standards for non-threshold (*i.e.,* carcinogenic) compounds should be developed using an incremental lifetime cancer risk of 1×10^{-6} per exposure medium. A health-based soil standard based on direct contact pathways only and an ILCR of one-in-one million can produce health-based soil standards between 1.0 and 2.0 mg arsenic/kg soil. The use of site-specific relative accessibility factor (RAF) adjustments to account for differences in accessibility between different environmental media (*i.e.,* water *versus* soil) could potentially increase (depending on site conditions) the health-based portion of the standard by 30 to 60%. This would in turn result in health-based soil standards for arsenic between approximately 1.5 and 3.0 mg/kg (assuming a 50% RAF).

By definition, health-based soil standards need to be added to an existing background concentration in soil. Given that health-based values for arsenic can range between 1.5 and 3.0 mg/kg (at an ILCR of 1×10^{-6}), health-based soil standards for inorganic arsenic rarely differ significantly from local background soil levels. The Ontario Ministry of Environment currently has a soil standard for residential/parkland and agricultural land uses of 20 mg/kg (25 mg/kg for medium/fine textured soils). These standards are not health-based but rather a reflection of the 98th percentile background concentration of 14 and 17 mg/kg for rural and urban parkland soils, respectively (*i.e.,* the OTR_{98}; 98th percentile of the Ontario Typical Range).

Given the above information, risk assessments involving arsenic are likely to produce quantitative health risk estimates (including both cancer and non-cancer effects) in excess of the acceptable level of risk set by a regional regulator. It is apparent that a variety of "tools" such as risk assessment, community health status and/or urinary arsenic studies must be used to in order to capture the true context of arsenic-related health risks (see *A Weight-of-Evidence Approach*, below).

ILCR estimates for inorganic arsenic represent the additional (or incremental) lifetime cancer risk resulting from the predicted lifetime average daily exposure to inorganic arsenic from all site-specific exposure pathways. It should be recognized that only those exposure pathways specific to each community of interest were included in the derivation of ILCR estimates. The collection and subsequent analysis of all environmental media samples produced chemical-specific concentrations in various environmental media which were used to facilitate exposure and human health risk predictions.

Lifetime CRLs associated with Sudbury-specific inorganic arsenic exposure in all COI ranged from 9.6×10^{-5} to 2.6×10^{-4} (*i.e.,* between approximately one and three-in-ten thousand), for CTE and RME scenarios, respectively (Figure 5.4). Lifetime cancer risk estimates for a female composite receptor, under typical Ontario conditions, were between five- and-six-in-one hundred thousand (*i.e.,* 5.5×10^{-5} and 6.3×10^{-5} for CTE and RME scenarios, respectively). As shown in Figure 5-7, inorganic arsenic exposures *via* drinking water and incidental soil/dust ingestion in Falconbridge were greater than those experienced by typical Ontario and Hanmer female composite receptors.

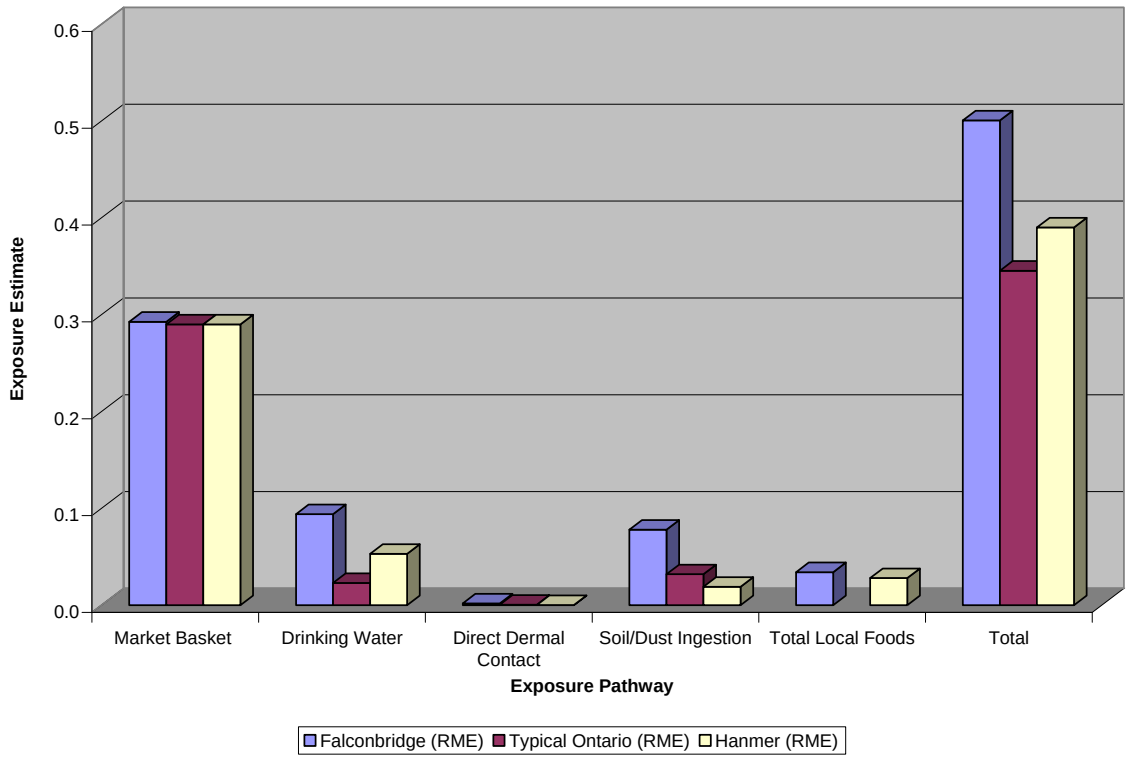

Figure 5-7 Arsenic Exposure Estimates by Exposure Pathway for the Female Toddler Receptor – RME Scenario

As previously mentioned the "total local foods" exposure pathway applies only to Sudbury-specific COI. The "market basket" risks for typical Ontario are slightly lower than those of Sudbury-specific COI because a proportion of an individual's diet (living within a COI) has been apportioned to locally derived foods.

Although lifetime cancer risk is typically the end-point of interest when assessing the human health implications of inorganic arsenic exposure, non-cancer end-points also exist and a reference dose (RfD) has been recommended by U.S. EPA. Therefore, non-cancer health risk estimates associated with exposures to inorganic arsenic were also calculated. The HQ estimates for the female preschool child living in Falconbridge were between 1.4 and 1.7 for the CTE and RME scenarios, respectively. HQ estimates in Falconbridge were slightly higher than those predicted using typical Ontario assumptions for the CTE and RME scenarios.

As illustrated in Figure 5-7 and Tables 5.4 and 5.5, the difference in the female preschool child's exposure to inorganic arsenic between Falconbridge and typical Ontario conditions can be attributed to two exposure pathways: drinking water and incidental soil/dust ingestion. This difference in exposures

can be attributed to the differences in exposure point concentrations (EPC) observed in drinking water and soil between Falconbridge and the typical Ontario scenario.

If the EPC for arsenic in Falconbridge soils (*i.e.,* the 95% UCL on the arithmetic mean of 188 samples) was reduced from the current 78.6 mg/kg to 17 mg/kg (background for non-agricultural sites in Ontario), the reduction in lifetime cancer estimates (under a RME scenario) would be approximately 12% (*i.e.,* from 2.6×10^{-4} to 2.3×10^{-4}). However, these are only rough estimations of overall improvement to health risks related to potential soil remediation activities, and do not account for any potential improvements observed in some secondary media impacted in some fashion by soil itself (*e.g.*, home garden produce, water, *etc.*). As such, while the linkage between soil and these alternate exposure pathways is uncertain and highly site-specific, any improvements in residential soil concentrations may also ultimately result in an observable decrease in COC concentrations in these other media.

Table 5.4 Estimated Total Daily Intakes for Arsenic – RME Female Receptors – Falconbridge (Average Soil Concentrations)

Exposure Pathway	Environmental Media Concentrations		Average Percent of Total ETDI [a]	Estimated Total Daily Intakes (ETDI) (µg/kg bw/day)					
	Value	Units	Percent	Female Infant	Female Preschool Child	Female Child	Female Adolescent	Female Adult	Female Lifetime
Inhalation of Fine Particulate – Outdoors	0.0024	µg/m^3	0.0%	0.000039	0.000084	0.000061	0.000038	0.000036	0.000076
Inhalation of Fine Particulate – Indoors	0.0024	µg/m^3	0.3%	0.00058	0.0013	0.00090	0.00056	0.00054	0.0011
Dermal Contact – Outdoors	79	µg/g	0.5%	0.0018	0.0017	0.0013	0.0012	0.00036	0.0015
Dermal Contact – Indoors	25	µg/g	0.0%	0.00014	0.00013	0.000097	0.000090	0.000040	0.00012
Soil Ingestion	79	µg/g	6.8%	0.024	0.049	0.0055	0.0034	0.0031	0.016
Indoor dust Ingestion	25	µg/g	4.4%	0.015	0.032	0.0036	0.0022	0.0020	0.010
Home Produced Fruits and Vegetables	0.025	µg/g fw	3.3%	0	0.015	0.011	0.0080	0.0066	0.014
Local Fruits and Vegetables	0.028	µg/g fw	2.6%	0	0.013	0.0089	0.0059	0.0046	0.011
Local Wild Blue Berries	0.0052	µg/g fw	1.0%	0	0.0066	0.0032	0.0016	0.0013	0.0037
Local Wild Game	0.00013	µg/g fw	0.0%	0	0.000023	0.000015	0.000010	8.4x10^{-06}	0.000018
Local Fish	0.00022	µg/g fw	0.0%	0	0.000097	0.00015	0.000083	0.00010	0.00016
Drinking Water	2.6	µg/L	29.1%	0.095	0.099	0.059	0.046	0.062	0.10
Market Basket Contribution	NA	µg/g	51.7%	0.00053	0.29	0.18	0.10	0.062	0.19
SUMMARY									
Estimated Total Daily Intake (µg/kg/day)			**100.0%**	***0.14***	***0.51***	***0.28***	***0.17***	***0.14***	***0.35***
Inhalation Route Only			0.3%	0.00061	0.0013	0.00096	0.00060	0.00057	0.0012
Direct Soil Contact Only			11.8%	0.041	0.083	0.010	0.0070	0.0055	0.028
Market Basket Foods and Drinking Water			80.8%	0.096	0.39	0.24	0.15	0.12	0.29
Local Foods (HGP; Berries; Beef; Dairy; Game; Fish)			7.0%	0	0.035	0.024	0.016	0.013	0.028

1

Table 5.5 Estimated Total Daily Intakes for Arsenic – RME Female Receptors - Typical Ontario (Average Soil Concentrations)

Exposure Pathway	Environmental Media Concentrations		Average Percent of Total ETDI [a]	Estimated Total Daily Intakes (EDI) (µg/kg bw/day)					
	Value	Units	Percent	Female Infant	Female Preschool Child	Female Child	Female Adolescent	Female Adult	Female Lifetime
Inhalation of Fine Particulate – Outdoors	0.0010	µg/m^3	0.0%	0.000016	0.000035	0.000025	0.000016	0.000015	0.000032
Inhalation of Fine Particulate – Indoors	0.0010	µg/m^3	0.2%	0.00024	0.00052	0.00037	0.00023	0.00022	0.00047
Dermal Contact – Outdoors	17	µg/g	0.2%	0.00038	0.00037	0.00027	0.00026	0.000078	0.00033
Dermal Contact – Indoors	18	µg/g	0.0%	0.00010	0.000095	0.000069	0.000064	0.000029	0.000089
Soil Ingestion	17	µg/g	2.2%	0.0051	0.011	0.0012	0.00074	0.00066	0.0035
Indoor dust Ingestion	18	µg/g	4.7%	0.011	0.023	0.0025	0.0016	0.0014	0.0075
Home Produced Fruits and Vegetables	NA	µg/g fw	0.0%	0	0	0	0	0	0
Local Fruits and Vegetables	NA	µg/g fw	0.0%	0	0	0	0	0	0
Local Wild Blue Berries	NA	µg/g fw	0.0%	0	0	0	0	0	0
Local Wild Game	NA	µg/g fw	0.0%	0	0	0	0	0	0
Local Fish	NA	µg/g fw	0.0%	0	0	0	0	0	0
Drinking Water	0.64	µg/L	10.8%	0.023	0.024	0.015	0.011	0.015	0.026
Market Basket Contribution	NA	µg/g	81.9%	0.00053	0.31	0.19	0.11	0.068	0.20
SUMMARY									
Estimated Total Daily Intake (µg/kg/day)			**100.0%**	***0.041***	***0.36***	***0.21***	***0.12***	***0.086***	***0.24***
Inhalation Route Only			0.2%	0.00026	0.00056	0.00040	0.00025	0.00024	0.00050
Direct Soil Contact Only			7.2%	0.016	0.034	0.0041	0.0027	0.0022	0.011
Market Basket Foods and Drinking Water			92.6%	0.024	0.33	0.21	0.12	0.083	0.23
Local Foods (HGP; Berries; Beef; Dairy; Game; Fish)			0.0%	0	0	0	0	0	0

5.2.1.1 Weight-of-Evidence Approach for Arsenic

Risk assessment of arsenic-contaminated sites is a complex and problematic exercise, the interpretation of which has been a source of controversy and complication when managing these sites. The issue of the cancer potency of arsenic, and the interpretation of and response to predicted risks in excess of the traditional *de minimis* or negligible risk levels of one-in-one-million has complicated issues surrounding the risk assessment and management of arsenic-contaminated sites. Use of the U.S. EPA slope factors to estimate possible risks from arsenic exposures to people through all pathways (air, water, food, soils) results in high predicted risk values from background (natural) sources. In Ontario, consideration of background soil levels (17 µg/g) and generic soil criteria (25 µg/g for residential), reveals risks in the one-in-one-hundred thousand range for the average Ontario resident living in an "uncontaminated" area. This immediately results in problems understanding and explaining what such risk estimates mean. Alternatively, a weight-of-evidence approach has been successfully used at several other sites across Canada and the U.S. [Port Hope (MOE, 1991); Deloro (MOE, 1999); Wawa (MOE, 2001a); Anaconda, Montana (Calabrese, unpublished; Hwang *et al*., 1997; Walker and Griffin, 1998); Balmerton (Gradient, 1985)]. Risk assessments involving multi-pathway exposure assessment (air, water, soil, backyard produce, fish and market basket foods) and use of the USEPA slope factors revealed risk levels in the one-in-one thousand range for many of these sites. In fact, the results predicted for arsenic as part of the Sudbury HHRA are consistent with those obtained at other similar sites.

