APPENDIX T

RESPONSE TO THE IERP COMMENTS ON THE HUMAN HEALTH RISK ASSESSMENT

APPENDIX T: RESPONSE TO IERP COMMENTS ON THE HUMAN HEALTH RISK ASSESSMENT

The following document provides responses to comments made by members of the Independent Expert Review Panel (IERP) (see Appendix S) and/or a description of the revisions made to the human health risk assessment (HHRA) report prepared for Flin Flon, Manitoba and Creighton, Saskatchewan. The numbering of the sections provided below corresponds to comments as presented in Appendix S.

5.1.2 Clarifying Questions

Comment: A panel member requested additional data on air concentrations for COC prior to 2002.

Response: Table 4-2 in Chapter 4 has been revised to include annual average concentrations measured at Ruth Betts School dating back 10 years to 1998. Sampling at the Creighton School did not begin until 2003, therefore, additional data could not be added for this location.

Comment: A panel member suggested that Tables 3-3 and 3-4 list local background concentrations and noted that the screening criteria used for arsenic and cadmium are below background levels found in some parts of the world.

Response: Concentrations representative of Provincial background concentrations have been added to Table 3-4 for comparison to the selected screening criteria and maximum concentrations. It should be noted that the screening criteria for both arsenic and cadmium are higher than the background concentrations.

Comment: A panel member requested that the report clarify the discussion in Section 2.10.1 which indicated that the "majority" of those residents that responded to the local food survey indicated that they only consume the filet portion of local fish.

Response: The results of the local food survey have been reviewed. Four hundred and fifty (450) of 506 respondents indicated that they consume local fish. Of those 450 consumers, 73 (16%) indicated that they consume parts other than the filet. The discussion in Section 2.10.1 has been revised for clarification.

Comment: The conceptual site model (Figure 3-2) needs an arrow from surface water to the receptor to represent ingestion of water while swimming.

Response: Figure 3-2 has been revised as suggested.

5.3.1 Charge Question 1

Charge Question 1: Comment on the adequacy of the data gap analysis and supplemental sampling. Were the appropriate types of data collected and analyses performed that are necessary to assess the extent of contamination? Did they adequately characterize the distribution and concentration of COCs in each of the media of interest?
Comment: Addition of more information about the nature of the data, including sample size and variability was recommended for the tables in Chapter 2. Some tables provide more of the

needed information than others, and it would be helpful to the reader if the authors provide sufficient data in all tables.

Response: Additional information has been added to tables provided within Chapter 2 as suggested.

Comment: Concern was raised over the approach used to eliminate boron as a COC. The analysis of boron within the Jacques Whitford study was for hot water soluble (HWS) concentrations, and it is unclear whether the concentrations reported within the Manitoba Conservation study are for HWS or total boron concentrations. In addition, it is unknown if the U.S. EPA soil criteria selected for the screening process is based on HWS or total boron concentrations. One reviewer suggested using a standard conversion between HWS concentrations and the acid digestible boron concentration. It was also suggested that the authors return to locations where high boron concentrations were found and do some split samples with the two techniques to confirm the ratio between results.

Response: Geoff Jones from Manitoba Conservation confirmed that concentrations of boron reported in their soil study are for total boron. Boron was extracted using the strong-acid leachable metals in soil method (SALM) followed by analysis using Inductively Coupled Argon Plasma Spectroscopy (ICAP), or other specific techniques such as Inductively Coupled Plasma Mass Spectrometry (ICP/MS).

A request for clarification was sent to the U.S. EPA regarding the soil guideline for boron and whether it represented total or HWS concentrations. Dave Crawford of the U.S. EPA indicated that the guideline was for total boron concentrations.

Given that soil guidelines that are protective of human health are based on total boron concentrations, the results of the Jacques Whitford study were converted to total concentrations assuming that the HWS fraction represents 5% of the total concentration as recommended by OMOE (1996) and Gupta (1979). The maximum HWS concentration of 19 µg/g was converted to a total concentration of 380 µg/g, which represents the highest total boron concentration for both studies. Since this concentration is significantly lower than the screening criteria of 3200 µg/g, and boron is not regarded as major component of smelter emissions and is not correlated with known constituents, boron was not retained as a COC for further evaluation.

5.3.2 Charge Question 2

Charge Question 2: Were the appropriate Chemicals of Concern (COC) selected for the communities?

Comment: The panel members recommended that the authors be more transparent in describing the screening of the sampling data to identify the chemicals of concern. Some panel members were not convinced that selenium should be retained as a COC. It was recommended to revise the approach used to exclude aluminum as a COC.

Response: Section 3.2.4 has been revised to provide a more transparent and consistent approach for the selection of COC. It is clearly indicated that those chemicals that were not found at concentrations above the soil criterion in more than 1% of samples were not retained for further consideration as COC.

Although selenium was only found in excess of the soil criterion in 2% of samples, there were a number of samples that were notably higher than the criterion as well as the Provincial background concentrations. In addition, Manitoba Conservation (2007) indicated a strong correlation between selenium and other known constituents of smelter emissions and recommended that further consideration be given to the potential impacts on human health. Therefore, to ensure that public interests related to the potential effects of selenium in soil are addressed in the HHRA, selenium will remain as a COC within the final HHRA.

The draft of the HHRA submitted to the IERP for review excluded aluminum from the COC screening process using a rationale based on its natural abundance within the earth's crust. This approach has been excluded and aluminum has been carried through the screening process including a comparison of concentrations to a selected human health screening criterion as well as provincial and regional background concentrations.

5.3.3 Charge Question 3

Charge Question 3: Does the conceptual model adequately demonstrate the potential human receptors and the related exposure pathways? Do the selected exposure scenarios sufficiently cover the situations, behaviours, and conditions under which receptors are likely to be exposed?

Comment: A reviewer questioned the reasonableness of estimates of food consumption, particularly for the toddler, noting that the local and market basket foods added up to over three pounds of food per day, which seems unreasonable. There is little difference between child and adult consumption rates and alternate sources of data should be considered to generate more realistic estimates, particularly since the toddler is the receptor of interest and market basket foods are such a high proportion of intake. Panel members suggested several other sources for food consumption data, particularly for the toddler. One noted that more recent Canadian fish consumption data are found in a methyl mercury report by Health Canada (2007). The Exposure Factors Handbook (EFH) of the U.S. EPA was suggested by several panellists who noted that this reference uses recent U.S. DA food survey data and so is based on U.S. data, but it might be useful for comparison with the Canadian data.

Response: As suggested by the reviewers, a comparison was made between the market basket food consumption rates recommended by Richardson (1997) as used in the HHRA with those rates recommended within the U.S. EPA EFH. Values taken from both resources represent average per capita intake rates. Values from U.S. EPA EFH were taken from the following tables:

Total Fruits	Table 9-3
Total Vegetables	Table 9-4
Fish and Shellfish	Table 10-19
Meats and Eggs	Tables 11-1 and 11-7
Dairy Products	Table 11-2
Total Grains	Table 12-1

Per capita intake rates recommended by U.S. EPA were in units of g/kg-day. These values were converted to g/day intake rates by multiplying them by the age-specific body weights used in the HHRA (*i.e.*, 8.2 kg for the infant, 16.5 kg for the toddler, 32.9 kg for the child, 59.7 kg for the teen, and 70.7 kg for the adult). U.S. EPA age categories did not directly correspond to

those used by Richardson (1997), therefore, data for multiple age categories were averaged for some categories. For the infant (age 0 to 6 months), the U.S. EPA <01 year age category was used. For the toddler (age 6 months to 4 years; a duration of 4.5 years), a weighted average approach was used for the U.S. EPA <01, 1 to 2, and 3 to 5 year age categories in which the recommended intake rates for each of these three categories were multiplied by 0.5/4.5, 2/4.5, and 2/4.5, respectively. For the child (5 to 11 years), the U.S. EPA 6 to 11 year age category was used. For the teen (12 to 19 years), the U.S. EPA 12 to 19 year age category was used. For the adult (20+ years), the U.S. EPA 20 to 39, 40 to 69, and 70+ age categories were averaged.

Comparison of Market Basket Food Ingestion Rates Recommended by U.S. and Canadian Resources (g/day)										
Food Group	***Infants***		***Toddlers***		***Child***		***Teen***		***Adult***	
	U.S.	***Canada***	***U.S.***	***Canada***	***U.S.***	***Canada***	***U.S.***	***Canada***	***U.S.***	***Canada***
Fruits	123	99	175	179	165	197	131	163	163	168
Vegetables	56	31	123	127	182	212	227	280	267	262
Fish & Shellfish	-	-	5.2	4.7	10	10	14	12	18	16
Meats & Eggs	30	15	78	77	112	115	137	158	141	158
Dairy Products	517	468	462	579	428	591	376	545	247	265
Total Grains	57	37	160	167	211	264	227	280	219	219
Fats & Oils	-	0	-	21	-	37	-	49	-	44
Nuts & Seeds	-	0	-	2.8	-	7.3	-	7.5	-	2.9
Sugars & Sweets	-	30	-	46	-	66	-	71	-	58
Total	783	680	1003	1204	1108	1499	1112	1566	1055	1193

Based on this comparison, the intake rates for most food categories were comparable between the U.S. and Canadian databases, and the total market basket intake rates were similar. For the toddler, the U.S. intake rate (1003 g/day) was approximately 17% lower than the Canadian intake rate (1204 g/day). One reviewer commented that there was little difference between the Canadian intake rates for the toddler (1204 g/day) and adult (1193 g/day) which seemed unreasonable; however, the U.S. intake rates follow a similar pattern (1003 g/day for the toddler and 1055 g/day for the adult). It should also be noted that the fruit intake rate includes fruit juices which are commonly consumed in large quantities by toddlers. Fruit intake is the second largest contributor to the total dietary intake for the toddler.

Although the overall market basket intake rate for the toddler based on the U.S. database was lower than the Canadian intake rate, the U.S. database did not allow for the further breakdown of certain food categories on an age-class basis as was done with the Canadian database. For example, the Canadian database allowed for the separation of nuts and seeds from total grains for each age category. This is an important distinction because concentrations of many COC are significantly higher in nuts and seeds than in other grain products. This allows the concentrations of these food items to be extracted from the derivation of an EPC for total grains. The relatively low ingestion rate for nuts and seeds can be used in combination with the elevated concentrations of COC in these foods to determine a specific and accurate COC intake rate from these items.

Therefore, although the total food ingestion rate for the toddler based on the U.S. database was approximately 17% lower than the Canadian ingestion rate, the COC intake rates associated with the consumption of market basket foods are not similarly affected. Based on this comparison, use of the Canadian market basket ingestion rates was considered to be appropriate and was retained within the final draft of the HHRA.

Comment: The inorganic fraction of arsenic in seafood is addressed in a paper by Schoof and Yager (2007) and includes more recent studies on arsenic. Another panel member asked whether the Schoof and Yager (2007) ratio of inorganic-to-organic arsenic would apply to a range of exposure levels. The first panelist noted that some of the studies used by Schoof and Yager (2007) were from contaminated mining/smelting sites.

Response: The Schoof and Yager (2007) paper describing arsenic content in various fish and seafood items was reviewed. Data was collected from 20 studies from around the world including studies conducted for the purpose of environmental monitoring. The average fraction of total arsenic that is inorganic was found to be 6.8% for freshwater finfish; 1.0% for anadromous fish; 1.1% for marine fish; 1.5% for crustaceans; and 2.0% for molluscs. Within the Draft HHRA, concentrations of total arsenic measured in local fish were converted to inorganic concentrations assuming inorganic arsenic represented 0.6% of the total arsenic measured in freshwater finfish as recommended in Shoof *et al*. (1999). Based on the findings of the Schoof and Yager (2007) paper, the HHRA has been revised to assume that inorganic arsenic represents 6.8% of the total arsenic measured in local fish.

Within the Draft HHRA, concentrations of total arsenic measured in market basket fish and shellfish were converted to inorganic concentrations assuming inorganic arsenic represented 0.2% of the total arsenic measured in these food items. Based on the findings of Shoof and Yager (2007), this may have under-predicted inorganic arsenic exposure *via* this pathway. Using the results of a seafood consumption survey completed by the U.S. EPA (2002), a

weighted inorganic arsenic adjustment factor for market basket fish and shellfish was derived as follows:

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

Therefore, the HHRA has been revised to assume that inorganic arsenic represents 2.4% of the total arsenic measured in market basket fish and shellfish.

Comment: A panel member suggested the authors further consider a nursing infant exposure pathway and scenario for the HHRA and discuss the issue in the uncertainty section. The panellist thought there may be an appropriate approach for this pathway for methyl mercury.

Response: An evaluation of potential exposure and risk associated with exposure to methyl mercury *via* the consumption of breast milk has been added to Section 7.3.1. The results of this assessment indicate that exposure to methyl mercury *via* this pathway is negligible.

Comment: A panel member suggested that the authors be careful in their use of qualitative terms to make sure they are appropriate and internally consistent. For example, on Page 2-40, 500 ml of blueberries is referred to as a small amount, but this is equivalent to about 2 cups, which does not seem small.

Response: The report has been reviewed and the use of qualitative terms has been eliminated wherever possible. For those that remain, the consistency and appropriateness of their use has been evaluated.

The reference to 500 ml of blueberries as being a “small amount” is considered to be appropriate because it is in response to question WB4 from the local food survey *How much local wild berries would your family eat per season?.* This has been clarified on page 2-40.

Comment: An author provided clarification on the wild game consumption estimates, indicating that they used an 8 ounce (oz) serving size based on the serving size for fish recommended by provincial and national agencies. Eight ounce serving size was used for wild game meat, and local fish. Up to 20% (32 grams (g)) of total meat consumed (market basket and wild game) was assumed to be wild game (percentage varied by age group) and it was also assumed that all age groups (excluding infants) ate wild game. A panel member thought that this sufficiently captures the upper bound of exposure, but less so for the average, and suggested explaining this in the text. Another reviewer suggested that if local foods do not have different concentrations than market basket foods, then this breakdown is not necessary, and the breakdown does cause problems for use of the IEUBK model where one would be double counting. An author agreed that this would result in double counting in the IEUBK model runs, but that they wanted the community to see the wild game results. He indicated that they could

compare market basket and local concentrations. A panel member suggested this be explained in the text or a footnote to it.

Response: A discussion has been added to Section 4.1.7.7 indicating that the wild game consumption rates used within the HHRA are representative of an upper bound estimate and that on average, the majority of the population would likely have lower consumption frequencies and would have lower exposure to COC *via* this pathway.

A comparison of the concentrations of COC measured in market basket meat and eggs with those predicted for local wild game is provided in the following table. Although the predicted concentration of inorganic arsenic in local wild game is lower than that in market basket meat and eggs, concentrations of all other COC are notably higher in local wild game.

Comparison of COC Concentrations in Market Basket Meat and Local Wild Game (µg/g) w.w.		
COC	***Market Basket Meat and Eggs***	***Local Wild Game***
Arsenic	0.00046	0.00017
Cadmium	0.015	0.079
Copper	1.0	2.0
Lead	0.0066	0.025
Mercury	0.0011	0.0068
Selenium	0.17	0.37

A comparison of the concentrations of COC measured in market basket fish with those measured in local fish is provided in the following table. Although the predicted concentration of copper and cadmium in local fish are lower than that in market basket fish, concentrations of all other COC are notably higher in local fish.

Comparison of COC Concentrations in Market Basket Fish and Local Fish (µg/g) w.w.		
COC	***Market Basket Fish***	***Local Fish***
Arsenic	0.0049	0.010
Cadmium	0.023	0.0074
Copper	1.3	0.31
Lead	0.0069	0.027
Mercury	0.29	0.45
Selenium	0.31	1.6

A comparison of the concentrations of COC measured in market basket vegetables with those measured in local home garden vegetables is provided in the following table. Overall, concentrations of COC in almost all local vegetables were notably higher than those measured in market basket vegetables.

Comparison of COC Concentrations in Market Basket Vegetables and Local Home Garden Vegetables (µg/g) w.w.				
COC	***Below Ground Vegetables***		***Above Ground Vegetables***	
	Market Basket	***Local***	***Market Basket***	***Local***
Arsenic	0.0043	0.012	0.0093	0.12
Cadmium	0.054	0.051	0.028	0.24
Copper	1.1	1.6	0.90	2.0
Lead	0.0073	0.033	0.0050	0.28

Comparison of COC Concentrations in Market Basket Vegetables and Local Home Garden Vegetables (µg/g) w.w.				
COC	***Below Ground Vegetables***		***Above Ground Vegetables***	
	Market Basket	***Local***	***Market Basket***	***Local***
Mercury	0.00022	0.0025	0.0059	0.0082
Selenium	0.014	0.3	0.016	0.30

Based on the above comparisons, distinguishing exposure to COC from local foods and market basket foods is considered to be appropriate and to provide relevant information to the HHRA.

5.3.4 Charge Question 4

Charge Question 4*: In vitro* bioaccessibility testing was conducted to provide information for the soil ingestion pathway. Were the approach and results valid? Are the recommended relative absorption factors (RAFs) appropriately calculated?

Comment: Panel members suggested the authors present soil concentrations and size fractions with the results and noted that Health Canada has some guidance on how to demonstrate independence of size fraction and bioaccessibility estimates.

Response: Table 2-16 in the Main Report and A-1 in the Bioaccessibility Report (included as Appendix G) contains this information. Further discussion of this issue has been included in the report.

Comment: One reviewer questioned the validity of the comparison of results from the different size fractions and solute:solid ratios in the bioaccessibility summary table (Table 2-16). The reviewer thought the authors were confounding the influence of the two variables by changing them both, and it is not clear which is changing the results. Another reviewer interpreted the table to show that Methods 1 and 2 indicate particle size does not make a difference and Methods 2 and 3 show that the solute ratio makes a large difference. An author explained that they did not intend to use eight different methods, but followed Health Canada guidance for different particle sizes and solute ratios. They tried to do a range finding study to see if one method was most reliable and then just run one method with 50 samples. The reviewers thought that this should be better explained in the report. The text needs to better describe the rationales for the method selections.

Response: The basis of the initial comment is unclear. Table 2-16 includes data for three combinations and permutations of dilution and particle size:

1. 100:1 and <45 um;
2. 100:1 and <250 um; and,
3. 2,000:1 and <250 um.

We concur with the comments of the second reviewer. The report has been clarified to better address this concern.

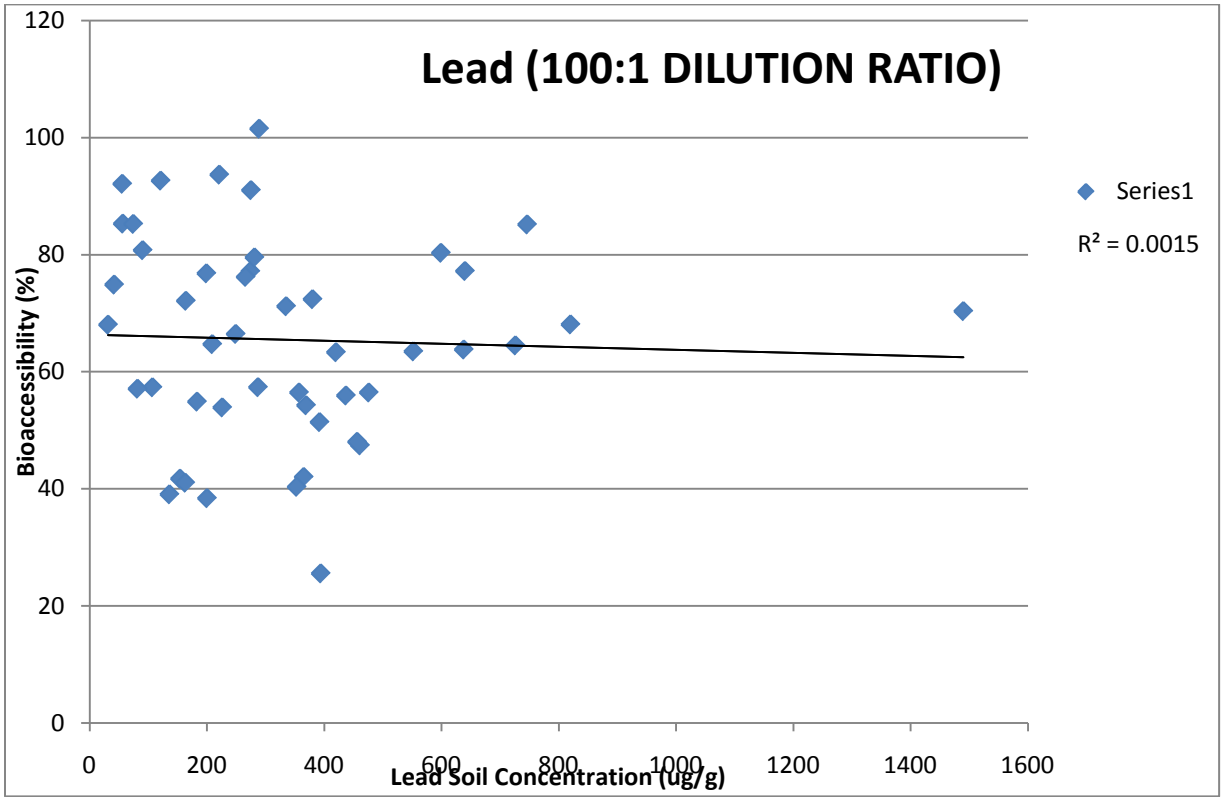
Comment: The panel agreed with use of the 100% bioaccessibility for the COCs other than lead and arsenic. The panel recommended that the authors move much of the bioaccessibility