The unsatisfactory nature of these arguments carries directly across to the discussion of potential health outcomes related to arsenic entering the environment from various human activities. To the risk assessor, the concern is not necessarily focussed on what risks are predicted for the specific population of study, but the risks relative to background or typical populations. In the case of arsenic, risks well above the *de minimis* level are routinely predicted for exposures associated with typical North American diets, and high-quality, regulated North American drinking water supplies. Further investigation into the risk assessment results for communities within the GSA revealed the following: (i) market basket foods and drinking water were the main contributors to arsenic related risks; (ii) generic criteria in Ontario (25 ppm) result in elevated risk levels (greater than one-in-one hundred thousand); (iii) the contribution of soil to overall arsenic related risks was small, and all other pathways were less significant; (iv) health-based intervention levels (remediation goals), as determined by the risk assessment, typically are economically and technologically impossible; and, (v) removal of all soil above the generic criteria would only result in a small overall risk reduction, with the assessment still predicting risks at the generic criteria level. It should be noted that the current Ontario drinking water standard for arsenic is based on treatment

technology constraints, and this standard is currently under review and will likely be revised shortly in both the federal guideline and provincial standards. Once water treatment facilities are required to treat water to a lower standard, a significant source of arsenic exposure will be reduced, thus reducing the background contribution of risk from arsenic exposure.

It is clear that additional information, beyond that typically contained within a risk assessment, is needed to complete the decision making process, incorporating more of a *weight-of-evidence* approach. When considering potential exposures to arsenic from soils, the ionic species of arsenic (typically found in soils) forms insoluble salts with a number of cations and is adsorbed by organic matter, iron and aluminum oxides within the soil. Arsenic thus becomes tightly bound to the soil and very difficult to liberate for biological uptake. Therefore, relatively high levels of arsenic in soil may pose little risk if they are indeed highly insoluble; and therefore, not available for absorption if ingested. In fact, the measured bioaccessibility of arsenic in the GSA soils was approximately 40% (see Section 3.4 for a discussion of the bioaccessibility study conducted on Sudbury soils). A review of the community's health status (see Section 6.8), including cancer incidence in the GSA, has not revealed any elevated incidences of disease (cancer or otherwise) that may be related to arsenic exposure.

The Falconbridge Arsenic Exposure Study (see Appendix I) compared the urinary arsenic levels of individuals from an "impacted" community (Falconbridge) to those from a "control" community (Hanmer). The results indicated that urinary arsenic measurements from the "impacted" community were similar to that of the control, despite the significantly higher soil concentrations present in the Town of Falconbridge (see Section 3.9 for further information regarding the Falconbridge Arsenic Exposure Study). Due to the unique nature of the arsenic sources within each community, the findings in the Falconbridge study cannot be directly applied to other communities within the GSA. However, the arsenic speciation results provide some context for this comparison. Examination of the results of the Speciation Study (see Appendix I for details) clearly shows that the forms of arsenic in the soil, dust and air are consistent between the various communities within the GSA (see Section 3.5). The comparison includes the community of Falconbridge, thereby providing some confidence in the use of the Falconbridge Arsenic Exposure Study across the entire GSA. As such, the results of the Falconbridge Arsenic Exposure Study, which demonstrated no statistical difference in levels of arsenic in urine between Falconbridge and comparison (unexposed) community residents, indicate that arsenic exposure for all resident of the GSA are similar to those in other communities with significantly lower arsenic soil concentrations.

Each of these elements provides strong, complementary lines of evidence to assist in the realistic evaluation of health risks associated with exposures to arsenic. Only after consideration of all pieces of evidence (*i.e.*, the risk assessment, a review of the scientific literature, community health status, urinary arsenic study, and speciation study), and the relative strength-of-evidence associated with each of these elements, is it possible to conclude that there are no unsafe exposures or increased health effects associated with the observed arsenic levels in the GSA.

To summarize, the results of the weight-of-evidence evaluation strongly indicate that there are no unsafe exposures or increased health effects associated with arsenic levels within the GSA because:

- There was no statistical difference in levels of arsenic in urine between Falconbridge and the comparison (unexposed) community;
- Overall predicted exposures for arsenic in all GSA communities were only slightly greater when compared to estimates for the typical Ontario resident;
- Market basket foods were the main contributor to arsenic related risks for both the typical Ontario resident and the typical GSA resident;
- The epidemiological review of cancer incidence and mortality data in the GSA (refer to Section 6.7 in this Volume) found that, for many potential arsenic related cancers, no incidence or mortality rate was high enough to warrant more detailed analysis of the statistics (though sufficient data and analysis is lacking for some forms of cancer); and
- The results of the speciation analysis indicated that the forms of arsenic in the soil, dust and air are consistent between the various communities within the GSA.

5.2.2 Cobalt

The HQ_{avg} estimates for the female preschool child associated with cobalt exposure (*via* the oral and dermal routes) were less than 1.0 and did not appear to differ between COI or the typical Ontario scenario (Figure 5-2). Predicted exposures of female preschool children to cobalt under RME receptor exposure assumptions in COI and the typical Ontario scenario were less than 10% of the recommended RfD of 10 µg/kg/day (HQ_{avg} estimates for both Copper Cliff and typical Ontario were less than 0.2). Figure 5-8 shows that approximately 91% of the ETDI of cobalt (for a female preschool child residing in Sudbury Centre) was a result of consuming market basket foods while 6% can be attributed to site-related soils and local fruit/vegetable consumption.

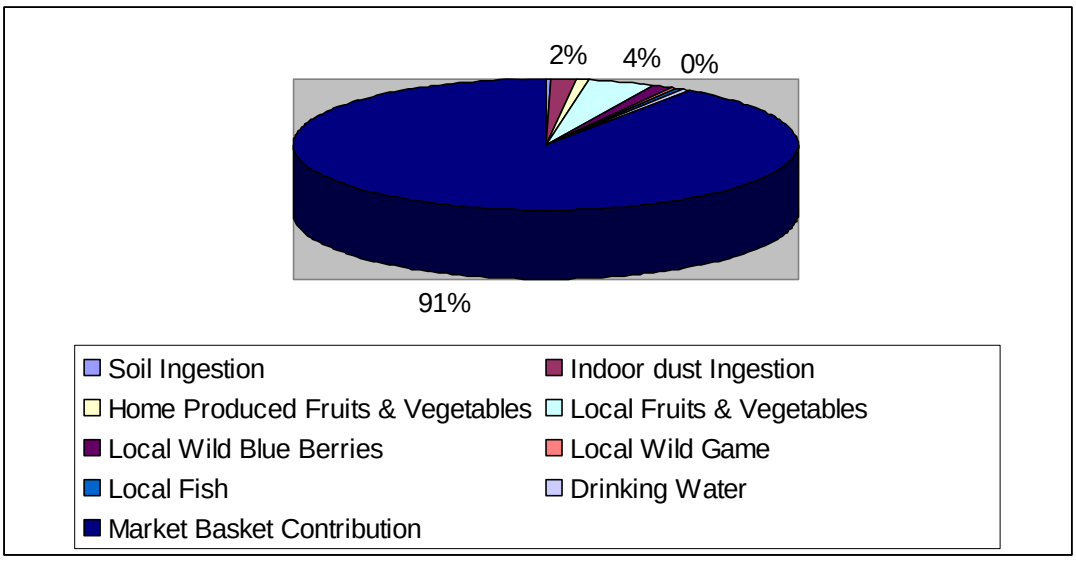

Figure 5-8 RME Estimated Pathway Contribution – Female Preschool Child – Sudbury Centre - Cobalt

As noted previously in Figure 5-3, all non-cancer inhalation HQ estimates for female preschool children were less than 1.0 for cobalt at all COI. Although the mean cobalt concentration (n=64) in air at the Sudbury Centre location was approximately 3.5- to 7-times greater than other COI and the typical Ontario scenario, all inhalation HQ estimates were less than 10% of the cobalt inhalation TRV.

5.2.3 Copper

Female preschool child HQ estimates associated with copper exposures were less than 1.0 under the RME scenario for all COI and typical Ontario assumptions (see Figure 5-2). Under the RME receptor exposure scenario, HQ estimates of 0.67 and 0.62 were predicted for the female preschool child living in Copper Cliff and typical Ontario, respectively. Central tendency (CTE) HQ estimates for Copper Cliff and typical Ontario female preschool children were 0.56 and 0.53, respectively. According to Figure 5-9, approximately 86% of the ETDI of copper (for a female preschool child) was a result of the consumption of market basket foods; an additional 7% was associated with local drinking water, while 2% of the ETDI was a result of incidental soil ingestion.

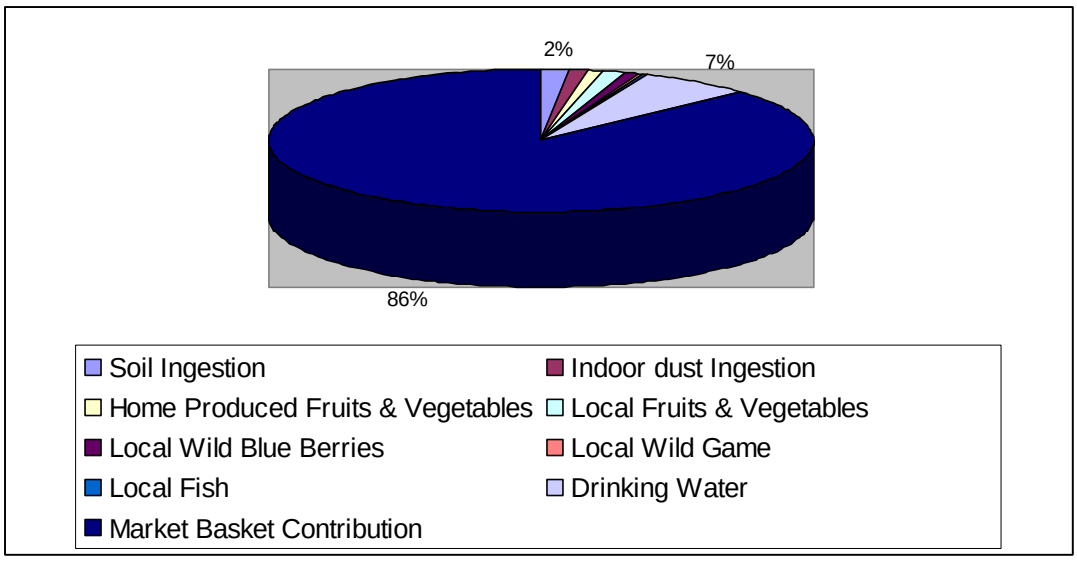

Figure 5-9 RME Estimated Pathway Contribution - Female Preschool Child – Copper Cliff – Copper

5.2.4 Lead

In addition to using the U.S. EPA IEUBK model to assess blood-lead levels in children (refer to Section Chapter 4 and Appendix Q for further discussion), the SARA-specific HHRA model was used to assess the hazards associated with lead exposure, as per other non-carcinogenic compounds. As previously indicated, all lead HQ_{avg} estimates for the general population of female preschool children were below a value of 1.0 (see Table 5-1). The highest HQ_{avg} estimate for lead was 0.94 for a female preschool child living in Copper Cliff under a RME receptor exposure scenario, with a corresponding CTE HQ_{avg} estimate of 0.70. The EPCs for lead in soil, based upon the estimated 95% UCLM concentration, at all Sudbury COI were less than the Ontario Typical Range (OTR) soil value of 120 mg/kg.

Figure 5-10 presents HQ_{avg} estimates for a female preschool child living within an avid hunting/fishing family (includes First Nation members). The scenario assumes that a significant proportion of her diet would consist of local fish and wild game. Exposures *via* incidental soil/dust ingestion were lower than those predicted for the typical Ontario scenario. Results of the assessment indicated that risks predicted for the avid hunter/fisher (including First Nation members) were not significantly higher than those predicted for the general population in Sudbury.

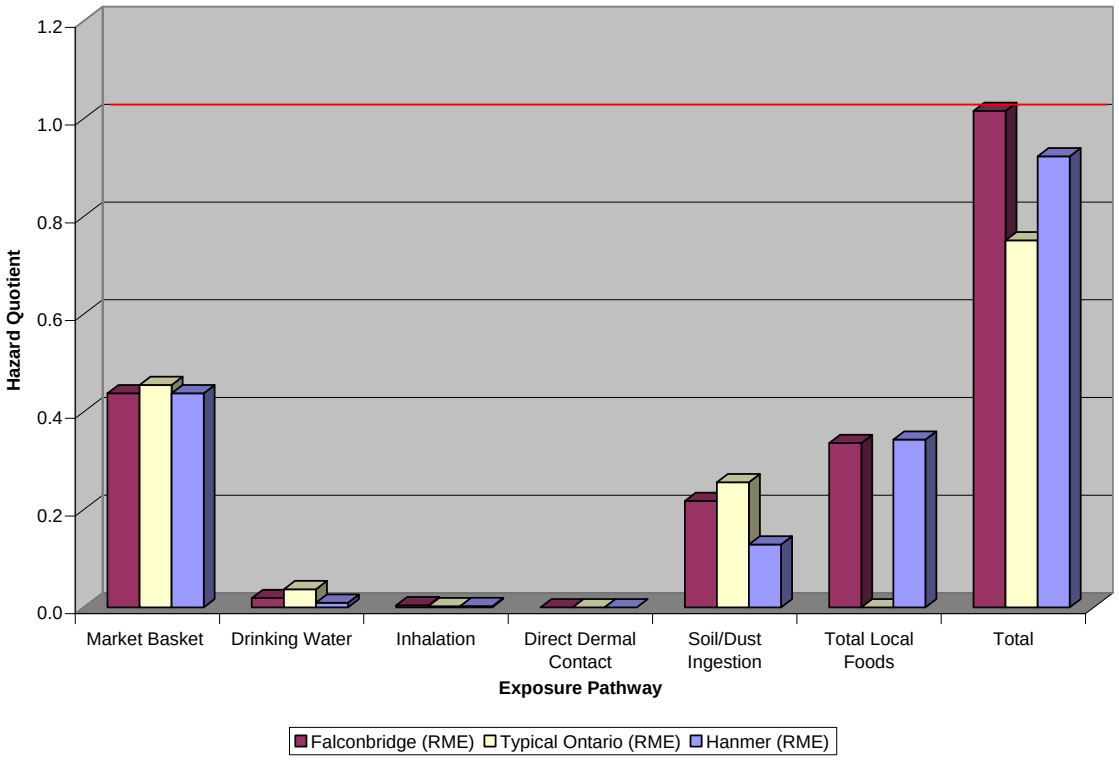

Figure 5-10 RME Hazard Quotients for a Female Preschool Child - Angling and Hunting Population - Lead

As previously discussed, when considering community-wide risks, it is prudent to consider exposures of individuals who may reside in areas that are associated with soil concentration greater than the community average. When maximum soils concentrations of lead in each COI were evaluated the calculated HQ exceeded 1.0 for Copper Cliff (HQ = 1.3), Falconbridge (HQ = 1.1), Coniston (HQ – 1.1) and Sudbury Centre (HQ = 1.1). Based on these results, it was deemed appropriate to derive SRML for lead to identify localized areas where risk management may be required. Section 5.3 discusses the basis of SRML and the SRML derived for lead. Section 5.4 discusses the uncertainties inherent in the derivation of SRML for lead and Section 5.5 discusses the weight-of-evidence approach for lead and provides recommendations with respect to localized areas that may be associated with elevated concentrations of lead in soil.

5.2.5 Nickel

5.2.5.1 Oral Exposure to Nickel

As with lead, all HQ_{avg} estimates for female preschool children (under both the RME and CTE scenarios) were less than a value of 1.0. The highest HQ_{avg} estimate of 0.70 was observed for a female preschool child living in Copper Cliff under the RME scenario (Figure 5-2). The corresponding CTE estimate was 0.52. Figure 5-11 provides a relative comparison of HQ values between Copper Cliff, typical Ontario resident and Hanmer (the regional background site).

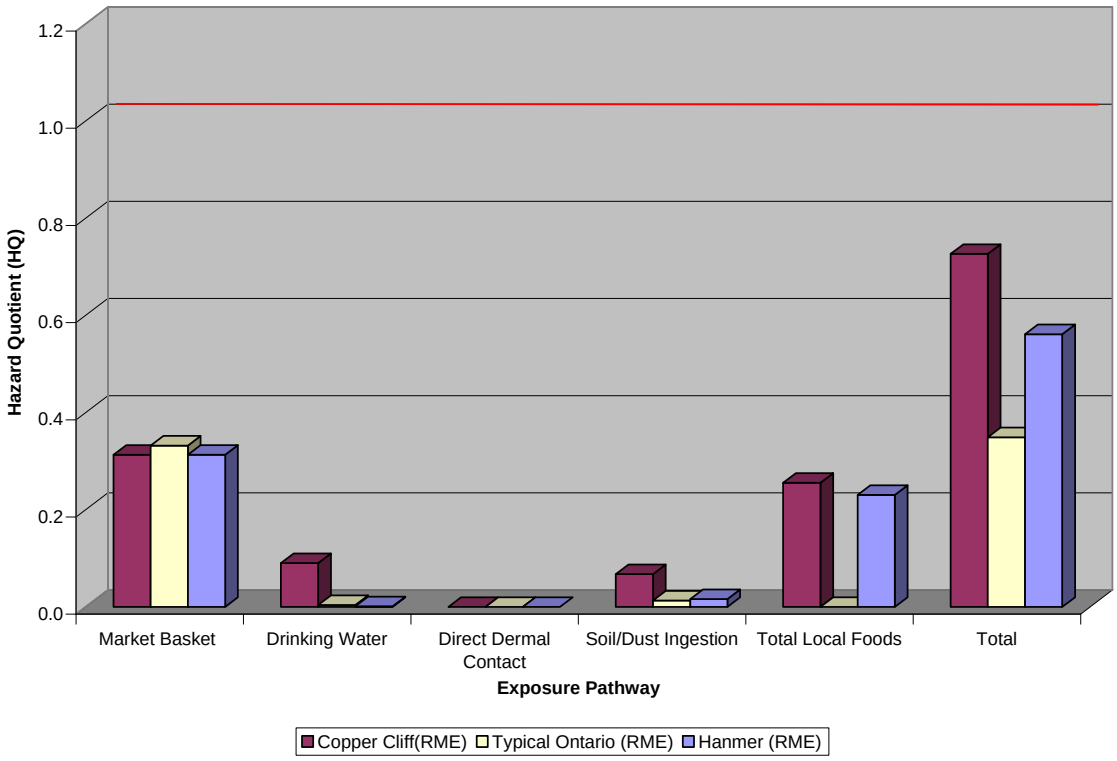

Figure 5-11 RME Hazard Quotients for a Female Preschool Child - Angling and Hunting Population - Nickel

Figure 5-11 indicates that nickel HQ estimates predicted at Copper Cliff (0.70) were approximately twice has high as those predicted for a female preschool child living under typical Ontario conditions (0.35). Exposures *via* drinking water, incidental soil ingestion and diet all appear to be greater in Copper Cliff than in typical Ontario or Hanmer.

As discussed in Chapter 4, "total local foods" include home garden vegetables, local fruits and vegetables, wild blueberries, wild game and fish caught and/or raised within the GSA. Figure 5-12 provides a complete exposure pathway breakdown, reporting the percent contribution each pathway makes to the estimated total daily intake (ETDI) for a female preschool child. Market basket foods and the consumption of local drinking water represent 56% of the ETDI. The consumption of home produced/local fruits and vegetables represents an additional 25% of the ETDI. Together these exposure pathways comprise over 80% of the ETDI to nickel for a female preschool child.

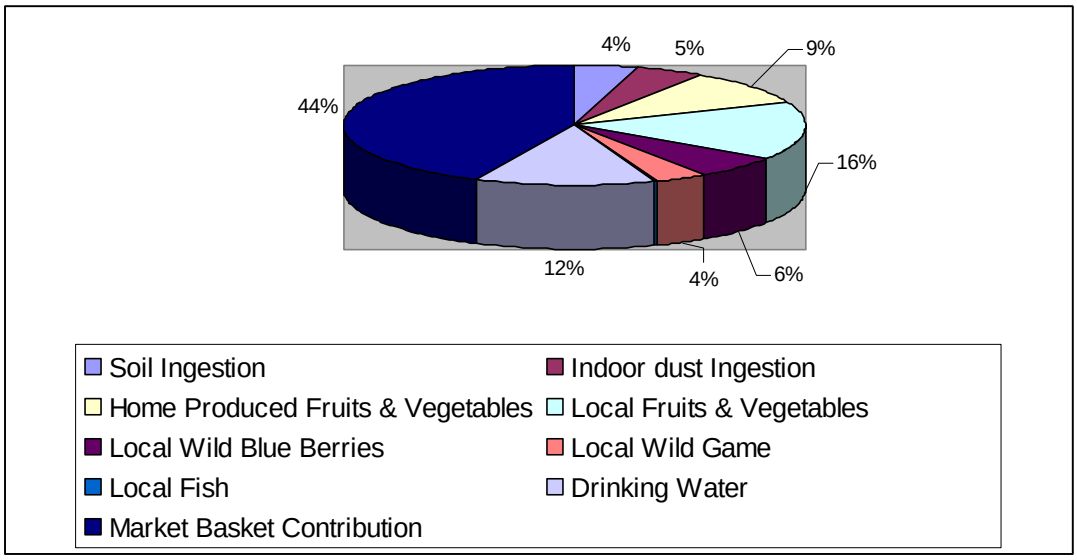

Figure 5-12 RME Estimated Exposure Pathway Contribution - Female Preschool Child – Copper Cliff - Nickel

Under the current scenario, reducing the EPC of nickel in soils in Copper Cliff (976 µg/g) to typical Ontario background levels would only result in a 8.5% reduction in exposure and estimated HQ values for a female preschool child living within Copper Cliff under the RME scenario. As noted previously, these are only rough estimations of overall improvement to health risks related to potential soil remediation activities, and do not account for any potential improvements observed in some secondary media affected in some fashion by soil itself (*e.g.*, indoor dust, home garden produce, *etc.*). As such, while the linkage between soil and these alternate exposure pathways is uncertain and highly site-specific, any improvements in residential soil concentrations may also ultimately result in an observable decrease in COC concentrations in these other media.

5.2.5.2 Inhalation Exposure to Nickel

Potential risks from inhaling airborne COC can be assessed simply by comparing annual average air concentrations in the GSA with the TRV from various agencies and sources. For nickel, these comparisons can be done for both non-cancer and cancer endpoints. While there are a variety of potential TRVs available for assessing risks to various forms of nickel, no one regulatory value provides the perfect TRV for assessing airborne risks to GSA residents. While a variety of valid TRVs for both cancer and non-cancer endpoints were evaluated in the current assessment as part of a weight-of-evidence approach, the inhalation TRV established by the European Union (OJEU, 2005) was ultimately selected as the primary benchmark to evaluate risks related to the inhalation of airborne nickel in the GSA. The remainder of the TRVs included in the overall weight-of-evidence approach are provided for comparative purposes, and demonstrate potential risks for a variety of different endpoints and potential nickel species (many of which are not directly relevant to the current assessment). In addition, the weight-of-evidence discussion provides risk estimates based on the various chemical species of nickel present in the Sudbury air, under a variety of wind conditions.

Primary Risk Evaluation

The European Union (OJEU, 2005) established a nickel TRV of 0.02 $\mu g/m^3$ primarily based upon non-cancer data on respiratory effects (specifically lung inflammation and fibrosis). It is important to note that this TRV is based upon exposure to total nickel in ambient air, and not one particular species or group of nickel species. Based upon the available data, the EU working group also believed that this value is compatible with the objective of limiting excess lifetime cancer risks to not more than one-in-a-million. Table 5.6 provides an evaluation of health risks estimated when one compares total nickel concentrations measured in ambient air at each key monitoring station to this TRV. Note that Sudbury Centre was represented by two monitoring sites (*i.e.*, Sudbury Centre South and Sudbury Centre West).