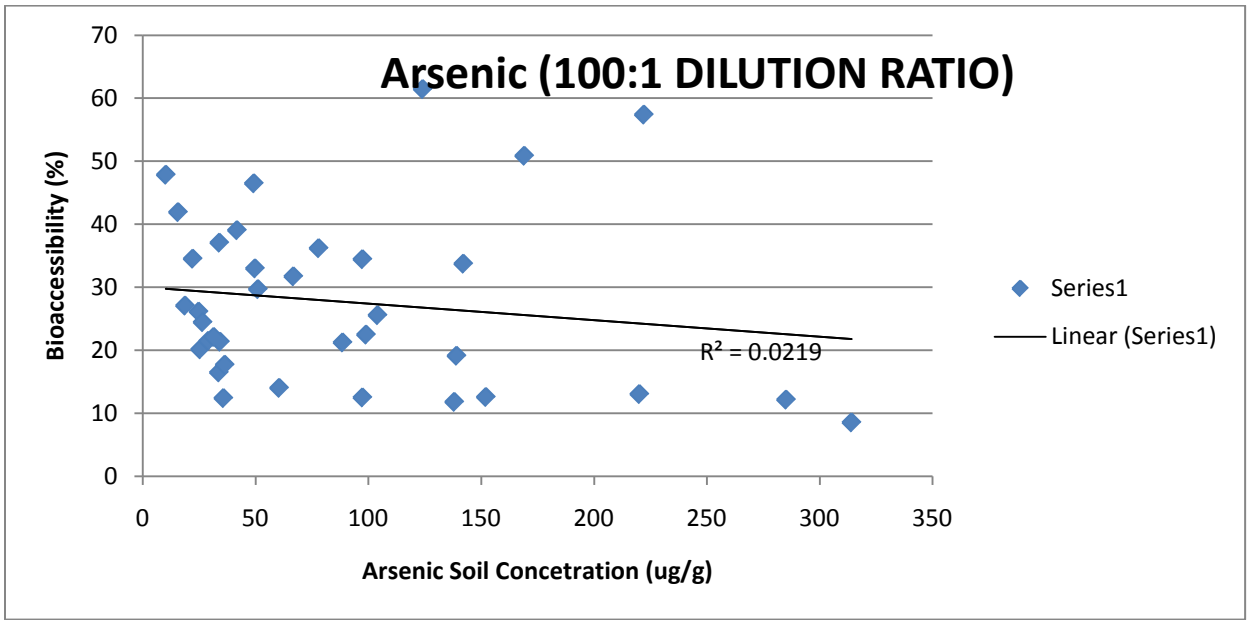
discussion to an appendix and focus the report text on the lead and arsenic testing and results used.

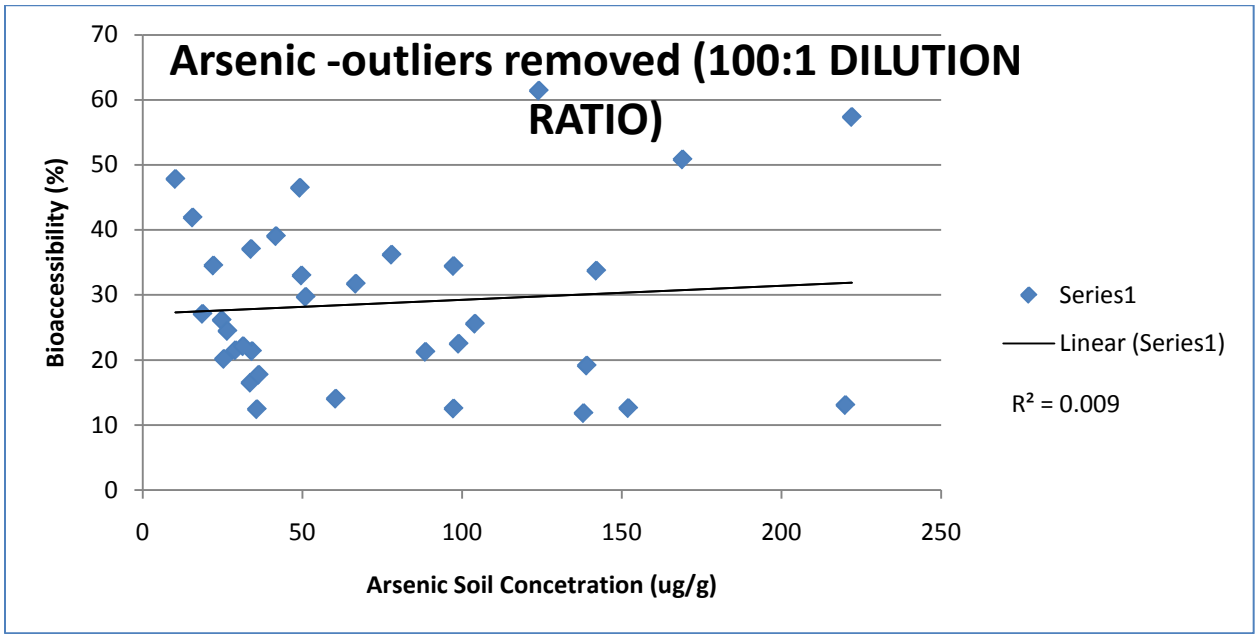
Response: The impact of utilizing the bioaccessibility data for COCs other than lead and arsenic has been included in sensitivity analyses (Section 7). Therefore, this data is pertinent to the assessment and as such has not been moved to an appendix.

Comment: A panelist suggested doing regression analysis to determine if there is a relationship between concentration and bioaccessibility. In response to panelist questions, an author said he thought they eliminated the non-detects, and a panel member agreed that would be the best approach rather than using half the detection limit. Another panel member entered the lead data into a spreadsheet and found there was no relationship between lead concentration and bioaccessibility; the arithmetic bioaccessibility mean was 0.69 compared to the 95% UCLM.

Response: The regression analyses for lead and arsenic (non-detects removed) are provided below. As indicated above, there is no relationship between soil concentration and bioaccessibility for lead and a slight trend for arsenic. The results of removing the two outlier arsenic data points are shown below. These data points have minimal influence on the calculations of average and 95% UCLM values for arsenic bioaccessibility and as such it was felt that these data points should not be removed from the analysis.







Comment: The panel discussed that the arsenic bioaccessibility results do not look reliable at lower concentrations and discussed approaches to address this. Two panelists suggested dropping results from all the samples for arsenic that are below 50 parts per million (ppm) and recalculating the 95% UCLM for arsenic bioaccessibility. They explained that the results from the assay are highly variable and therefore uncertain at concentrations that are close to the detection limit; small differences are significant because the denominator is so small. These

panelists suggested 50 ppm would be a good cut-off based on their personal experience that the method is very sensitive to perturbations at low concentrations.

Response: An analysis of the arsenic bioaccessibility data did not reveal the noted variability at soil concentrations less than 50 ppm. It would appear that the analytical laboratory was able to obtain reliable bioaccessibility analysis at levels much lower than this (as low as 10 ppm). Removal of the data for soils less than 50 ppm significantly weakens the dataset and has a limited impact on the calculation of average and 95% UCLM bioaccessibilities. All non-detect samples were removed from the analysis.

5.3.5 Charge Question 5

Charge Question 5: Are there any concerns or limitations of these studies that affect the usefulness of the data in the HHRA? Do you have any further concerns or comments regarding the problem formulation or supplemental sampling?

Comment: A reviewer questioned the use of 100% relative absorption for lead in house dust and noted that the IEUBK model default is 30%. An author clarified that the TAC asked them to use 100%.

Response: Within the IEUBK model, the default value of 30% for the absolute bioavailability of lead in house dust (ABA_{dust}) was used. Within the spreadsheet model, a value of 100% was used for the relative bioavailability (RBA_{dust}) as recommended by members of the TAC since indoor dust samples did not undergo bioaccessibility testing. The ABA_{dust} is equal to the RBA_{dust} multiplied by the absolute bioavailability of soluble lead ($ABA_{soluble}$) as follows:

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

Therefore, the IEUBK default ABA_{dust} is equivalent to an RBA_{dust} of 60%. This value is nearly equivalent to the RBA_{soil} of 58% measured for Flin Flon soils and used in the HHRA spreadsheet model for predicting exposure to lead in outdoor soil. Therefore, based on this comparison and discussions on this topic during the IERP meeting in Winnipeg, the RBA of 58% measured for soil was used to represent the RBA for indoor dust within the spreadsheet-based calculations. This is consistent with the IEUBK model which indicates that since there is a common source of lead in indoor dust and outdoor soil, a similar bioavailability should exist between these media. All associated exposure and risk calculations have been revised accordingly.

Similar to lead, the HHRA assumed that the RBA for arsenic in indoor dust was 100% since site-specific bioavailability testing was only completed for outdoor soil. To be consistent with the revised approach for lead, the site-specific RBA for arsenic measured in Flin Flon soils (34%) was used to represent the RBA in indoor dust.

Comment: Panel members agreed that the objectives were not clear; in particular they were concerned with Objective 2 – development of a risk management plan.

Response: Agreed, the intention was to develop risk management objectives (referred to as preliminary remediation goals and provisional trigger concentrations) as part of the HHRA. The final risk management objectives and risk management plan (mitigation plan) will be developed

as part of a separate stand-alone summary document. The text in Section 1.3 has been revised to clarify this point.

6.1.1 Clarifying Questions

Comment: A same panelist questioned the adjustment factor for inorganic to organic arsenic and wondered if contamination of the gardens is from aerial deposition, in which case the plant may not be transforming inorganic arsenic to organic arsenic. Mr. Safruk agreed it would be helpful to go back and determine the source of contamination in the study used to derive the inorganic arsenic adjustment factor.

Response: The adjustment factors for converting total arsenic concentrations in food items to inorganic arsenic concentrations were taken from Schoof *et al*. (1999) in which common food items were collected from U.S. supermarkets. These food items are considered to be representative of foods typical consumed in North American market basket items. Although aerial deposition of arsenic in the Flin Flon area has the potential to contribute to the total arsenic concentrations in home garden vegetables, the samples collected as part of the Manitoba Conservation study and used within the HHRA were washed prior to analysis. Therefore, the potential contribution of arsenic from aerial deposition to the total concentration reported for these food items should be minimal. Although it may be difficult to remove all deposited materials from lettuce, the HHRA conservatively assumed that 100% of the total arsenic measured in home garden lettuce was inorganic. For the two other above-ground vegetables (beans and tomatoes), the adjustment factors recommended by Schoof *et al*. (1999) (0.57 and 0.09, respectively), were considered to be appropriate to apply to concentrations measured in washed samples. Adjustment factors for the below-ground vegetables collected in home gardens (0.53 for carrots and 0.29 for potatoes) were also considered to be appropriate for estimating inorganic arsenic content since samples were washed and these vegetables are unlikely to be directly influenced by aerial deposition.

Comment: A panel member questioned the use of the Student t-test rather than a parametric value for the fish data. The authors indicated that they would check this as it may be a software mistake and they generally choose what the statistical software recommends.

Response: It is agreed that the ProUCL recommendation to use the 95% Student's t UCLM for data sets that are not normally distributed does not seem appropriate. This occurred for arsenic, cadmium, and lead in local fish. Data for these chemicals contained a large number of non-detect values. Samples with non-detects were originally assumed to be present at the detection limit. The 95% UCLMs have been re-calculated using ProUCL and by indicating which values represent measured values and which represent non-detect values. The new recommended values are the 95% Kaplan-Meier UCLM for non-parametric data sets.