Table 5.6 Non-cancer Risk Prediction based upon Comparison of Air Concentrations at Various Monitoring Stations with the EU RfC

Monitoring Station	Annual Average Air Concentration	OJEU (2005) RfC	HQ
	µg/m^3	µg/m^3	
Coniston	0.012	0.02	0.6
Copper Cliff	0.059		**2.95**
Falconbridge	0.028		**1.4**
Hanmer	0.012		0.6
Sudbury Centre South	0.017		0.85
Sudbury Centre West	0.26		**13**
TOR	0.0014		0.07

Based upon this approach, potential health risks were noted at the Copper Cliff, Falconbridge, and Sudbury Centre West monitoring stations. These results indicate that further attention should be given to airborne nickel concentrations in the areas surrounding the Copper Cliff and Sudbury Centre West monitoring stations. However, the potential risks around the Falconbridge monitoring station are considered to be negligible given the degree of safety built into the assessment.

The above calculations predict risk for a generic receptor, and represent the typical approach used for regulatory evaluation of airborne risks. A more detailed evaluation was also conducted as part of the Sudbury exposure model calculations which predicted risks for specific lifestages by converting the OJEU (2005) RfC to an RfD with units of µg/kg body weight/day (*i.e.*, multiplying by 20 µg/m^3 and dividing by 70 kg). This calculated RfD was then used to evaluate risks to specific sensitive lifestages with varying inhalation rates and body weights. This more realistic approach resulted in similar, though slightly higher (with Coniston and Hanmer showing HQ values at or slightly higher than 1), risk estimates outlined in Table 5.6 (Figure 5-3). Similar to the assessment of alternative TRVs provided below, this conservative approach provides another element of the weight-of-evidence approach describing potential implications of nickel inhalation in the GSA.

Assessments of Alternative TRVs

As part of an overall weight-of-evidence approach, potential airborne risks can also be evaluated based upon the specific nickel species identified in the year-long air monitoring program carried out in Sudbury as part of this study. As detailed in Section 3.5, the form of airborne nickel in the GSA is very site-specific and dependent on a number of factors, such as proximity to various sources, wind direction, and other meteorological conditions (*e.g.*, wind speed, precipitation, snow cover, *etc*.). Results of the speciation analysis conducted on the air filters from the air monitoring program demonstrated a fairly

consistent nickel species "fingerprint" across the entire GSA, with the exception of one specific localized area. As discussed in Section 3.5, results indicated that when the wind is blowing across the Vale Inco Copper Cliff facility, fugitive dusts appear to give a unique nickel species fingerprint which includes the presence of a small amount of nickel subsulphide at the Sudbury Centre West and Copper Cliff monitoring stations. As a result, the SARA Group (in consultation with a Technical Committee-established speciation task force) developed two specific nickel speciation fingerprints to assist in the evaluation of potential risks in the GSA. These two speciation fingerprints were presented in Section 3.5, and are reiterated in Table 5.7 below.

Table 5.7 Summary of Proposed Nickel Species Fingerprints

Nickel Species	Typical Ambient Fingerprint	Copper Cliff Facility Impacted Fingerprint
Nickel Oxide (NiO)	80%	75%
Nickel Sulphide	10%	10%
Nickel Sub-sulphide (Ni_3S_2)	0%	10%
Nickel Sulphate	10%	5%

Wind in the GSA, on average, originates from a westerly direction 39% of the time and from an easterly direction 61% of the time (also discussed in Section 2.1.1.2). When originating from a westerly directly, ambient air monitors located at the Sudbury Centre West station showed a speciation fingerprint impacted by fugitive dusts from the Vale Inco Copper Cliff facility. Similarly, it is expected that when winds originate from an easterly direction, Copper Cliff air monitors would show a similar fingerprint (refer to Figure 3-1 for a geographic overview of the air monitoring locations in relation to the specific COI). When wind is blowing from the opposite direction and is apparently not affected by fugitive dusts from the Copper Cliff facility, the typical nickel species fingerprint (*i.e.*, absent any nickel subsulphide) is observed.

Table 5-8 provides a summary of some inhalation TRVs available for the evaluation of species-specific nickel-related risks (also outlined in Section 4.2.1 and in detail in Appendix A). It should be noted that the IURs from U.S EPA, WHO and Health Canada considered similar occupational data sets in the derivation of their unit risk values. The range in unit risk values (see Table 5-8) and subsequent cancer risk estimates is a consequence of how each agency interprets the cancer mortality data, as well as the mathematical models used to conduct low-dose extrapolation of the dose-response information. The occupational cohorts utilized by the U.S. EPA, WHO and Health Canada to derive their unit risk factors were developed for nickel refinery dust which contains varying percentages of oxidic, sulphidic, and

soluble forms of nickel, as well as concurrent exposures to a myriad of other chemicals. Conversely, the Seilkop (2004) unit risk values were based on controlled animal studies, which subjects groups of rats and mice to varying levels of oxidic or sulphidic forms of nickel.

Table 5.8 Summary of Species-Specific Inhalation Unit Risks

Type of TRV	Source of TRV	IUR
IUR	Seilkop (2004) – NiO	2.3×10^{-5} $(\mu g/m^3)^{-1}$
	Sielkop (2004) - Ni_3S_2	6.3×10^{-4} $(\mu g/m^3)^{-1}$
	U.S. EPA (refinery dust)	2.4×10^{-4} $(\mu g/m^3)^{-1}$
	U.S. EPA (subsulphide)	4.8×10^{-4} $(\mu g/m^3)^{-1}$
	WHO	3.8×10^{-4} $(\mu g/m^3)^{-1}$
	Health Canada	1.3×10^{-3} $(\mu g/m^3)^{-1}$
REL	OEHHA (NiO)	0.1 $\mu g/m^3$
	OEHHA (non-NiO)	0.05 $\mu g/m^3$

The potential risks related to exposures to the typical nickel species fingerprint (*i.e.*, year-round in all of the COI except at the Sudbury Centre West and Copper Cliff monitoring stations; when the wind comes from the east at the Sudbury Centre West station, and when the wind comes from the west at the Copper Cliff station) are summarized in Table 5.9.

Table 5.9 Risk Estimates Based on Exposure to the Typical Nickel Species Fingerprint (no Ni_3S_2 exposure)

Monitoring Station	Nickel Air Concentration ($\mu g/m^3$)			Occurrence Frequency	HQ Estimates			Cancer Risks
	NiO (90%)[a]	Non-NiO (10%)	Total		NiO	Non-NiO	Total	NiO
Coniston	0.011	0.0012	0.012	100%	0.11	0.024	0.13	2.5×10^{-7}
Copper Cliff	0.053	0.0059	0.059	39%	0.21	0.046	0.25	4.8×10^{-7}
Falconbridge	0.025	0.0028	0.028	100%	0.25	0.056	0.31	5.8×10^{-7}
Hanmer	0.011	0.0012	0.012	100%	0.11	0.024	0.13	2.5×10^{-7}
Sudbury Centre South	0.015	0.0017	0.017	100%	0.15	0.034	0.19	3.5×10^{-7}
Sudbury Centre West	0.23	0.026	0.26	61%	1.4	0.32	**1.7**	**3.3×10^{-6}**
TOR	0.0014	-	0.0014	100%	0.014	-	0.014	3.2×10^{-8}

[a] Nickel sulfides which are not Ni_3S_2 were conservatively included in the NiO grouping.

These estimates make the following assumptions:

- Only Seilkop (2004) provides an IUR specific for NiO; therefore cancer risks are estimated using this IUR only;
- HQ estimates are based on the OEHHA (2005) derived RELs;
- Two air monitors (*i.e.*, the Sudbury Centre West and South stations) were used to characterize the air quality for the Sudbury Centre COI. However, nickel concentrations at the Sudbury Centre West location, given its proximity to the Vale Inco Copper Cliff facility, were much higher than those observed at the Sudbury Centre South monitoring station. As such, to avoid underestimating risks in the area surrounding the Sudbury Centre West monitor, as well as overestimating the risks in the remaining Sudbury Centre COI, risks were predicted separately for both Sudbury Centre monitoring locations;
- Total nickel air concentrations were proportioned based on the typical fingerprint for all COI with the exception of TOR, which assumed 100% NiO; and,
- Air concentrations were pro-rated based on wind direction for the Copper Cliff and Sudbury Centre West monitoring locations, as the risk assessment is dependent on whether the typical or facility-impacted nickel species fingerprint is used.

Only the Copper Cliff and Sudbury Centre West monitoring locations experience nickel fingerprints influenced by Vale Inco Copper Cliff facility (*i.e.*, presence of nickel subsulphide). During times when this fingerprint would be expected, the risk estimates are summarized in Table 5-10.

Table 5.10 Risk Estimates Based on Exposure to the Facility-Influenced Nickel Species Fingerprint

Monitoring Station	Nickel Air Concentration (µg/m^3)				Occurrence Frequency	HQ Estimates (OEHHA, 2005)			Cancer Risks (Seilkop, 2004)		
	NiO (85%)	Ni_3S_2 (10%)	Ni Sulphate (5%)	Total		NiO	Non-NiO	Total	NiO	Ni_3S_2	Total [a]
Copper Cliff	0.050	0.0059	0.0030	0.059	61% [b]	0.31	0.11	0.41	7.0×10^{-7}	2.3×10^{-6}	3.0×10^{-6}
Sudbury Centre West	0.22	0.026	0.013	0.26	39% [c]	0.86	0.30	1.2	2.0×10^{-6}	6.4×10^{-6}	8.4×10^{-6}

[a] Cancer risks from NiO and Ni_3S_2 are assumed to be additive due to similar mechanisms.
[b] Wind blowing from east to west, across the Vale Inco facility, impacting the Copper Cliff station 61% of the time.
[c] Wind blowing from west to east, across the Vale Inco facility, impacting the Sudbury Centre West station 61% of the time.

When the risks from the two potential wind-directional nickel species fingerprints are combined, the overall risk estimates for each monitoring station are predicted. These results are summarized in Table 5.11. The shaded rows indicate situations where the risk HQ is > 1, or the cancer risk exceeds one in one million.

Table 5.11 Overall Risk Estimates Based on Ambient Air Concentrations at each of the Evaluated Monitoring Stations

Monitoring Station	HQ Estimates			Cancer Risks		
	NiO	Non-NiO	Total	NiO	Ni_3S_2	Total
Coniston	0.11	0.024	0.13	2.5×10^{-7}	-	2.5×10^{-7}
Copper Cliff	0.51	0.15	0.67	1.2×10^{-6}	2.3×10^{-6}	3.4×10^{-6}
Falconbridge	0.25	0.056	0.31	5.8×10^{-7}	-	5.8×10^{-7}
Hanmer	0.11	0.024	0.13	2.5×10^{-7}	-	2.5×10^{-7}
Sudbury Centre South	0.15	0.034	0.19	3.5×10^{-7}	-	3.5×10^{-7}
Sudbury Centre West	2.3	0.62	2.9	5.3×10^{-7}	6.4×10^{-6}	1.2×10^{-5}
TOR	0.014	-	0.014	3.2×10^{-8}	-	3.2×10^{-8}

The assessment indicates that all HQ estimates were less than 1.0, with the exception of the Sudbury Centre West monitoring station where the summed non-cancer risks were estimated to be approximately three-fold higher (HQ=2.9) than the established benchmark. Furthermore, all cancer risk estimates are less than one-in-one million, with the exception of the Sudbury Centre West and Copper Cliff monitoring stations. At the Sudbury Centre West station, cancer risk estimates are approximately twelve per million, while cancer risk estimates at the Copper Cliff station are approximately 3.5 per million. Therefore, a predicted risk greater than one in a million related to inhalation of nickel in ambient air appears to be restricted to the area surrounding the Vale Inco Copper Cliff facility.

As noted previously, the more conservative IURs put forward by the US EPA, WHO, and Health Canada would not be appropriate for the current risk assessment as they do not account for the correct nickel species present in the ambient air throughout the GSA, and would incorrectly overestimate risk. However, to provide risk managers with additional information as to the magnitude and uncertainty surrounding inhalation cancer risk, Table 5.12 provides a comparison of the calculated cancer risks for these alternate regulatory and suggested IURs (see Chapter 4 and Appendix A5 for a full discussion of these alternate IURs).

Table 5.12 Comparison of Cancer Risk Estimates Based on Alternate IURs

IUR Source	Coniston	Copper Cliff	Falconbridge	Hanmer	Sudbury Centre South	Sudbury Centre West	TOR
Seilkop	2.5×10^{-7}	3.4×10^{-6}	5.8×10^{-7}	2.5×10^{-7}	3.5×10^{-7}	1.2×10^{-5}	3.2×10^{-8}
- NiO contribution	2.5×10^{-7}	1.2×10^{-6}	5.8×10^{-7}	2.5×10^{-7}	3.5×10^{-7}	5.3×10^{-6}	3.2×10^{-8}
- Ni3S2 contribution	-	2.3×10^{-6}	-	-	-	6.4×10^{-6}	-
U.S. EPA (Refinery dust)	2.9×10^{-6}	1.4×10^{-5}	6.7×10^{-6}	2.9×10^{-6}	4.1×10^{-6}	6.2×10^{-5}	3.4×10^{-7}
WHO	4.6×10^{-6}	2.2×10^{-5}	1.1×10^{-5}	4.6×10^{-6}	6.5×10^{-6}	9.9×10^{-5}	5.3×10^{-7}
HC	9.6×10^{-6}	4.7×10^{-5}	2.2×10^{-5}	9.6×10^{-6}	1.4×10^{-5}	2.1×10^{-4}	1.1×10^{-6}
Diamond (POD 0.01)	2.9×10^{-6}	1.4×10^{-5}	6.8×10^{-6}	2.9×10^{-6}	4.1×10^{-6}	6.2×10^{-5}	3.4×10^{-7}
Zhao (Weibull with POD 0.01)	2.9×10^{-6}	1.4×10^{-5}	6.8×10^{-6}	2.9×10^{-6}	4.1×10^{-6}	6.2×10^{-5}	3.4×10^{-7}
Zhao (Gamma with POD 0.01)	2.9×10^{-6}	1.4×10^{-5}	6.8×10^{-6}	2.9×10^{-6}	4.1×10^{-6}	6.2×10^{-5}	3.4×10^{-7}
Zhao (Multistage with POD 0.01)	4.5×10^{-6}	2.1×10^{-5}	1.1×10^{-5}	4.5×10^{-6}	6.4×10^{-6}	9.6×10^{-5}	5.3×10^{-7}
B. Conard	3.5×10^{-7}	3.3×10^{-6}	8.1×10^{-7}	3.5×10^{-7}	4.9×10^{-7}	1.2×10^{-5}	4.0×10^{-8}
- NiO contribution	3.5×10^{-7}	1.6×10^{-6}	8.1×10^{-7}	3.5×10^{-7}	4.9×10^{-7}	7.5×10^{-6}	4.0×10^{-8}
- Ni3S2 contribution	-	1.6×10^{-6}	-	-	-	4.6×10^{-6}	-

The estimates in Table 5.12 make the following assumptions:

- Only Seilkop (2004) provides an IUR specific for NiO; therefore only the Seilkop based risk estimates incorporate NiO specific risks;
- NiO and Ni_3S_2 related cancers are similar and considered additive;
- U.S. EPA (Ni_3S_2) risk estimates applies the Ni_3S_2-specific IUR to Ni_3S_2 only air concentrations;
- For the Seilkop and U.S. EPA (Ni_3S_2) risk estimates, total nickel air concentrations were proportioned based on the facility impact fingerprint;
- WHO, HC and refinery dust risk estimates apply these IUR to the total nickel air concentration, as these IURs are not species specific; and,
- Air concentrations were pro-rated based on wind direction for the Copper Cliff and Sudbury Centre West monitoring locations, as the risk assessment is dependent on whether the typical or facility-impacted nickel species fingerprint was used.

Conclusions of Airborne Nickel Assessment

The results indicate that airborne nickel concentrations in the region directly surrounding the Vale Inco Copper Cliff facility exceed the air quality regulatory benchmark selected by the SARA Group for the current study (*i.e.*, the EU TRV) which results in HQ values > 1.0 at three of the monitoring sites – Copper Cliff, Sudbury Centre West, and Falconbridge (see Table 5.6). While, the predicted risks at the Copper Cliff and Sudbury Centre West stations are of potential concern, it is the opinion of the SARA Group that the potential risks around the Falconbridge monitoring station are marginal given the degree of safety built into the assessment.

A similar outcome is produced if airborne risks are calculated using the detailed Sudbury exposure model by converting the EU regulatory RfC ($\mu g/m^3$) to an equivalent RfD (µg/kg body weight/day), and evaluating exposures to each assessment lifestage (refer to discussion in Section 5.1.1.2). Though not the typical approach used in the regulatory evaluation of inhalation risks, this alternate approach does provide a realistic, receptor-specific quantification of risk.

Finally, the assessment of various alternative endpoint TRVs for nickel inhalation also resulted in risk predictions exceeding one in a million in the areas surrounding the Vale Inco Copper Cliff facility (see Table 5-11).

It should be noted that there is uncertainty involved in evaluating inhalation risks based upon a number of the IURs listed above. For example, there is a considerable amount of uncertainty surrounding the appropriateness of the Seilkop IUR for use as a regulatory benchmark (*i.e.*, while it has been published in the scientific peer review literature, it has not been accepted by a reputable regulatory agency). Furthermore, the speciation fingerprint developed for the current assessment is based upon only one year of sampling, and as such is limited by the size of the dataset. Risk managers should take into account these uncertainties (and those listed in Chapter 7) to evaluate the relative strength-of-evidence for each element of the weight-of-evidence evaluation.

The above results weight-of-evidence evaluation indicates the calculated risk to airborne nickel exceeds regulatory benchmarks for both cancer and non-cancer health effects in the community of Copper Cliff and western end of Sudbury Centre. This information, as well as other elements of the weight-of-

evidence evaluation, can be used as a basis to make informed risk management decisions on addressing potential health risks related to airborne nickel in the GSA.

5.2.6 Selenium

As illustrated in Figure 5-2, predicted exposures of female preschool children to selenium in the GSA exceeded the recommended RfD under both the CTE and RME scenarios, resulting in HQ estimates greater than 1.0. Figure 5-13 provides an exposure pathway analysis and a relative comparison between Copper Cliff (the COI associated with the greatest HQ values), Hanmer (regional background) and typical Ontario conditions.

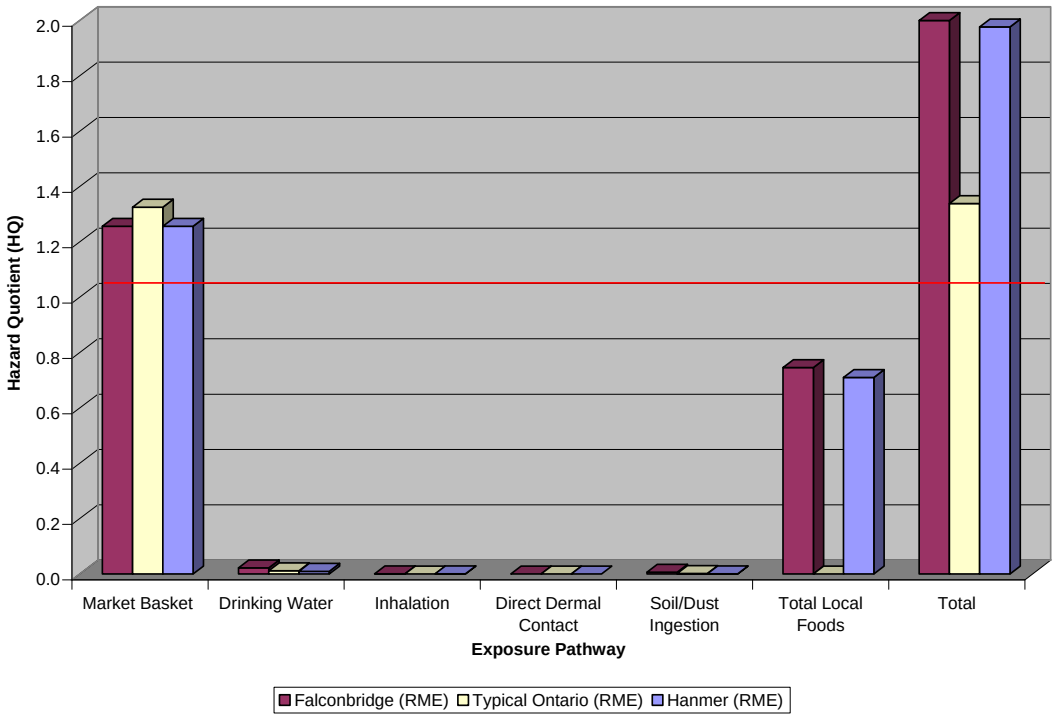

Figure 5-13 RME Hazard Quotients for the Female Preschool Child - Angler and Hunter Sub-Population - Selenium

Figure 5-13 demonstrates that a significant proportion (approximately 75%) of the ETDI of selenium (and hence risk) for the female preschool child resulted from consuming general market basket (or supermarket) foods, even when considering the angler and hunter subgroup. The ETDI from market basket foods alone exceeded the recommended selenium RfD under the RME scenario. Market basket foods are not specific to the GSA and represent the best estimate of daily selenium intake as a result of consuming general supermarket foods in Ontario.

It is noted that the "total local foods" exposure pathway applies only to Sudbury-specific COI. The "Market Basket" HQ estimates for Sudbury-specific COI are slightly higher than those for typical Ontario because a proportion of an individual's diet (living in a COI) has been applied to locally derived foods.

Estimates of exposure to selenium as a result of direct contact with soil and dust for preschool children living in Copper Cliff were approximately ten times greater than those observed for Hanmer (*i.e.*, the regional "background" COI) and four times greater than typical Ontario (background). Exposures resulting from direct soil and dust contact represent less than 2% of the estimated total daily intake of a female preschool child living in Copper Cliff.

The consumption of local foods (including local berries, vegetables, wild game and fish) represented the most significant site-specific pathways for selenium exposure, accounting for more than 20% of the total HQ estimate for the female preschool child in Copper Cliff. Of this 20%, greater than half the exposure could be attributed to the consumption of local wild game.

In summary, the difference in total HQ estimates observed between female preschool children in Copper Cliff and Typical Ontario can be attributed to the consumption of local foods, particularly wild game. It is noted that wild game concentrations were modeled based on environmental media concentrations taken from Zone 2 (as defined in Volume III). As illustrated in Figure 5-13, reducing selenium soil concentrations in Copper Cliff to levels similar to that of Hanmer or typical Ontario would result in only a marginal decrease in the overall selenium HQ estimate. As such, the health risks to Sudbury residents associated with exposure to selenium would be no different than those observed in other parts of Ontario or the rest of Canada.

5.3 Development of Soil Risk Management Levels (SRML)

When considering community-wide risks, it is prudent to consider exposures of individuals who may reside in areas that are associated with soil concentration greater than the community average. As discussed previously, the lead HQ_{max} estimate of 1.1 in Coniston, Falconbridge, and Sudbury Centre exceeded 1.0 when the maximum concentration of lead in soil (310, 335 and 310 µg/g, respectively) was used in the estimate of risk. Similarly, the use of the maximum concentration of lead in soil (582 µg/g) at Copper Cliff resulted in an HQ_{max} estimate of 1.3 for the general population of female preschool children. Refer to Table 5.16 for lead soil concentrations and risk predictions for each COI. While these predicted risks are only marginally above the established HQ benchmark, it was considered appropriate to derive a COI-specific soil risk management level (SRML) for lead to ensure the protection of receptors in locally impacted zones.

A preliminary remediation goal (PRG) or, in the case of the current assessment, SRML can be defined as the average COC soil concentration within an exposure unit (EU) that corresponds to an acceptable level of risk (U.S. EPA, 2001a). In other words, the SRML is the exposure point concentration (EPC) in soil within a given EU (*i.e.,* a community of interest) which would yield an acceptable level of risk.

Given the available data, the SARA Group considered it appropriate to use a weight-of-evidence approach in the evaluation of health risk estimates and the development of SRML.

5.3.1 Weight-of-Evidence Evaluation for Lead

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

- Risk predictions from the Sudbury Exposure Model for each of the COI;
- Information regarding the uncertainties in the model derived values;
- The empirical relationship between lead in soil and blood lead level (BLL) reported in the literature and how this information has formed the basis for SRML values derived at other sites; and
- An evaluation of the selected SRML in both the Sudbury Exposure Model and the U.S. EPA IEUBK model to determine the level of estimated risk posed by soil concentrations at the SRML.

The following section provides an overview of the lines of evidence used to establish the recommended SRML value for lead in the GSA.

Sudbury Exposure Model Results

As one line of evidence in the weight-of-evidence approach, the Sudbury exposure model was used to estimate potential site-specific lead SRML values which would be protective of human health. However, the Sudbury exposure model uses linear regression models to describe the natural-log of indoor dust concentrations as a function of the natural-log of co-located yard soils. However, the relationship between indoor dust and outdoor yard soil is not linear and therefore, back calculation methods could not be used to generate site-specific SRML.

The U.S. EPA (2001a) recommends the use of iterative forward calculation methods when generating SRML with non-linear parameters. The iterative forward calculation method outlined by the U.S. EPA (2001) was used to generate SRML for this study. This method involves collecting data from multiple model runs. Each run uses a different EPC in soil. The HQ (or ICRL) values *versus* EPCs in soil can then be plotted and linear trend lines developed to express HQ (or ICRL) as a function of EPC in soil. Alternatively, the Solver tool included in MS Excel can be used to complete this calculation. The calculation is conducted until the EPC corresponding to an HQ value of 1.0 is determined. This EPC corresponds to the SRML as it indicates the soil level within a specific community, for a specific chemical of concern, which corresponds to an acceptable level of risk (HQ < 1.0).