Comment: Reference to the inhalation of fine particulates in indoor air by the outdoor commercial worker is a typographical error in the title of equation 9.0 in Chapter 4 and should refer to the inhalation of fine particulates in outdoor air.

Response: This error has been corrected.

Comment: Panel members had a number of clarifying questions regarding the mercury exposure estimate. A panel member asked how the authors determined the organic fraction of

mercury in air. Mr. Safruk replied that the fractions of organic mercury in drinking water and air were based on recommendations from the CCME, which he believes are measured mercury data, and that CCME says it is methyl mercury in the air, but he will confirm. He also said that the fraction was also applied to background exposures.

Response: Within the CCME (1996a) background document for the Canadian Soil Quality Guideline for Mercury, CCME assumes that methyl mercury represents 20% of total mercury in air based on ambient air measurements collected by Schroeder and Jackson (1987) in Toronto. This study found that 95% of the total mercury in ambient air id in the vapour phase, of which 76% is elemental and 19% is organic. CCME (1996a) also assumed that methyl mercury represents 25% of total mercury in drinking water based on concentrations measured in river and lake water in the Ottawa valley (Schintu *et al*., 1989).

Comment: A panel member pointed out that a standard fish size was used, but that fish of different sizes and age will have different concentrations of mercury.

Response: For the purpose of predicting exposure through the consumption of local fish, a standard 8 oz. serving per meal was assumed. The concentrations of COC in local fish were based on the 95% UCLM concentrations for all fish collected as part of a local fish survey completed by Stantec and Manitoba Conservation. As discussed in the Stantec (2008) report, fish sampling focused on the collection of sport fish and targeted sizes that would be caught through recreational fishing. Fish were collected by angling as well as gillnets ranging in size from 3" to 6" mesh. Total fish weights ranged from 80 grams to 12,600 grams. It is anticipated that residents will consume fish of varying sizes over a chronic duration, therefore, the derivation of exposure point concentrations based on the fish collected is considered to be representative of a typical long-term exposure scenario.

Comment: A panel member questioned dental amalgams assigned to toddlers, as generally baby teeth are not filled, and recommended reconsidering this.

Response: Within the CCME (1996a) background document for the Canadian Soil Quality Guideline for Mercury, CCME assumes a daily mercury intake from dental amalgam for the child (0.52 µg/day), teen (1.52 µg/day), and adult (2.81 µg/day). Richardson *et al*. (1995) reported that amalgam is considered to be the most significant source of total mercury exposure for receptors aged 5 and older. Although the CCME does not recommend daily exposures from dental amalgam for the infant or toddler, as a conservative measure, the daily intake for the child was used as a surrogate for the toddler within the Draft HHRA. The recommended value was converted to exposure on a per body weight basis for the toddler using the age-specific body weight recommended by Health Canada. Based on discussions on this topic during the IERP meeting in Winnipeg, the daily exposure to the toddler resulting from dental amalgam has been excluded from the HHRA. This approach is consistent with methodology used by the CCME (1996a) to derive the Canadian Soil Quality Guideline.

Comment: The estimate of the amount of blueberries consumed per day per child (4 g/day) was considered excessive by a panel member, but Mr. Safruk explained that this value was based on U.S. EPA's Exposure Factors Handbook for those with berries in home gardens and noted that blueberries are widely available in the area and free for picking.

Response: The local blueberry consumption rate used in the HHRA is the average consumer-only consumption rate of homegrown "other berries" of 0.48 g/kg/day and a standard error of 0.042 (*i.e.*, 0.52 g/kg/day) recommended in the U.S. EPA Exposure Factors Handbook. The HHRA initially assumed that receptors would consume this amount of blueberries 365 days per year. The report has been revised to assume that receptors would consume this amount once per week throughout the year. Therefore, the average daily consumption rate over the course of the year is 0.074 g/kg/day, or 5.2 g/day for an adult. This equates to an annual consumption rate of 1.9 kg/year (or 4.2 pounds/year) for an adult. Receptors of all age categories were assumed to consume local berries at a rate of 0.074 g/kg/day

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

6.2.1 Charge Question 6

Charge Question 6. The authors evaluated the sampling data, and calculated the exposure point concentrations (EPCs) for ambient air, indoor air, drinking water, garden produce, fish, indoor dust, wild game, blueberries, surface water, and snow. Are the selected exposure point concentrations appropriate for the risk assessment?

Comment: The panel recommended that the authors list beside each exposure factor, the statistic used (*e.g.*, 95% of the distribution, 95% UCLM, mean value, or combination) so that it is clear to the reader what the number represents.

Response: This information has been added to the receptor characteristics tables in Chapter 3.

Comment: The panel suggested that the authors calculate the percentage of properties that exceed the PRGs and indicate the magnitude of the exceedence, to put the concentrations in perspective. In addition, the authors could show the hazard quotients in a frequency distribution and avoid identifying individual properties.

Response: Additional figures have been added to Chapter 5 to illustrate the magnitude of the STC exceedances for arsenic, lead, and mercury as suggested.

6.2.3 Charge Question 8

Charge Question 8. Were the best available data used to calculate appropriate background exposure values?

Comment: A reviewer again noted that for arsenic, copper, and zinc, recent data 2005 and/or 2006 (Tables 2-5 and 2-12 of the HHRA) show that the concentrations were higher in the finished drinking water than the raw water, which indicates the potential of contamination by these metals from other sources. Another reviewer noted that the drinking water data seem to suggest that the water distribution systems may contain lead and copper. For example, lead and copper concentrations from the Creighton Distribution System are <0.1 and 3 µg/L, respectively

(Table 2-5). Yet the exposure point concentration terms for lead and copper in Creighton are 3.1 and 124 µg/L, respectively. If this holds true for the other COCs, it appears that a source of these inorganics (*i.e.,* plumbing) has not been discussed in this report. Another reviewer thought it appropriate to discuss and calculate the contribution from other sources to provide perspective, and the authors should be careful about attributing chemicals in the drinking water to the municipal water source and be sure there is evidence that the source has contaminated the drinking water supplies. The panel cautioned the authors to not send mixed messages that a risk manager will find hard to interpret.

Response: Table 2-12 presents surface water concentrations for Douglas Lake (which serves as a raw water source for Creighton municipal water) from 1993. Table 2-5 presents concentrations in Creighton municipal drinking water at the Distribution Centre from 2005/2006. Table 4-6 presents the Creighton drinking water EPCs used in the HHRA which are based on recent HBMS monitoring and the Jacques Whitford study. These results are summarized below for comparison.

Comparison of COC Concentrations in Douglas Lake and in Treated Municipal Drinking Water (µg/L)

COC	*Douglas Lake (1993)*	*Creighton Drinking Water Distribution*		*Creighton HHRA Drinking Water EPCs*
		2005	*2006*	
Arsenic	8	2.2	1.4	2.2
Cadmium	<1	<0.5	0.4	0.89
Copper	<1	13	3	124
Lead	<5	<0.1	<0.1	3.1
Mercury	-	-	<0.05	0.052
Selenium	<1	0.2	0.1	1.1

It is agreed that based on this comparison, it appears that plumbing may be a source of copper and lead to municipal drinking water. Discussion has been added to Section 4.1.1.4.

It should be noted that regardless of the source of COC in drinking water, exposure *via* this pathway needs to be considered in the HHRA to account for all sources of exposure to COC and to allow for the use of an acceptable HQ of 1.0.

Comment: A reviewer thought that consumer products should be accounted for with the other COCs (in addition to lead), while other reviewers thought that the consumer product pathway would not be that relevant for the others. However, all panelists agreed that the consumer product pathway should be discussed qualitatively for the other five COCs (in addition to lead).

Response: A qualitative discussion of the potential exposure to COC from consumer products has been added to Chapter 7.

6.2.4 Charge Question 9

Charge Question 9. Are the selected receptor characteristics and values the most appropriate for use in this assessment? Were the assumptions and exposure input parameters appropriate and were the intake rates calculated correctly?

Comment: Reviewers recommended that the authors standardize the exposure equations; many are missing exposure duration or frequency components and some (*e.g.*, indoor/outdoor air apportionment) are not needed. Concentration units should be used for the inhalation equations to be consistent with the toxicity values.

Response: Exposure equations have been standardized as suggested. Given that indoor air concentrations were assumed to be equal to measured outdoor air concentrations, a single equation is now presented to calculate exposure *via* inhalation.

As explained during the IERP meeting in Winnipeg, the assessment of risk associated with the inhalation pathway was considered through two methodologies. The contribution of exposure *via* inhalation to the total internal dose was calculated using the equations presented in Chapter 4. This was combined with the dose associated with all other pathways for comparison to the TDI. In addition, inhalation risks were calculated through a direct comparison of the air EPC to the RfC or unit risk. It was not considered to be necessary to show the exposure equation for this approach since exposure is simply equal to the air EPC.

Comment: Several reviewers commented on terminology, noting that labelling of parameters as RME (reasonable maximum exposure) was not accurate as the RME describes the individual who receives the highest amount of exposure that can be reasonably anticipated, not the individual parameters themselves. The equations and parameters used to develop an RME risk estimate combine both high-end and central tendency inputs. They suggested adding a column to the tables to indicate what each parameter represents (*e.g.*, percentile of the distribution).

Response: The use of the term reasonable maximum exposure (RME) has been removed from the report. It is now indicated that the exposure scenarios represent a central tendency estimate (CTE). Information regarding the receptor characteristics and parameters have been added to the receptor characteristics tables in Chapter 3.

6.2.7 Charge Question 12

Charge Question 12. Do you have any further concerns or comments regarding the exposure assessment?

Comment: The authors should check the number of significant figures used throughout the document. For example the fraction of inorganic arsenic was reported to one figure and the organic arsenic fraction to two.

Response: It is recognized that for some of the adjustment factors used to convert total arsenic concentrations in food items to inorganic concentrations, the factors are reported as a single significant figure. Most EPCs and receptor characteristics used in the HHRA are reported as two or more significant figures. Although some TRVs contain only one significant figure, most contain two.

Overall, it was decided that the use of two significant figures was appropriate. Reducing exposure and risk estimates to one significant figure would minimize the considerable effort incorporated into the HHRA to accurately characterize these parameters.

Comment: Small errors in the text are distracting and reduce confidence in the assessment. For example, the number of samples for the West Flin Flon area is reported as 77 in some places and 76 in others. Table 5-15 lists different concentrations than reported in the text.

Response: The report has been revised to consistently say that 77 properties were sampled in West Flin Flon. Table 5-15 has been revised to consistently present the PRG derived for cadmium.

Comment: Indicate in the text whether the household water data were first draw or flushed samples.

Response: Discussion has been added to Chapter 4 indicating that drinking water samples are flushed samples.

Comment: Yost *et al*. (2004) corrects values for grapes and watermelon used by Schoof *et al*. (1999).

Response: These values have been revised. It should be noted that Yost *et al*. (2004) indicate that "corrections have negligible impact on findings of Schoof *et al*. (1999)".

Comment: Sieved soil samples are more indicative of what gets on children's hands. 100% is too conservative. Look at data for another estimate that can be derived.