If the SRML is defined as the EPC (*i.e.,* the 95% UCLM) in soil within a given community which yields an acceptable level of risk, then it is possible that some residential properties may exceed the EPC. Depending on how the soil concentration data are distributed, it is plausible that the remediation of a number of highly impacted soils within the community could bring the overall EPC for that community below the SRML. If the property or site of concern was a single residential lot, it would be reasonable to assume that an individual would move in random fashion within his or her own residential property. Under these circumstances, a reasonable approach may be to remove highly impacted soil to facilitate the reduction in the EPC of the single property. However, because the exposure units in this study represent entire communities, in which individuals do not move in a random fashion, the remediation of locally impacted zones to reduce the overall EPC for the community is not valid. Hence, the SRML values should be applied to individual residential properties, not necessarily the community as a whole.

As previously discussed, it was deemed necessary to calculate SRML for lead in all COI to ensure protection of locally impacted zones. Table 5.13 provides the community specific SRML mathematically derived for lead. As described above, the SRMLs were derived from the results of the HHRA. Also included in the table are site specific soil lead criteria previously established by the U.S. EPA (2001b) and the MOE (2001).

Table 5.13 Soil Risk Management Levels (SRML) for Lead (µg/g)

COI	Model Derived SRML	US EPA SRML		MOE SRML	
		Play area	Bare soil Remainder	Bare play area	Elsewhere on property
Coniston	190	400	1200	400	1000
Copper Cliff	170				
Falconbridge	180				
Sudbury Centre	200				

An examination of the results provided in Table 5.13 indicates that the model-derived SRML (*i.e.*, those based on the assumptions inherent in the HHRA) appear very conservative relative to published guidelines. It is noted that the Canadian Council for Ministers of the Environment (CCME, 1996) provide a soil lead criterion of 140 µg Pb/g of soil; however, this criterion is considered a screening value and not an intervention level. The Ontario Ministry of Environment (Ontario Regulation 153/04) provides a generic soil standard of 200 µg Pb/g soil. Again, this standard is not considered an intervention level but rather used for the purposes of screening.

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

(U.S. EPA) had sufficient confidence in the likelihood of harm that abatement seemed warranted to achieve the associated level of risk reduction."

In March 2001, the MOE (2001) developed a fact sheet related to lead contamination issues. In addition to providing background information related to lead exposure and toxicity, the fact sheet provides a commentary on risks related to soil-borne lead. The fact sheet indicates that there is minimal risk from exposure to soil with lead levels below 200 ppm (µg/g). Furthermore, it indicates that when soil lead levels are greater than 400 ppm in bare soil areas of a child's play area or greater than 1,000 ppm elsewhere on the property, the MOE strongly advises that measures be taken to reduce or minimize the exposure of children. The fact sheet provides guidance on minimizing exposures for children. The fact sheet also indicates that there are minimal risks related to consuming home grown vegetables in soils containing less than 200 ppm lead and that vegetables grown in soil containing greater than 1000 ppm lead should not be consumed.

Model Sensitivity Analyses

As discussed in further detail in Chapter 7, it is useful to conduct a sensitivity analysis to identify how variation in the output of a model (*e.g.*, SRML) is influenced by uncertainty in the HHRA input variables. Table 5.14 contains a number of model scenarios that demonstrate the impact these assumptions have on the model-derived SRML. A change in key assumptions and parameters can have a significant influence on the SRML for lead. Even minor changes to any number of these key parameters can result in significant changes in the calculated SRML. The community of Copper Cliff is used for example purposes only and is not intended to single out this community.

The key input parameters that can be changed include soil ingestion rate, soil to dust ratio, food consumption rate, bioavailability of lead in soil and dust, and the TRV used for comparison. By altering one or more of these parameters the Copper Cliff SRML can vary substantially (-41% to +2200%) (Table 5-14). The range of SRML values demonstrates the sensitivity of the model to the input parameters. It is important to note that the selection process behind the choice of most of these input parameters is largely based on policy, rather than science as the input values in Table 5-14 all have scientific merit.

Table 5.14 Relative Impact to Calculated SRML from Changing Key HHRA Assumptions for the Female Preschool Child *Lead in Copper Cliff*

Scenario	MOE Soil Consumption Rate	Previously Reported Scenario	IEUBK Soil-to-Dust Concentration Ratio	USDA Food Consumption + MOE soil consumption rate	Bioavailability Adjustment	USDA Food Consumption	USDA Food Consumption + Two-Phase Bioaccessibility	Two-Phase Bioaccessibility	Using Health Canada TRV	USDA Food Consumption + Two-Phase Bioaccessibility + Health Canada TRV
SRML (play area bare soils)	100	170	220	250	350	380	930	1100	1700	3900
% Change	59%	-	129%	147%	206%	224%	547%	647%	1000%	2294%
Projected SRML (bare soil remainder)	300	510	660	750	1100	1100	2800	3300	5100	12000
Oral TRV (µg/kg bw/day)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	1.85 (MOE)	3.57 (HC)	3.57 (HC)
Soil/Dust Consumption Rate (mg/day)	100	80	80	100	80	80	80	80	80	80
Food Consumption Database	HC	HC	HC	USDA	HC	USDA	USDA	HC	HC	USDA
Soil-to-Dust Relationship	Regression Equation	Regression Equation	IEUBK Soil-to-Dust Concentration Ratio (0.7)	Regression Equation	Regression Equation	Regression Equation	Regression Equation	Regression Equation	Regression Equation	Regression Equation
Bioaccessibility	Soil = 66% Dust = 83% (one-phase)	Soil = 66% Dust = 83% (one-phase)	Soil = 66% Dust = 83% (one-phase)	Soil = 66% Dust = 83% (one-phase)	Soil = 100% Dust = 100%	Soil = 66% Dust = 83% (one-phase)	Soil = 38% Dust = 43% (two-phase)	Soil = 38% Dust = 43% (two-phase)	Soil = 66% Dust = 83% (one-phase)	Soil = 38% Dust = 43% (two-phase)
Bioavailability	100%	100%	100%	100%	50% (IEUBK)	100%	100%	100%	100%	100%
Percent Change	-41%	-	29%	47%	105%	124%	447%	547%	900%	2200%

Table notes:

- Projected SRML (bare soil remainder) is calculated by multiplying the calculated (play area bare soil) SRML by three-fold to account for the three-fold difference inherent in the two lead soil action levels proposed by the U.S. EPA (400 versus 1200 mg/kg). It is acknowledged that the 3-fold adjustment factor is arbitrary and has limited applicability to the protection of human.
- Food Consumption Database indicates the effect on SRML between selection of food consumption data from the older Health Canada/Nutrition Canada database (*i.e.*, Canadian receptor data) and the more recent USDA data from the Northeastern U.S. (*i.e.*, U.S. receptor data).
- Adjustment of the soil-to-dust relationship has some impact on the calculation of the SRML under the current proposed scenarios because most of the ADI is taken up by non-soil/dust related sources (such as market basket foods). The 95 UCLM of soil lead concentrations in Copper Cliff is 98 mg/kg. If the regression equation developed using data from the indoor dust survey were used, a dust concentration of 150 mg/kg is calculated, resulting in an estimated HQ of 0.93. However, if the IEUBK soil-to-dust relationship of 0.7 is used, a dust concentration of 69 mg/kg is calculated, resulting in an estimated HQ of 0.84. As such, a more than doubling of the dust concentration changed the HQ by only 0.09.
- Two phase bioaccessibility (gastric plus intestinal) was originally utilized in this study as it inherently makes physiological sense. Independent peer review questioned the validity and validation of these values and as such the study now relies on the one-phase (gastric only) results. The impact of utilizing two-phase results is provided for comparative purposes only.

Conservatism of Selected Model Parameters

As discussed in Section 5.4, the parameters and assumptions used in the exposure model have a significant impact on the calculated risk estimates, as well as the derived SRML values. A parameter that has particular impact on model estimates, and is believed to largely drive the conservatism inherent within the exposure model calculations, is the lead bioaccessibility values used for both soil and dust.

Bioaccessibility issues related to lead are discussed in considerable detail in both Section 3.4 and Appendix J. However, a recently published paper (van de Wiele *et al*., 2007) provides a comparison of five *in vitro* digestion models to established *in vivo* experimental results as they apply to lead bioaccessibility in the human gastrointestinal tract. In this multi-laboratory comparison study, the oral bioaccessible lead fraction was significantly different between the *in vitro* methods and ranged for the simulated fasted conditions from 2% to 33% and for the fed conditions from 7% to 29%. These were evaluated *versus* the *in vivo* bioavailability data from the literature of 26.2 ± 8.1% for fasted conditions and 2.5 ± 1.7% for fed conditions.

This comparison of various *in vitro* lead bioaccessibility methods demonstrates the significant variability inherent with the selection of a simulated gastrointestinal model for lead, as well as the implications of fasting *versus* fed exposure conditions. Finally, the soil and dust bioaccessibility values selected for the current Sudbury assessment are considerably higher (66% and 83%, respectively) than any of those estimated by models reported in the van de Wiele *et al*. (2007) study, as well as the *in vivo* bioavailability data from the literature for lead exposures. It should be noted that the *in vitro* bioaccessibility results used in the current study were based upon gastric absorption only (*i.e.*, one phase), and that the two-phase bioaccessibility (*i.e.*, gastric + intestinal) results (38% for soil and 43% for dust) were more consistent with the range of *in vitro* study results summarized in the van de Wiele (2007) paper, as well as the *in vivo* bioavailability data from the literature.

Relationship between Lead in Soil and Blood Lead Levels

Recent scientific literature suggests that exposure to lead may cause adverse neurological changes in children at blood lead concentrations lower than 10 µg/dL. Soil and dust are a major exposure pathways for lead. Therefore, understanding the relationship between soil and dust levels with corresponding blood lead levels in children is important for development of environmental standards. The primary literature was reviewed to identify studies in which an empirical approach was used to investigate this relationship. The empirical approach generates a slope factor (µg/ Pb/dl blood/µg Pb/g soil) based on the correlation between measured soil lead concentrations and the blood lead concentrations in children assumed to be

exposed to the soil (Stern, 1994). Empirical slopes reflect site-specific and study-specific exposure scenarios; therefore, these slope factors may not be generalized unless the factors that mediate soil lead levels and blood lead levels are taken into consideration (Stern, 1994). In addition, the relationship between lead intake and blood lead level is sublinear for higher intake levels (U.S. EPA, 1986); therefore, linear slopes derived from sites with high soil lead levels will underestimate the relationship (Stern, 1994).

The empirical results of the primary literature show that a blood lead level of approximately 5 µg/dl results from exposure to soil containing lead concentrations ranging from 500 to 1,500 µg/g (Angle *et al.*, 1984; Steele *et al.,* 1990; Stern, 1994; Lewin *et al.,* 1999; Johnson and Bretsch, 2002; Mielke *et al*.,2007) (Table 5.15).

Lewin *et al*. (1999) examined the relationship between the concentrations of lead in soil and blood lead levels in children residing near four Superfund sites in the U.S by calculating a slope factor for the dose-response curve of children. The data was taken from concurrent investigations of populations near four National Priorities List sites where smelting and/or mining existed (ATSDR, 1995). In total, there were 1015 measurements of blood lead in children (6-71 months) and lead soil samples from corresponding households. A slope factor was calculated by applying a multivariate linear regression model to double-log transformed soil lead and blood levels. The appropriateness of the regression model and data transformation were verified by statistical tests (Lewin *et al*., 1999). After adjusting for income, education of the parents, presence of a smoker, sex and dust lead, a slope factor of 0.1388 was derived. Strengths of this study include a large sample size, household-specific environmental data, control of covariates, and strong quality control procedures.

Lewin *et al.* (1999) predicted blood lead concentrations in children based on household-specific soil lead concentrations in three models: high-risk, low-risk, and no covariate (Table 5.15). Overall, concentrations of lead in soil ranging from 500 to 1,500 µg/g resulted in blood concentrations ranging from 4.1 to 9.8 µg/dl. The high-risk population encompassed children who were male, and who lived in households with low income and education levels, without air conditioning, and that contained a smoker. For this population, soil concentrations of 500 µg/g and 1,500 µg/g resulted in predicted blood lead levels of 8.4 µg/dl and 9.8 µg/dl, respectively. The low-risk population was defined as children who were female, and who lived in households with high income and education levels, with air conditioning, and with non-smokers. In this population, soil concentrations of 500 µg/g and 1,500 µg/g resulted in predicted blood lead levels of 4.1 µg/dl and 4.9 µg/dl, respectively. A third model using a regression model without factoring in the covariates yielded predicted blood lead levels of 6.1 µg/dl and 7.6 µg/dl from soil

concentrations of 500 µg/g and 1,500 µg/g, respectively. The blood lead levels from the no covariate model were thought to be over-estimated.