Response: Soil samples were sieved to remove particles greater than 2 mm. While it is agreed that smaller soil particles are more likely to adhere to hands and be available for ingestion, the soil ingestion rate represents the amount of soil ingested per day, regardless of the number of hand-to-mouth events that occur. Therefore, although larger soil particles may not adhere to hands and be ingested, a mass of the smaller particles equivalent to the daily ingestion rate was assumed to be ingested. This methodology has an underlying assumption that concentrations in the fraction that is small enough to adhere to hands are equal to the concentrations measured in the total sample.

Comment: Including the non-detected samples in the averaging of bioaccessibility results is not reliable. One panel member suggested removing the bioaccessibility results from samples with low arsenic concentrations before calculating the average bioaccessibility of arsenic in soil.

Response: All non-detects values have been removed from the data set and the analysis of the bioaccessibility data has been revised. The 95% UCLM for arsenic has been changed to 33% (from 34%) in the revised RA. There were no non-detect values for lead, therefore, the bioaccessibility for lead has not been revised.

7.2.1 Charge Question 13

Charge Question 13. Were the most appropriate exposure limits or toxicological criteria selected for each of the COCs, and are the rationales for the selections defensible?

Comment: A panellist noted that if the focus of the document is on long-term protection, then a chronic assessment is most important. Others agreed and questioned the need for acute values. After some additional discussion, the panel agreed that the acute values are problematic and recommended that the authors use their best professional judgment whether such values were even needed. If they are needed, the authors should clearly present the reasoning for their choices. If no appropriate acute values are available, or determined not to be needed, then the authors should explain this and provide a qualitative discussion.

Response: Agreed, there is a paucity of reliable information regarding acute toxicity benchmarks for use in environmental risk assessments. This section has been revised to incorporate the most relevant data available. Further discussion will enhance this section of the report.

Comment: The panel discussed the oral carcinogenicity estimates for arsenic, noting that there is sufficient controversy with regard to arsenic carcinogenicity that the assessment should address some of the additional data and issues surrounding arsenic. For example, there are other cohorts with positive associations, such as those in Argentina and northern Chile despite sufficient nutrition unlike in SE Taiwan. However, other data from SE Taiwan, China, West Bengal, and Bangladesh have been evaluated and it has been shown that poor nutrition and certain nutrients or factors do affect the potency of arsenic. There are also low-level drinking water studies in the U.S. and other countries (*e.g.*, Finland, Argentina) with populations with good nutrition (Mink *et al.*, 2007) and meta analyses of groups of studies showing no relationship between arsenical cancers (*i.e.*, bladder) and arsenic, that should be considered. Several agencies and organizations (*e.g.*, Health Canada Biostatistics Units, U.S. EPA, U.S. NRC) have published more recent arsenic assessments than the U.S. EPA IRIS (1998) used in the HHRA. The panel recommended that the other assessments and their differences should be presented.

Response: Agreed, this information has been added to the relevant section of the HHRA report.

Comment: A panelist pointed out that the selected TRV for copper is lower than the RDA for copper (IOM, 2003), which is based on an upper limit of the safe dose. The authors should look into this TRV further.

Response: The recommended dietary allowance (RDA) for copper provided by IOM (2000) is 900 µg/day for an adult. The selected adult TRV for copper used in the HHRA is 100 µg/kg/day. Assuming an adult body weight of 70.7 kg, this is converted to 7070 µg/day, which is clearly significantly higher than the RDA. IOM (2000) indicates that the adult Tolerable Upper Intake Level (UL) for copper is 10,000 µg/day which is protective of liver damage.

Comment: Panel members discussed whether the assessment might present a range of arsenic risk values based on different selections. Some thought that using a range of values is

leading to probabilistic assessment and the methods are not good enough. The panel thought that the authors should qualitatively discuss the range of risk values for arsenic.

Response: Agreed, the weight of evidence evaluation of arsenic has been enhanced.

7.2.3 Charge Question 15

Charge Question 15. Do you have any further concerns or comments regarding the hazard assessment?

Comment: The Ontario intake of concern for lead should be mentioned in the text. The value of 1.85 µg/kg-day is a population-based value.

Response: A discussion of this value has been added to the Toxicological Profile in Appendix A. Given that Flin Flon does not fall under the jurisdiction of the Ontario Ministry of the Environment, it was not considered to be necessary to include this discussion in the main report.

Comment: The definition of incremental lifetime cancer risk needs to be rewritten to make it clear that it refers to a population risk and not a prediction of risk for an individual. The upper bound lifetime cancer risk is an upper bound risk estimate. The true risk is likely to be lower and could even be zero. Health Canada's TD05, however, is not an upper bound, but is a linear extrapolation from the TD05 representing the best estimate. If one divides the TD05 by 5000, the result is a dose with a best estimate risk of 1 in 100,000.

Response: The report has been clarified to indicate that the ILCR represents an upper bound lifetime cancer risk to an exposed population.

Comment: The discussion on interactions between COCs could be put into a table. Most of the interaction data are on high doses where people would be expected to see effects, but what is really of interest for this assessment is interactions at low concentrations below the "safe" level, resulting in risk to those exposed. Panel members suggested using ATSDR toxicological profiles and the *Handbook of Metals Toxicology* (2007). Several other metals are elevated or present in site soils that might interact with the COCs, *e.g.*, zinc antagonizes the absorption and effects of several metals such as cadmium.

Response: Additional information has been added to this discussion as suggested.

Comment: A panel member suggested mentioning limited sunlight and Vitamin D deficiency as a potential special consideration.

Response: A qualitative discussion has been added to the revised report as suggested.

Comment: For arsenic, a correction for early life exposure is not needed as arsenic is not mutagenic and the data are from full generation exposures.

Response: The discussion in Chapter 6 has been revised to indicate that a correction factor for early life exposure was not applied in the assessment of carcinogenic risk from exposure to arsenic.

Comment: The community blood intervention level for Saskatchewan and Manitoba is not an absolute 10 µg/dL.

Response: This point has been noted. The HHRA will not include discussion to suggest this.

Comment: There is a statement in the document regarding lead indicating that the assessment is conservative in respect to adverse health effects, but elsewhere the authors acknowledge the potential for effects at lower levels of lead.

Response: The report has been revised to indicate that the assessment of lead exposure using the IEUBK model is considered to represent likely blood lead concentrations, consistent with the IEUBK supporting documentation, rather than an overly conservative estimate. Overall, the report has been revised to indicate that exposure and risk estimates are representative of central tendency estimates rather than reasonable maximum exposures. Specific to lead, language has been revised so as not to indicate that probability of adverse health effects are conservative.

8.2.1 Charge Question 16

Charge Question 16. Was the approach used to estimate Concentration Ratios (CRs) and Hazard Quotients (HQs) for acute inhalation and ingestion risk, respectively, consistent with accepted risk assessment methods, and were the values calculated correctly?

Comment: The panel again discussed the problems quantifying acute risks – the lack of defensible acute TRVs and large uncertainties in the results led them to recommend that the authors characterize acute risks qualitatively, but not quantitatively. Panelists voiced concerns that the acute results presented in the draft may be interpreted inappropriately causing people to be concerned when they should not. Panelists noted that if the authors do not have one-hour sampling data then they should not calculate one-hour hazard quotients. The characterization of chronic risk will protect for acute exposures.

A panelist noted that most of the pathways (*i.e.*, soil, drinking water, food) are chronic exposure situations, with the exception of the pica child behavior and so acute values are not important. But, the panelist pointed out that air is a relevant acute pathway, as there may be short term excursions. The assessment should focus the acute discussion on the air pathway. In summary, the panel agreed that estimating acute exposures is very problematic, as is identifying medical conditions or adverse effects from acute exposures. Acute exposure concerns apply only to the air pathways. Current air excursions are addressed through sulphur dioxide monitors and warnings. The assessment should characterize acute risks qualitatively. They cautioned the authors to be careful in characterizing acute hazards and risks so that the public does not misinterpret the true risk and take actions that are inappropriate to the magnitude of the risk.

Response: We disagree that the evaluation of acute exposures is not important and as such the quantitative evaluation of acute scenarios have not been removed from the HHRA. This section has been revised to incorporate the most relevant data available. Further discussion will enhance this section of the report. Reference to 1-hour air exposures and hazard quotients has been removed since 1-hour air data is not available.

8.2.2 Charge Question 17

Charge Question 17. Was the approach used to calculate the HQs and Incremental Lifetime Cancer Risks (ILCRs) for residential, outdoor workers, and recreational scenarios consistent with accepted risk assessment methods, and were these calculated correctly?

Comment: A panel member suggested the authors review their use of significant figures for the HQs, pointing out that in Table 5-4 for example; a HQ of 1.2 for arsenic should be one, as only one significant figure is appropriate.

Response: As discussed previously, the authors feel that it is appropriate to use two significant figures for HQ and ILCR estimates.

Comment: A panelist suggested the authors consider some way to assess risk from fish consumption for specific lakes. The issue is that some lakes have elevated concentrations and if these are favourite lakes for some people, exposure may be higher than the average for all the lakes. The panellist suggested a "favorite lake" scenario or some use of a weighted average based on data collected. Another panelist suggested evaluating the impact of a fish advisory on the overall risk to determine if a fish advisory would be an appropriate risk management consideration. An author clarified that the risk assessment back-calculated the concentration of mercury in local fish that would result in an HQ of 1 using the assumed consumption rates and additional sources of exposure. This concentration (0.19 ppm) was lower than the provincial (Manitoba and Saskatchewan) and federal (Health Canada) guideline of 0.5 ppm, likely as a result of the higher local fish consumption rate assumed in the risk assessment. The risk assessment provided a comparison of the 0.5 ppm guideline to the 95% UCLM mercury concentrations for each fish species and for fish in each individual lake. A panel member pointed out that there is no pattern of elevated methyl mercury levels in fish with distance from the source.

Response: The contribution of local fish to the total exposure and risk was minor for all COC other than mercury. As discussed, individual lakes and individual fish species were considered in the assessment of risks from mercury associated with the consumption of local fish. Therefore, a favourite lake or favourite fish scenario was considered.

Comment: A panel member questioned why the authors did not use a composite receptor for arsenic given its carcinogenicity. Others agreed the composite receptor would be appropriate and the panel recommended its use for arsenic.

Response: A composite receptor was used to assess carcinogenic risks for arsenic within the HHRA. However, the derivation of the arsenic soil PTC was completed using the adult soil ingestion rate (0.02 g/day) and body weight (70.7 kg) following the approach used by the CCME in the derivation of the soil quality guideline. This was done because the weighted soil ingestion rate and body weight for the composite receptor are very similar to those for the adult. The HHRA has been revised to use a soil ingestion rate (0.02 g/day) and body weight (63 kg), reflective of a composite receptor, in the derivation of the soil PTC.

Comment: The panel discussed the outdoor worker scenario and questioned use of the maximum value for exposure point concentrations for the outdoor worker, noting that the

resulting cancer risk for arsenic does not look right as it indicates the worker has a risk when the public who lives in the community does not. A panelist suggesting that a mean exposure value would be more appropriate because the worker would be expected to move around the community and not stay in one location. A panelist questioned if the outdoor scenario is necessary in that if indoor and outdoor air concentrations are assumed equivalent, and the general public is not at risk, then anyone working outdoors and not living in the community would be protected as well.

Response: One of the primary differences between the residential and outdoor worker scenarios was that the outdoor worker scenario was assessed using soil concentrations collected from non-residential properties as part of the Manitoba Conservation soils study. This scenario allowed for the consideration of this data which was not used in the residential scenario because it was from locations that were not representative of residential soil. As suggested by the panel, the report has been revised to use the 95% UCLM concentrations in place of the maximum concentrations to predict risks under the outdoor worker scenario.

Comment: The HHRA calculated non cancer hazard quotients for arsenic and cadmium for five age groups, and the results showed that toddlers have the highest risk. Panel members questioned the appropriateness of this – the toddler drinks more water per kilogram body weight than the other groups and this is the basis for the larger HQ. The authors should look closely at the data and endpoints that the TRVs are based upon to determine if smaller age categories are appropriate. For example, the arsenic TRV is based on populations exposed over an entire lifetime, including children and *in utero* exposures; much longer than the few toddler years would be needed to elicit effects from arsenic. There is no indication that children would be more sensitive to arsenic than adults on a body weight basis, therefore use of age groups for arsenic is not appropriate. However, for methyl mercury and lead, the TRVs are based on effects from shorter exposures and breakdown by age category may be more appropriate, although the methyl mercury TRV is based on an adult female intake per body weight and associated fetal exposure. A panelist explained that risk assessors try to keep the exposure metric equivalent to the toxicity metric. To look at chronic exposure greater than 7 years, one compares these exposures to chronic risk values. For exposures less than 7 years, one would use a subchronic value. The panelist noted that ATSDR has developed a shorter term arsenic value and the panelist will provide this to the authors. An author agreed that the arsenic results alone do not provide a realistic assessment and that was why they presented multiple lines of evidence for consideration. The authors think that a urinary arsenic study can provide additional information to better characterize risk.

Response: While Intrinsik agrees with the scientific basis of the argument not to apply TRVs based on lifetime exposure to smaller age categories such as the toddler, this approach was used in the current assessment because it is a federal requirement for risk assessments in Canada. It is addressed within risk assessment guidance documents prepared by Health Canada and the CCME uses the characteristics of the toddler to derive Federal soil guidelines for non-carcinogenic compounds such as cadmium, mercury and selenium. To be consistent with the Federally endorsed recommendations, the HHRA has not been revised as suggested. The overall outcome of using this approach is the derivation of conservative risk estimates and PRGs.

Comment: Panel members recognized the sediment data are limited and will not materially affect the quantitative results, but recommended that sediment exposure (*e.g*., oral exposure while swimming and playing near shore) be addressed qualitatively because it is a common pathway considered.

Response: The assessment of exposure and risk associated with COC in sediment while swimming has been added to the recreational scenario within the revised report.

Comment: Panel members recommended that the authors consider providing an indication of the magnitude of concern by communicating the number of properties that exceed a target level for at least one of the COCs. They should avoid double counting properties.

Response: The majority of properties with concentrations of COC in excess of the PTCs were located in West Flin Flon. Those properties with elevated concentrations generally had concentrations above all three PTCs (*i.e*. for arsenic, lead, and mercury). Including the requested information does not significantly add to the understanding of the magnitude of concern.

8.2.3 Charge Question 18

Charge Question 18. To assess lead exposure, the authors used the HHRA exposure model as well as the U.S. EPA IEUBK model. Comment on the analysis and scientific defensibility of the results.

Comment: The panel members discussed the problems with presenting two different approaches (deterministic model and IEUBK model) for characterizing the risk from lead exposure. Members noted that both are good tools, but the results cannot be compared quantitatively (apples and oranges comparison).

Response: The use of both the deterministic model and the IEUBK model in the HHRA was done to provide two distinct methodologies for assessing risks to a complex chemical that could be considered within an overall weight of evidence approach. The purpose of including both models was not to directly compare the results of each, but to acknowledge that assessing lead exposure in children is an uncertain process and that multiple lines of evidence should be considered. The deterministic approach follows the framework endorsed by Canadian Federal agencies, while the IEUBK model is used internationally for assessing childhood exposure to lead. Each approach provides important information that should be considered by risk assessors and risk managers.

Comment: Panel members raised issues with use of the IEUBK model; in particular one noted that the model is meant to be applied to a homogenous exposure scenario. The authors took the UCLM of a variable exposure scenario and used it as an input; the distribution that comes out of the IEUBK is meant to reflect individual behaviors and physiology, not variability in the population's exposures. The exposure concentrations from the Manitoba soils study and the Jacques Whitford (2008) study are not homogenous; the coefficient of variance is about 0.8.

Response: It is recognized that the IEUBK model was designed to represent variability in individual behaviours and physiology (in addition to variability in repeat sampling, variability in sample locations, and analytical variability). However, according to the U.S. EPA (1994)

Guidance Manual for the IEUBK Model for Lead in Children, "the output of the model may also be considered to be the predicted geometric mean blood lead of a population of children with the same lead exposure scenario, and the upper tail of the probability distribution to be the fraction of children exceeding the chosen blood lead level of concern when all of these children have the same exposure history". It is not practical to independently model blood lead concentrations in children based on measured concentrations of lead in soil and dust on individual properties. The assessment is intended to provide an estimate of lead exposure in a sub-population of the study area.

Additional discussion has been added to Section 4.1.9.2 to further clarify that the use of the IEUBK model as a means of predicting the fraction of children in a population that may exceed a blood lead level of concern assumes that all individuals are subject to similar exposure point concentrations. Individuals within this population that may be exposed to significantly higher concentrations, particularly for prolonged periods (*i.e.*, a home with elevated levels in a backyard play area) may be subject to higher blood lead concentrations. Use of exposure point concentrations based on the 95% UCLM of data sets is intended to provide a general representation of the geometric mean blood lead concentration in children within a population and the fraction of children which may have blood lead concentrations in excess of a level of concern.

Comment: The panel discussed comparing results of the deterministic model with the IEUBK outputs. Several panel members suggested ways that might make the two results more comparable. One panel member suggested that using the same parameter values would make the comparison of results a little better, but there is still the issue of bioavailability to contend with. Another panel member noted that even with the same assumptions, the two models use different toxicity benchmarks. A third agreed and noted that the two models are fundamentally different in that that the deterministic approach uses conservative estimates and the IEUBK model uses central tendency estimates and then using an assumed geometric standard deviation, calculates the 95th percentile blood lead level, which is then compared to 10 µg/dL. The authors stated that they did not intend to compare the two approaches and added that the deterministic approach used Canadian parameter values, while the IEUBK model uses U.S. values.

Response: As discussed previously, the intention of including both the deterministic model and the IEUBK model was not to derive a set of results for direct comparison but rather to provide two distinct lines of evidence for assessing childhood exposure to lead. As recommended by the U.S. EPA, default values were only changed when there was strong site-specific data available to support deviating from the default values. This included measured concentrations of lead in various environmental media, which were consistently used in both models. Receptor characteristics such as soil ingestion rates and consumption rates were not site-specific, and therefore were not changed within the IEUBK model. The deterministic model used values endorsed by Canadian agencies, while the IEUBK model used values endorsed by the U.S. EPA. Both sets of characteristics are scientifically valid and warrant consideration in the HHRA.

Comment: One panel member thought that using the IEUBK model results in inappropriate comparisons between the two methods. This reviewer noted that the U.S. CDC does not recommend that their lowest blood lead intervention level (10 µg/dL) be used as a toxicity reference value for setting allowable concentrations of lead in the environment (Brown and Rhoads, 2008; Brown and Meehan, 2004; CDC, 1991). The blood lead intervention levels are

derived to guide secondary prevention (after exposure has already occurred), not establish acceptable levels for primary prevention (defining acceptable levels of exposure). This reviewer did not think the model adds to the HHRA or should be used, stating that the IEUBK model is most appropriately used for interventions, not for prevention activities, which should be based on the TRVs. Another agreed saying the deterministic model reflects the concept of a community blood level and the two should be kept in separate contexts. A third panelist thought that there is uncertainty with all models, but the results of the two models are within the same range. Other panel members agreed that the two results generally support each other, but were concerned about making an explicit or quantitative comparison. A panelist pointed out that if the authors do not use the IEUBK model they will be subject to criticism from those who know it is an available tool which is used widely. The authors agreed that they would be considered remiss if they did not use the IEUBK model but they understand and agree with the panel's concerns and comments. They clarified that they used the IEUBK defaults for the most part, but used some site specific parameters as they were justified.

Response: A reviewer commented that the U.S. CDC does not recommend that their intervention level of 10 µg/dL be used as a toxicity reference value for setting allowable concentrations of lead in the environment, and that the blood lead intervention levels are derived to guide secondary prevention after exposure has already occurred. In the Flin Flon/Creighton area, exposure to lead has already occurred, and the focus of the current HHRA is to predict resulting blood lead concentrations and derive preliminary remediation goals to prevent the occurrence of blood lead levels above the intervention level. The IEUBK model and blood lead level of concern are not being used to derive soil guidelines for the general population; they are being used to help provide risk managers with guidance for addressing concerns in an impacted community.

Comment: The panel was comfortable with the lead hazard indices from the deterministic modeling and noted that the authors used the same Canadian and site specific assumptions as the other COCs. The panel recommended that the authors mention the IEUBK model and results very briefly in the text, but move all the details to an appendix where they can explain how the results of the IEUBK model support those of the HHRA. The primary reasons for moving the IEUBK to the appendix is the homogeneity issue and that the two models are not directly comparable. Moving the details to an appendix will also improve the readability of the chapter. The authors should attempt to harmonize the input parameters so that the results are even more comparable. The final paragraph in section 5.2.4 recommending blood lead monitoring should be retained (except for comparison to 10 µg/dL, see below). A panel member noted that it is useful to compare different models, but they need to use the same exposure inputs, and thought that if the authors used the same inputs as the spreadsheet model, the IEUBK results will be lower. An author noted that one difficulty is that the spreadsheet model does not have parameter values for all the different age groups evaluated within the IEUBK model.

Response: As discussed previously, the intention of including both the deterministic model and the IEUBK model was not to derive a set of results for direct comparison but rather to provide two distinct lines of evidence for assessing childhood exposure to lead. As recommended by the U.S. EPA, default values were only changed when there was strong site-specific data available to support deviating from the default values.

The results of the biomonitoring study (completed following the IERP review) have indicated that the predicted geometric means and fractions of children above the blood lead level of concern using the IEUBK model are very consistent with measured results. Although the results of the biomonitoring study will not be incorporated into the HHRA document, but will be included in a final Bridge Document, knowledge of the data to support the IEUBK model predictions support the inclusion of this assessment in the HHRA.

Comment: A panel member questioned the accuracy of calling 10 µg/dL the Canadian community blood lead intervention level, noting that the appropriate guidance is CEOH (1994). That guidance does not identify 10 µg/dL as a standalone value, but recommends community intervention be considered when the mean blood lead levels of a sample from the community exceeds a reference mean plus three standard deviations from the reference mean, or when the percentage of children in a community with values above 10 µg/dL is double that seen in the general population.

Response: Agreed, further explanation of this issue has been provided in Chapters 4 and 5 of the revised HHRA.