Steele *et al*. (1990) examined thirteen epidemiological studies that investigated the relationship between soil/dust lead and blood lead concentrations in children residing in urban and smelter areas, and in regions near mine wastes from inactive smelter sites in the U.S. Overall, slope factors ranged between 0.76 and 8.1 µg/dl per 1,000 µg/g of soil lead (Table 5.15). The majority of the slopes were calculated by the U.S. EPA (1986) using a basic linear model assuming normal distribution. A fraction of the studies reported slopes that did not take other sources of exposure into account. The slopes from the studies conducted in regions with active smelters and urban areas encompassed the entire range of values reported, and the U.S. EPA (1986) estimated an overall slope value 2 µg/dl per 1,000 µg/g of soil lead. The slopes reported from studies in areas with inactive smelter sites were in the low end of the overall range (0-4 µg/dl per 1,000 µg/g of soil lead), and a mean slope value of 1.7 µg/dl per 1,000 µg/g of soil lead was estimated. The soil concentrations resulting in blood lead levels of 5 µg/dl and 10 µg/dl were calculated using the slope factor range (0.67-8.1) reported by Steele *et al.* (1990). A blood lead concentration of 5 µg/dl corresponds to a soil concentration ranging from 620 to 660 µg/g.

A soil-specific increase in blood lead concentrations in young children exposed to residential soils in the U.S. was derived by Stern (1994). This approach defines an absolute contribution of lead from a single medium (soil), and it is assumed that this concentration will have a uniform effect across an exposed population, independent of other factors such as the blood lead distribution, or lead contribution from other sources (Stern, 1994). The approach employs a mechanistic model which estimates the total change in blood lead concentration from ingestion exposure to soil and soil-derived dust under steady-state conditions, with various input parameters. A slope factor of 10 µg/dl per 1,000 µg/g of soil lead was reported, which results from a blood level of 2 µg/dl in children with soil lead concentrations of 200 µg/g (Stern, 1994). Using this slope factor, soil concentrations of 500 µg/g and 1,000 µg/g resulting in blood lead concentrations of 5 µg/dl and 10 µg/dl were calculated (Table 5.15).

Angle *et al.* (1984) applied a linear model to blood lead data in 1074 children ages 1-18 years in urban and suburban areas on Omaha. Lead sources from house dust, air, and soil were incorporated in the model. A slope factor of 6.8 µg Pb/dl blood/ 1,000 µg Pb/g soil was reported. Soil concentrations of 735 µg/g and 1,470 µg/g were calculated using the slope factor, resulting in blood lead levels of 5 µg/dl and 10 µg/dl, respectively (Table 5.15). von Lindern *et al.* (2003) also utilized a linear regression model to derive a slope factor of approximately 4 Pb/dl blood/ 1,000 µg Pb/g soil. This study encompassed data from the Bunker Hill Superfund Site in Idaho near an abandoned lead/zinc smelting complex. Yard,

neighbourhood, and community soil sources were analyzed in relation to blood lead levels in children, and the overall slope factor derived from the model was additive. Using the slope factor, a blood lead level of 5 µg/dl resulted in a calculated soil concentration of 1,250 µg/g.

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

Johnson and Bretsch (2002) derived a logarithmic model of soil lead concentrations and lead blood levels in children (0-6 years) similar to the non-linear association reported by Mielke *et al. (*2007). Geo-referenced data sets were merged by a geographic clustering method, covering a 3 km^2 area in Syracuse, New York. A highly significant correlation was found in the model, where soil lead values ranged from 50 to 350 µg/g, and blood lead values ranged from approximately 4 to 10 µg/dl. A slope factor was not derived in this study. The range of values reported by Johnson and Bretsch (2002) are similar to empirical results from other studies reviewed that show a blood lead level of approximately 5 µg/dl resulting from exposure to soil containing lead concentrations ranging from 500 to 1,500 µg/g (Table 5-15).

Concentrations of lead in soil less than 500 µg/g were found to result in blood level concentrations of 5 µg/dl in children living near mine waste, inactive smelter sites, and urban areas (Table 5.16). Jin *et al*. (1997) summarized 22 cross-sectional studies of populations in areas with polluted soil and three prospective studies of soil lead abatement trials. Concentrations of lead in soil less than 500 µg/g were found to result in blood level concentrations of approximately 5 µg/dl in children living near mine waste and inactive smelter sites (Table 5.16).

Louekari *et al.* (2004) measured lead blood levels children aged 0-6 years living near a former smelter in Finland, and reported corresponding lead soil concentrations from home yards and day-care centres. Blood lead concentrations ranging from <2.1 to 5 µg/dl (average 2.7 µg/dl) were reported in 10 children living in the most contaminated areas near the former smelter, and corresponding soil lead concentrations ranged from 160 to 434 µg/g (average 242 µg/g). In other areas near the site with lower lead soil

concentrations (15 to 81 µg/g; average 40 µg/g), blood lead concentrations in 42 children ranged from <2.1 and 4.1 µg/dl (average 2.1 µg/dl). In reference areas, soil lead was approximately 20 µg/g, and blood lead levels in children were <2 µg/dl. The blood lead concentrations reported in this study were much less than those measured when the smelter was operational.

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

Ren *et al*. (2006) also found that concentrations of lead in soil less than 500 µg/g resulted in blood level concentrations of approximately 5 µg/dl in children living in urban areas (Table 5.16). The study measured lead in child blood and soils at ten kindergartens in Shenyang, China where lead pollution resulted primarily from automobile exhaust and industry emissions. Concentrations of lead in the soil at kindergartens ranged from 53 to 350µg/g, and blood lead levels in children aged 3-5 years ranged from approximately 1 to 5 µg/dl (Table 5.16). The blood levels were lower in the younger children, with levels ranging from approximately 1 to 1.85 µg/dl in 3-year olds, 1 to 2.3 µg/dl in 4-year olds, and up to a maximum of 5 µg/dl in 5-year olds.

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

Table 5.15 The Relationship Between Lead in Soil (µg/g) and Blood Lead Levels (µg/dl) in North American Populations

Slope Factor (µg Pb/dl blood/ 1,000 µg Pb/g soil)	Receptor	Description	Blood Lead Concentration (µg/dl)	Soil Concentration (µg/g)	Reference
10	Children	Based on residential soil concentrations in the U.S. The calculated slope factor was assumed to be linear.	5	500[d]	Stern, 1994
			10	1,000[d]	
	High Risk Children[a]	Four concurrent investigations of populations residing near four National Priorities List sites in the U.S. A natural logarithm regression was assumed.	8.4	500	Lewin *et al*., 1999
			9.2	1,000	
			9.8	1,500	
	No Covariate Children[b]	Four concurrent investigations of populations residing near four National Priorities List sites in the U.S. A natural logarithm regression was assumed.	6.0	500	Lewin *et al*., 1999
			7.1	1,000	
			7.6	1,500	
	Low Risk Children[c]	Four concurrent investigations of populations residing near four National Priorities List sites in the U.S. A natural logarithm regression was assumed.	4.1	500	Lewin *et al*., 1999
			4.6	1,000	
			4.9	1,500	
	Children	Census tract data from residential communities in New Orleans, LA, 2000-2005. A curvilinear model was utilized.	5	300	Mielke *et al.,* 2007
			5.9	500	
			7.5	1,000	
			8.7	1,500	
6.8	Children	Based on urban/suburban soil concentrations in Omaha. The calculated slope factor was assumed to be linear.	5	735[d]	Angle *et al.,* 1984
			10	1,470[d]	
4	Children	Based on paired blood lead/ soil samples from the Bunker Hill Superfund Site in Idaho. The calculated slope factor is assumed to be linear.	5	1,250[d]	Von Lindern *et al.,* 2003
			10	2,500[d]	
	Children	Aggregation of geo-referenced datasets from Syracuse, New York. A logarithmic regression model was assumed.	4-10[e]	50-350	Johnson and Bretsch, 2002
0.76 – 8.1	Children	Thirteen epidemiological investigations of populations residing in urban towns and towns with operating smelters in the U.S. The calculated slope factors were assumed to be linear.	5	620 – 6600[d]	Steele *et al*., 1990
			10	1,200 – 13,000[d]	

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

[b] A simple no-covariate regression model was used

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

[d] The soil concentrations resulting in a blood lead concentration of 5 and 10 µg/dl were calculated using the slope factor. It was assumed that the calculated slope factors were linear (*e.g.*, [(slope factor /desired blood lead concentration (5 and 10 µg/dl)) = (1,000 µg/g soil / calculated soil concentration (x) (µg/g))]

[e] Levels approximated from graphical representation

Table 5.16 Studies of the Relationship of Soil Lead to Blood Lead in Populations Living Near Mine Waste, Inactive Smelter Sites, and Urban Areas

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

[a] Average, minimum and maximum values are presented
[b] Levels approximated from graphical representation

A blood lead screening study (Decou *et al*., 2001) was commissioned by the Regional Niagara Public Health Department in 2001 to determine exposure to and potential health impacts of lead on Port Colborne, Ontario, and specifically the Eastside Community which had elevated lead concentrations in soils (arithmetic mean of 203 µg/g and a maximum of 1350 µg/g). In total, blood lead was measured in 1,065 individuals, with approximately one-third of all participants from the Eastside Community. The geometric means and their confidence intervals for the blood lead concentrations for all participants were well below the 10 µg/dL screening benchmark, with the geometric mean for the Eastside Community reported as 2 µg/dL. While blood lead results for children were compared to soil lead levels on each particular residential property, no statistical relationship was apparent between the two variables (Decou *et al*., 2001).

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

Health Department continued to recommend limiting exposure to soil containing known contaminants, such as lead (Decou *et al*., 2001).

5.3.2 Selection of Proposed SRML for Lead

The information provided in Tables 5.13 and 5.14 indicate that the model derived SRML (based on the assumptions inherent in the HHRA) appear conservative relative to soil lead values derived for screening purposes by the Ontario Ministry of the Environment and U.S. EPA. The wide range of potential SRML for lead, when input parameters are varied, further indicate that a detailed weight-of-evidence evaluation is required for the SRML development process. The conservative nature of the model-derived SRML has received much scrutiny by the SARA Group and the Technical Committee. As part of the weight-of-evidence evaluation, the SARA Group has taken the following steps:

- Listened to the concerns and recommendations provided by members of the TC;
- Provided the quantitative assessment model to MOE for review;
- Responded to MOE, SDHU, Xstrata and Vale Inco comments and questions;
- Consulted with experts in the relationship between soil lead and blood lead;
- Reviewed the empirical data in the scientific literature; and
- Considered a number of combinations and permutations in the SRML model, presenting the effects of changing "policy"-based and site-specific assumptions.

The empirical approach generates a slope factor (µg/ Pb/dl blood/µg Pb/g soil) based on the correlation between measured soil lead concentrations and the blood lead concentrations in children assumed to be exposed to the soil. Empirical slopes reflect site-specific and study-specific exposure scenarios but provide insight into general trends. A blood lead level of approximately 5 µg/dl results from exposure to soil containing lead concentrations ranging from 500 to 1,500 µg/g. Concentrations of lead in soil less than 500 µg/g were found to result in blood level concentrations of approximately 5 µg/dl in children living in urban areas and near mine waste, inactive smelter sites. The acceptable blood lead level in most jurisdictions is generally considered to be 10 µg/dL in children. However, it is important to note that recent literature suggests that a level approaching 5 µg/dL may be more appropriate. In fact, the MOE now uses a probability of a 5% exceedanc of 5 µg/dL blood lead level as the policy basis of their recently published new lead air standard (MOE, 2007).

Blood lead is a true marker of exposure, eliminating many of the assumptions and uncertainties inherent in the HHRA. Blood lead data is not available for the Greater Sudbury area, and all indications suggest that this HHRA model is conservative (refer to detailed discussions provided in Sections 5.4 and 7.0). Collection of blood lead data in the future would aid in minimizing many of the uncertainties inherent in the assessment.

Based on the weight-of-evidence (*i.e.*, the conservative risk assessment, the strong indication provided in the literature that 500 µg Pb/g soil is a safe level for residential properties, and the previously established regulatory SRML for children's play areas of 400 µg Pb/g soil (U.S. EPA, 2001b; MOE, 2001c)), and the relative strength-of-evidence associated with each of these elements, it is concluded that an SRML of 400 µg Pb/g soil would be appropriate for the GSA.

As the U.S. EPA indicated in the derivation of their lead criteria, consideration of the uncertainty of the scientific evidence regarding environmental lead levels at which health effects would result, a SMRL of 400 µg Pb/g soil provides a sufficient level of protection to minimize the likelihood of harm to human health. The following section provides supplemental model results from both the SARA model and the U.S. EPA IEUBK lead model evaluating the applicability of 400 µg Pb/g soil as an appropriate soil intervention level for the Greater Sudbury Area.

5.3.3 Supplemental Model Results for Selecting SRML

A useful exercise involves the evaluation of the selected soil risk management level in both the Sudbury Exposure Model and the U.S. EPA IEUBK model to determine the level of estimated risk posed by the SRML soil concentrations of 400 µg Pb/g soil.