Comment: Another panel member asked the authors how they calculated the TDI and equated it to the 10 µg/dL blood lead level. An author indicated that the TDI was based on studies of formula fed infants, breast milk, and infant food and that 58% absorption factor is used. The panel member thought this absorption factor seemed high given that the absolute gastrointestinal absorption of soluble lead in water is 50%.

Response: Further information regarding this issue has been provided in Chapter 4.

Comment: A panel member suggested that when calculating the contribution of lead exposure from each medium that soil and dust contributions be separated. This reviewer also cautioned presenting absolute values and predictions from the model and that it is more appropriate to use the results in a relative comparison of two scenarios. Several reviewers suggested rewording some of the conclusions in section 5.2.5, in particular the inappropriately predictive statements regarding the percentage of properties containing levels of lead that may have adverse effects on young children.

Response: The IEUBK model does not distinguish between contributions from outdoor soil and indoor dust. The influence of each of these media cannot be separated.

The discussion in Section 5.2.5 has been revised as suggested. The discussion now refers to the percentage of properties that contain concentrations of lead in excess of the PTC rather than the percentage of properties with concentrations that may have adverse effects on young children.

Comment: A reviewer noted the variability in bioaccessibility of the samples, which leads this reviewer to think there are different sources of lead. The reviewer suggested looking for consistency in bioaccessibility by source or area and noted that mineralogical analysis will help understand which bioaccessibility estimates to apply to which soil concentrations. Plotting these as contour lines on a map would be helpful. This reviewer also thought that the risk at most sites is not from soil, but from the house (*e.g.*, paint). The information on the relationship between concentrations of lead in soil and blood lead levels from different North American populations

and sites (Table 5-27) does not convey a straight story. In the well-controlled studies, the relationship of soil to blood lead is not very strong. The reviewer thought that the IEUBK model will indicate the problem is soil, when it is likely the lead is from house paint.
Response: Following the completion of the biomonitoring study, recommendations for the collection of additional data will be evaluated. The mineralogical analysis of indoor dust and/or outdoor soil samples is one option that is being considered to provide information on the source of lead.

Comment: In summary, the panel agreed that the HHRA should use the hazard indices derived from the deterministic model for lead risk. However, they also suggested the authors use the IEUBK model to support the spreadsheet model results, harmonizing the parameters between the two as much as possible. The authors should move the detailed discussion of the IEUBK to an appendix.

Response: As described previously, the intention of including both the deterministic model and the IEUBK model was not to derive a set of results for direct comparison but rather to provide two distinct lines of evidence for assessing childhood exposure to lead. As recommended by the U.S. EPA, default values were only changed when there was strong site-specific data available to support deviating from the default values.

The results of the biomonitoring study (completed following the IERP review) have indicated that the predicted geometric means and fractions of children above the blood lead level of concern using the IEUBK model are very consistent with measured results. Although the results of the biomonitoring study will not be incorporated into the HHRA document, but will be included in a final Bridge Document, knowledge of the data to support the IEUBK model predictions support the inclusion of this assessment in the HHRA.

8.2.4 Charge Question 19

Charge Question 19. Soil Preliminary Remediation Goals (PRGs) and Provisional Trigger Concentrations (PTCs) were derived in Chapter 5 for the COCs. Was the approach consistent with accepted risk assessment methods and were the values calculated correctly?

Comment: A panel member again reiterated that inclusion of market basket foods and dental amalgams has to be clearly communicated to the risk managers, noting that in the U.S. PRGs are derived for contaminants when risk decision criteria are exceeded (*i.e.*, an unacceptable risk occurs). In this assessment, PRGs and PTCs were derived regardless of whether the risk assessment results were above or below decision criteria. This approach seems to be counterproductive and begs the question of why the risk assessment was even done. A particularly egregious example is that of selenium. All calculated non-cancer risks were below a hazard index of 1.0. The market basket dietary intake accounted for 73% of that risk, whereas soil accounted for only 2%. Yet a PRG was developed for selenium. Cadmium and copper were also below non-cancer decision criteria with market basket dietary intakes accounting for the majority of the estimated risk. Yet PRGs were developed for these inorganics. In the case of copper this could result in unnecessary and expensive soil cleanups, while completely ignoring what appears to be a significant contribution of copper from the water distribution systems in homes.

Response: As discussed within the report, risk assessments typically employ the 95% UCLM to characterize the exposure point concentration (EPC) of a given exposure unit. In this report, the exposure units were defined as the communities under assessment in the HHRA. The underlying assumption used when developing the chronic residential exposure scenarios was that individuals would move randomly within each community and, therefore, over time, come into contact with the average soil concentration within a given community (or exposure unit). In reality, individuals do not move in a random fashion within their community, but rather exhibit predictable spatial patterns in their movements. For example, many individuals will tend to spend the majority of their time between home and work or school. Therefore, the evaluation of risks on the basis of average EPCs (assuming random movement) in an area-wide risk assessment may underestimate risks for some receptors. As a result, in addition to predicting risks using the community-based EPCs, soil preliminary remediation goals (PRGs) were derived for each COC to be protective of residential receptors. These PRGs can then be used to determine on a property-by-property basis, which properties contain concentrations that have the potential to cause unacceptable risks.

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

Given that there was a large degree of variability in concentrations of COC in residential soil, it was not considered to be sufficiently protective of individuals to base conclusions of the HHRA on the forward risk calculations using the community-based EPC soil concentrations. Consideration was given to individual properties through comparison of property-specific concentrations to the PRGs to assess the potential for risk on a property-by-property basis.

Although PRGs were derived for cadmium, copper, and selenium, the HHRA concluded that no remediation or risk management was required to address risks to these COC.

Comments: Another reviewer noted that the market basket foods are not an adjustable factor and therefore PRGs should not be derived for this pathway. That leaves just air and soil because if one predicts a risk from a pathway, then one needs to look at where something can be done to reduce exposure. Unfortunately, only 10% of properties were sampled for soils and nothing is known about the other 90%. Another reviewer suggested the author set the air levels to background to calculate a PRG because the smelter will be shut down.

Response: Within the HHRA, PRGs were not derived for the market basket foods pathway. Consistent with federal guidance on HHRA in Canada, the HHRA considered exposure to COC through all relevant exposure pathways, including those that are unrelated to the HBMS complex. Assuming that exposure to all media other than soil and dust remains constant, the PRGs/PTCs were derived by determining the concentrations of COC in these media that are associated with the residual TDI (for non-carcinogenic COC). For carcinogenic COC (*i.e*., arsenic), the PTC represents the soil concentration associated with an acceptable risk level (*i.e*., an ILCR of 1.0×10^{-5}), independent of any additional exposures. Given that the focus of the HHRA was evaluating the potential exposure and risks associated with COC from the HBMS complex, reducing exposure to COC in soil/dust was considered to be the primary method for reducing total exposure. Within the final Bridge Document, if it is concluded that risk

management measures are required, reducing exposure from additional pathways such as consumption of drinking water will be evaluated.

The sensitivity analysis in Chapter 7 evaluated the effect of reducing air concentrations by a factor of 50% to account for reduced future emissions from the HBMS complex. Although it is anticipated that the smelter will be shut down, given that the smelter is currently operational and that there is always the potential that it will remain in operation past the scheduled shut down date, it was not considered to be appropriate to derive PRGs/PTCs assuming background air concentrations. In addition, contributions from re-suspended particulates from impacted soils would not be considered if air concentrations were set to background levels.

8.2.5 Charge Question 20

Charge Question 20. Chapter 6 identifies and evaluates other risk issues relevant to the HHRA. Are the analyses and conclusions for these issues scientifically sound? Have the issues been appropriately considered in the overall HHRA and recommendations? Have potentially sensitive populations been adequately addressed?

Comment: A panel member suggested that the discussion of mixtures found on page 6-14 would benefit from inclusion of some newer publications from the U.S. EPA and referred the authors to www.epa/ncea for these publications (publications listed in Appendix E).

Response: There is limited information on the interaction of COCs with non-COCs. However, as suggested, the ATSDR interaction toxicological profiles have been consulted and the relevant information has been extracted and included in this section (see Table 6-3). It is agreed that interactions at low concentrations would be very interesting in this risk assessment. However, that data is scarce. Therefore, to be protective of the entire population the available interaction data (although for high doses) were used in the risk assessment and represent a "worst-case" scenario which is a conservative approach.

Comment: In Section 6.8 and discussions of lead body burden, panelists questioned statements that lead is partitioned to bone. The panel members pointed out that lead in bones is a dynamic process, with ossification and de-ossification over time. Panelists also noted that cadmium accumulates in the kidney, another organ that accumulates metals over a lifetime.

Response: While some ossification and de-ossification will occur over time, ATSDR (2007) indicates that approximately 94% of the total body burden of lead is found in the bones. Furthermore, ATSDR (2007) indicates that lead concentrations in bone increase with age throughout the lifetime, indicative of a relatively slow turnover of lead in adult bone. ATSDR (2008) indicates that cadmium is accumulated in the liver and kidneys of exposed individuals. Section 6.8 will be modified to clarify these two points.

Comment: A panelist sought clarification from the authors whether they applied age-dependent uncertainty factors to increase the cancer dose response assessment slopes. An author indicated they did not and will clarify this in the text.

Response: The discussion in Section 6.1.3 has been revised to indicate that a correction factor for early life exposure was not applied in the assessment of carcinogenic risk from exposure to

arsenic. This was not deemed necessary since the arsenic cancer slope factors are based on lifetime studies that inherently address increased susceptibility following exposures to infants, children and unborn fetuses.

Comment: Referring to the body burden discussion in Section 6.8.1, a panel member noted that there are *in vitro* studies that suggest that methylated daughter products of arsenic may be more toxic than the inorganic form. Another panel member noted that ingested methylated arsenical compounds have been determined by the U.S. EPA's Science Advisory Board to be threshold carcinogens, and that more extensive methylation capacity results in more efficient excretion and less *in vivo* exposure to inorganic arsenic and certain more toxic metabolic intermediates (*e.g.*, trivalent monomethyl forms).

Response: The discussion in Section 6.8.1 has been modified to reflect the information provided by the panel member.

Comment: Under the discussion of arsenic uptake, distribution, storage, and elimination, a panel member questioned how one could have inhalation exposure to arsenobetaine as it is a non-toxic, water-based compound that is excreted unchanged from the body. An author indicated they would check on this.

Response: This text has been clarified in the HHRA as the reference is referring to the excretion of arsenobetaine following inhalation of inorganic arsenic (AsH_3) (Apostoli *et al.*, 1997).

Comment: A panelist clarified that the discussion at the bottom of page 6-5 regarding the NAS (1993) recommendations on a child protective uncertainty factor. Discussions related to children's exposure and risk, for example, as found on page 6-3, should include some additional publications for a more balanced presentation. On the bottom of page 6-5, the NRC statement that EPA uses an uncertainty factor when developmental toxicity data are available is not correct; rather EPA uses a database uncertainty factor when such data are missing. Several publications were suggested and are listed in Appendix E.