Sudbury Exposure Model Results

As noted previously, a change in key assumptions and parameters within the Sudbury exposure model can have a significant influence on the model-calculated SRML for lead. Even minor changes to any number of these key parameters can result in significant changes in the calculated SRML. As such, due to the sensitivity of the exposure model, it is useful to evaluate the number of potential assumption scenarios when discussing the establishment of a SRML for lead.

Estimated health risks for exposure to the SMRL soil concentrations of 400 µg lead/g soil were evaluated by the Sudbury exposure model using the selected set of assumptions and parameters used to calculate the model-derived risk estimates and SRML (Table 5.17). For comparative purposes, Table 5.17 also

contains HQ_{avg} (based on average soil concentrations in each of the five COI) and HQ_{max} (based on maximum soil concentrations in each of the five COI) model result. Also provided are soil and dust concentrations corresponding to an HQ of 1.0 (this soil concentration corresponds to the model calculated SRML).

As an aside, if a soil consumption rate of 100 mg/day, rather than 80 mg/day, is utilized in the model calculation, HQ estimates for the SRML scenario (soil concentration of 400 µg/g and a model-calculated dust concentration of 220 µg/g) increase from 1.2 to 1.3.

Table 5.17 SARA and IEUBK Model Results at the Selected SMRL

Parameters	Coniston	Copper Cliff	Falconbridge	Hanmer	Sudbury Centre
HQ_{avg}					
Soil Concentration	52	98	82	19	36
Dust Concentration	127	150	144	98	116
HQ	0.9	0.9	0.9	0.8	0.8
Geometric Mean BLL	2.0	2.5	2.6	1.6	1.8
95th Percentile BLL	4.3	5.4	5.6	3.5	3.9
Probability of exceeding a BLL of 5 µg/dL	2.5%	7.2%	7.7%	0.72%	1.5%
Probability of exceeding a BLL of 10 µg/dL	0.030%	0.17%	0.19%	0.004%	0.014%
HQ = 1					
Soil Concentration	190	167	182	187	201
Dust Concentration	178	173	176	178	181
HQ	1	1	1	1	1
Geometric Mean BLL	3.1	3.1	3.3	3.1	3.2
95th Percentile BLL	6.7	6.7	7.1	6.7	6.9
Probability of exceeding a BLL of 5 µg/dL	16.2%	15.0%	20.0%	16.2%	17.7%
Probability of exceeding a BLL of 10 µg/dL	0.70%	0.60%	0.98%	0.69%	0.82%
HQ_{max}					
Soil Concentration	310	582	335	78.5	309.8
Dust Concentration	203	239	207	142	203
HQ	1.1	1.3	1.1	0.9	1.1
Geometric Mean BLL	4.0	5.6	4.4	2.3	4.0
95th Percentile BLL	8.7	12.1	9.5	5.0	8.7
Probability of exceeding a BLL of 5 µg/dL	30.9%	59.7%	38.4%	4.70%	31.0%
Probability of exceeding a BLL of 10 µg/dL	2.4%	11.0%	3.8%	0.082%	2.4%
SRML					
Soil Concentration	400	400	400	400	400
Dust Concentration	217	217	217	217	217
HQ	1.2	1.2	1.2	1.2	1.2

Table 5.17 SARA and IEUBK Model Results at the Selected SMRL

Parameters	Coniston	Copper Cliff	Falconbridge	Hanmer	Sudbury Centre
Geometric Mean BLL	4.5	4.6	4.7	4.5	4.5
95th Percentile BLL	9.7	10.0	10.2	9.7	9.7
Probability of exceeding a BLL of 5 µg/dL	41.3%	42.7%	45.6%	41.5%	41.4%
Probability of exceeding a BLL of 10 µg/dL	4.5%	4.9%	5.6%%	4.6%	4.5%

IEUBK Model Scenario Results

As discussed in Chapter 4 and Appendix Q, the Integrated Exposure Uptake Biokinetic (IEUBK) model, developed by the U.S. EPA, was used to predict childhood lead exposure and risk. The IEUBK model was also utilized to evaluate several scenarios related to the proposed SRML of 400 µg Pb/g soil. Table 5.17 provides blood lead levels (*i.e.*, geometric mean BLL, 95th percentile BLL) and the probability of exceeding both BLLs of 5 and 10 µg/dL, for the selected SMRL, HQ_{avg}, HQ_{max} and HQ=1 scenarios. The results of this evaluation reveal that default IEUBK assumptions, as outlined in Appendix Q, correspond to estimated average (geometric mean) blood lead levels less than 5 µg/dL, for all scenarios. Upper bound (95th percentile) blood lead level estimates range between 3.5 µg/dL and 5.6 µg/dL at average measured soil concentration in the five COI and between 5.0 µg/dL and 12.1 µg/dL at maximum measured soil concentration in the five COI. Upper bound blood lead level estimates range between 9.7 µg/dL and 10.2 µg/dL at the SMRL soil level (400 µg/g). As discussed elsewhere, blood lead is a true measure of exposure. Since actual blood lead information is not available for the GSA, it is reasonable to assume that actual levels will fall somewhere below those predicted in the IEUBK scenarios considered. It is also of interest to consider the soil concentrations which correspond to a *5% Probability of Exceeding a Blood Lead Level* of 5 µg/dL or 10 µg/dL. The following sections provide this information for the Community of Copper Cliff.

Using a Blood Lead Level of Concern of 10 µg/dL

A soil concentration of 405 µg/g (and a corresponding indoor dust concentration of 217 µg/g) is associated with a 4.984% probability of exceeding a blood lead level of 10 µg/dL for the community of Copper Cliff assuming homogeneous concentrations of lead in environmental media and diet and incorporating a geometric standard deviation of 1.6. This also assumes soil and dust bioavailabilities of 33 and 40%, respectively, and that other environmental media concentrations remain as the EPCs used within the RA.

The geometric mean blood lead concentrations associated with this soil concentration (405 µg/g) are presented in Table 5.18 for the range of infant and child age categories. Figure 5-14 also provides the distribution of blood lead concentrations predicted for children in Copper Cliff at a soil concentration of 405 µg/g. As noted in both presentations, the geometric mean for blood lead concentrations, when exposed to this soil concentration of lead, is 4.6 µg/dL, with the 95th percentile at 10 µg/dL. Thus, a soil lead concentration of 405 µg/g would be considered protective of a 5% exceedance of a blood lead level of concern of 10 µg/dL.

Table 5.18 Predicted Blood Lead Concentrations for Children of Copper Cliff at a Soil Concentration of 405 µg/g

Age Categories (years)	Blood Lead Concentration (µg/dL)
0 to 1	5.2
1 to 2	5.9
2 to 3	5.5
3 to 4	5.2
4 to 5	4.2
5 to 6	3.5
6 to 7	3.1
Geometric Mean	4.6
95th Percentile	10.0

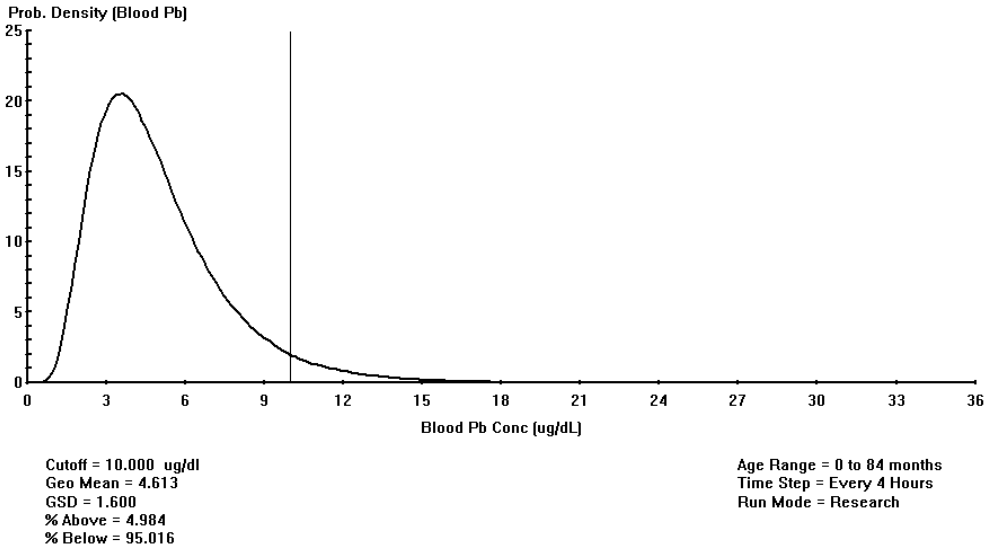

Figure 5-14 Predicted Blood Lead Concentration Range for Children of Copper Cliff at a Soil Concentration of 405 µg/g

Using a Blood Lead Level of Concern of 5 µg/dL

A soil concentration of 75 µg/g (and a corresponding indoor dust concentration of 140 µg/g) is associated with a 5.041% probability of exceeding a blood lead level of 5 µg/dL for the community of Copper Cliff assuming homogeneous concentrations of lead in environmental media and diet and incorporating a geometric standard deviation of 1.6. This also assumes soil and dust bioavailabilities of 33 and 40%, respectively, and that other environmental media concentrations remain as the EPCs used within the RA.

The geometric mean blood lead concentrations associated with this soil concentration (75 µg/g) are presented in Table 5.19 for the range of infant and child age categories. Figure 5-15 also provides the distribution of blood lead concentrations predicted for children in Copper Cliff at a soil concentration of 75 µg/g. As noted in both presentations, the geometric mean for blood lead concentrations, when exposed to this soil concentration of lead, is 2.3 µg/dL, with the 95th percentile at 5 µg/dL. Thus, a soil lead concentration of 75 µg/g would be considered protective of a 5% exceedance of a blood lead level of concern of 5 µg/dL.

Table 5.19 Predicted Blood Lead Concentrations for Children of Copper Cliff at a Soil Concentration of 75 µg/g

Age Categories (years)	Geometric Mean Blood Lead Concentration (µg/dL)
0 to 1	2.7
1 to 2	2.9
2 to 3	2.7
3 to 4	2.5
4 to 5	2.1
5 to 6	1.7
6 to 7	1.5
Geometric Mean	2.3
95th Percentile	5.0

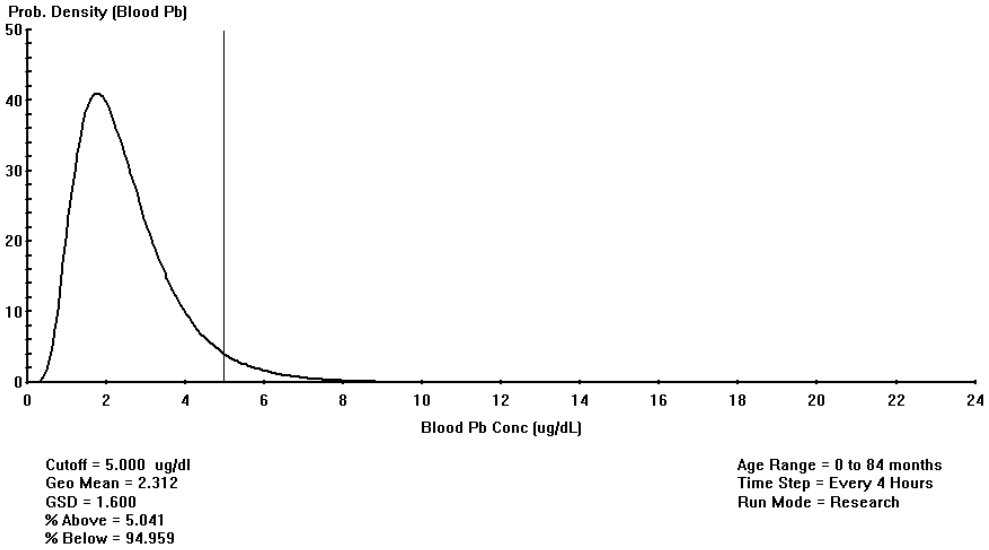

Figure 5-15 Predicted Blood Lead Concentration Range for Children of Copper Cliff at a Soil Concentration of 75 µg/g

5.3.4 Recommended SRML for Lead

Based on the above weight-of-evidence analyses and the relative strength-of-evidence associated with each of these elements, it is recommended that a soil risk management level for lead of 400 µg/g would be appropriate for use in the Greater Sudbury Area. As the U.S. EPA indicated in the derivation of their lead criteria, consideration of the uncertainty of the scientific evidence regarding environmental lead levels at which health effects would result, an SMRL of 400 µg Pb/g soil provides a sufficient level of protection to minimize the likelihood of harm to human health.

As noted previously, blood lead is a true marker of exposure, eliminating many of the assumptions and uncertainties inherent in the HHRA. Collection of blood lead data in the future would aid in minimizing many of the uncertainties inherent in the assessment, and provide further confidence in the selection of an appropriate SRML.

5.4 References

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