Response: It appears as though the panelist may have misread this section, since it indicates the following:

> *'the NAS therefore recommended that a child-protective uncertainty factor of up to 10-fold be considered where there is either evidence of developmental toxicity, or data from toxicity testing relative to children are incomplete'*

This statement is consistent with the statement that EPA uses a database uncertainty factor when such data are missing.

8.2.6 Charge Question 21

Charge Question 21. Are there additional issues or concerns that the authors should have addressed regarding the hazard assessment, the selection of exposure limits and the appropriate use of the values in the risk assessment? Do you have additional comments regarding aspects of the risk characterization and results?

Comment: Panel members discussed the community health assessment with Dr. James Irvine, an observer from Saskatchewan Ministry of Health during a break and asked him if there were key disease endpoints missing from the community assessment. Dr. Irvine told them that the assessment could not address skin cancer due to differences in nomenclature between the two provinces. Dr. Irvine explained to the panel that community assessment combined data from the two provinces. It was meant to be an overall approach to a community health assessment to compare demographics, non-medical health determinants, cancer incidence, and the common causes of death for Flin Flon and Creighton with their corresponding health regions and provinces but it was not specifically related to the COCs. Unfortunately, they could not get accurate data for skin cancer.

The panel recommended that the authors more fully discuss the community assessment and the strengths and weaknesses of the study and conclusions, particularly that the most likely cancer from arsenic exposure would be skin cancer and the community assessment did not evaluate skin cancer.

Response: Within the discussion of arsenic cancer risk estimates in Chapter 5 and in the summary of the community health assessment in Section 6.8, it has been noted that the community health assessment could not include incidence of skin cancer due to differences in nomenclature between the Provinces. It has also been noted that given that one of the most common forms of cancer associated with arsenic exposure is skin cancer, the results of the Community Health Status Assessment may not have captured increased incidence in arsenic-related cancers in the Flin Flon area population relative to the provincial averages.

8.2.7 Charge Question 22

Charge Question 22. Chapter 7 presents uncertainty and sensitivity analyses. Were all the significant sources of uncertainty identified and characterized? Were quantitative uncertainty and sensitivity analyses done correctly? Are the conclusions regarding the significance and impact of the uncertainties on the resulting assessment correct?

Comment: A panel member noted that normally with a sensitivity analysis, one changes each parameter by a certain amount (*e.g.*, 5%). But since linear regression was used, all the parameters will be the same. In this assessment, the authors evaluated different choices and so it is hard to understand the true sensitivity in the model when you vary the parameter values.

Response: When selecting exposure parameters or characteristics within the HHRA, there are often situations when there is notable uncertainty about which value to use. Within a deterministic assessment, a single value needs to be selected. The sensitivity analysis provides an opportunity to determine the impact of using alternate values for key input variables on the calculated health risks. Rather than adjusting parameters by an arbitrary amount, the sensitivity analysis illustrated the effect of using these logical alternate values.

Comment: Another panelist suggested adding a qualitative discussion about potential exposure from breast milk for each of the COC and whether breast milk would be a pathway of concern for any of the COC. The panelist suggested a number of papers on concentration of constituents in breast milk and blood, and thought that breast milk exposure will be important for methyl mercury, less so for lead. Another panel member noted that the arsenic TRV is based on a population with exposure to drinking water throughout all life stages and therefore breast

milk exposure would have been included. Exposures to arsenic in soil and dust for the Flin Flon population for pregnant and nursing women would be considerably lower than for children compared to in a population exposed by drinking water. Thus, the Flin Flon population would not receive much exposure *in utero* or through breast milk as the population that forms the basis for the arsenic TRV.

Response: A qualitative/semi-quantitative discussion regarding exposure to COC *via* breast milk has been added to Chapter 7.

Comment: Information on fish consumption for the child receptor is mentioned in Chapter 7, but was not described in earlier chapters and should.

Response: A description of the fish consumption rates for the toddler, child, teen, and adult is provided in Section 4.1.7.6.

9.2.1 Charge Questions 23 and 24

Charge Question 23. Are the conclusions for each COC valid and are they supported by the data and the risk assessment? What is the likelihood that actual health risks have been over or under estimated? Are the potential human health hazards of the COCs adequately addressed?

Charge Question 24. The authors discuss biomonitoring and make recommendations regarding arsenic, lead and mercury. Are these recommendations appropriate and adequately supported?

• Chronic Endpoints – Residential Scenario (8.1.2)

Comment: A panel member disagreed with the approach for the systemic toxicity endpoints and recommended that the authors keep the intake metric consistent with the toxicity metric (for inhalation - $\mu g/m^3$). The inhalation and oral hazard quotients can then be added for a hazard index. This reviewer thought that the authors' approach was not correct in that they did not take into account absorption and distribution, and when there are systemic toxicity values for both routes, they should not convert the inhalation intake to a dose. Another panelist agreed with the authors' approach for assessing endpoints that are portal of entry effects in addition to systemic endpoints where doses from multiple routes were added by the authors and compared against a TRV for a systemic endpoint for one route. The panelist explained that one is concerned with the dose to the target tissue and the separate HQs (for portal of entry effects as well as systemic effects from multiple routes of exposure) is defensible. The approach taken by the authors is defensible and allows for assessing portal of entry affects against a portal of entry TRV as well as adding exposure from multiple portals of entry for evaluating risk based on a systemic endpoint. However, this reviewer thought that the approach would be limited in terms of adding HQs for different endpoints based on toxicokinetic considerations. Another reviewer agreed and thought that the oral and inhalation results should be added together for systemic toxicity endpoints because what gets absorbed and distributed systemically is then available. A fourth panel member agreed that systemic dose is what is important and that one needs to account for relative bioavailability by pathway.

Response: Although the panel did not reach a consensus on which approach should be used to assess systemic toxicity endpoints, the majority of the panel agreed with the approach used within the HHRA in which inhalation, dermal, and oral daily intakes (mg/kg-day) were added and then compared to the oral TRV. In addition, the inhalation intake alone was compared to the inhalation TRV.

Comment: Panel members also sought clarification on how the authors adjusted dermal concentrations to calculate intake noting that it should be based on gastrointestinal bioavailability from the oral toxicity study. An author indicated that they used relative dermal absorption factors provided by Health Canada (2006). The panel members suggested explaining this in the text and providing the reference for the Health Canada values.

Response: Details are provided in Section 4.1.7.1. Additional discussion has been added for clarification.

Comment: One panelist noted that the 3% dermal absorption for arsenic that the authors used is based on a study by Wester *et al*. (1993) that had some design issues and used soluble arsenic freshly added to soil rather than arsenic in soil from a site. Lowney *et al*. (2007) repeated the study with a breathable patch and aged soils. They found that they could not detect any dermal absorption of arsenic.

Response: It is recognized that that the study by Lownney *et al*. (2007) indicated that dermal absorption of arsenic from field soil is likely negligible, however, the most recent recommendations from Health Canada (2008) and RAIS (2008) encourage the use of a 3% RAF_{Dermal} for arsenic. The contribution of the dermal route to the total arsenic exposure is very minor, therefore it was considered to be conservative to retain the use of the 3% value recognizing that it will not have a significant impact on the overall risk estimate.

Comment: The panel recommended that in addition to what the authors have presented, they should calculate separate oral and inhalation HQs when toxicity values are available. These HQs should then be added together and presented with the authors' original approach. The alternative HI should be discussed with the results in Chapter 8 (*e.g*., Table 8-4).

Response: The authors do not feel that including this additional Hazard Index improves the interpretation of risk estimates and may unnecessarily complicate the risk characterization. For example, the assessment of non-carcinogenic risks from arsenic currently includes an HQ based on a comparison of total systemic dose occurring from oral, dermal, and inhalation pathways to the oral TRV. In addition, the EPC air concentration is compared to the RfC to produce an inhalation CR. If an HI were to be included, this would involve deriving a separate HQ that only considers oral and dermal exposure and then adding this HQ to the CR. Given that the oral TRV and the inhalation RfC are based on separate endpoints, the toxicological basis for combining these values is questionable. In addition, the inclusion of an HI would not impact the derivation of the PRGs/PTC.

Comment: The panel also cautioned that one must be careful interpreting the hazard quotients for toddlers. The toddler's few years of exposure is being compared to chronic toxicity values and for most of the COCs these hazard quotients are not appropriate because the toxicity values are based on lifetime exposure. For example, the arsenic and cadmium TRVs are calculated from studies with whole life human exposures.

Response: While Intrinsik agrees with the scientific basis of the argument not to apply TRVs based on lifetime exposure to smaller age categories such as the toddler, this approach was used in the current assessment because it is a federal requirement for risk assessments in Canada. It is addressed within risk assessment guidance documents prepared by Health Canada and the CCME uses the characteristics of the toddler to derive Federal soil guidelines for non-carcinogenic compounds such as cadmium, mercury and selenium.

Chronic Endpoints – Outdoor Commercial/Industrial Workers (8.1.3)

Comment: The panel recommended the authors use the 95% UCLM rather than the maximum value.

Response: The commercial scenario has been revised as suggested.

Chronic Endpoints – Recreational Scenario (8.1.4)

Comment: The panel recommended the authors qualitatively address the potential for exposure through sediments.

Response: A quantitative assessment of exposure and risk through ingestion of sediments has been added to the recreational Scenario.

Summary of Discussion of Chronic Residential Results (8.2)

<u>Arsenic</u>

Comment: The panel discussed the recommendation for arsenic biomonitoring of a sample population of children up to 16 years of age. Some thought the recommendation appropriate, based on results of the HHRA. Others expressed concerns with the recommendation. One panelist did not think biomonitoring would be justified because the results of the HHRA were not elevated much above background and the panelist did not think that biomonitoring would show anything above background levels. Another expressed concern that the bulk of intake is from market basket foods and the panelist was not convinced that one would be able to distinguish site sources from food. A third thought that if the biomonitoring finds that levels are not above background, this would be a useful finding and one could conclude that exposure levels in the COIs are similar to other populations in Canada. This panel member thought that communicating risk on arsenic has always been difficult and if they do not do a urinary arsenic study, what other measures can they take? Others cautioned that the TAC must think about risk communication and identify beforehand what health screening benchmarks to use and what message to communicate about higher values and whether they will give individual their results.

Response: The TAC decided that the recommendation for arsenic biomonitoring was justified and would be a beneficial component of the overall study.

Comment: Panel members discussed what to use for a reference population. One thought that having a reference population was not always necessary. If the effect of soil concentrations is the question, then the authors could look at biomarker levels of children within the community exposed to different soil concentrations to see if a relationship exists. Another panel member

suggested the authors use a nearby referent population; otherwise community members will see the study as deficient. Another suggestion was to use the population being testing for internal reference and the question is whether the individual's soil and biomonitoring results are correlated.

Response: The biomonitoring protocol was developed by a team of physicians, epidemiologists, statisticians, toxicologists, and field investigators. This protocol underwent peer reviews focusing on both ethical and scientific aspects of the study.

Comment: The chair asked if the panel could definitively recommend biomonitoring if all the concerns they raised were addressed. Half the panel recommended that biomonitoring for arsenic be done. One of these suggested using a local population as reference and split properties below and above a trigger concentration to see if there is a difference between them. Others thought biomonitoring could provide the community with assurances regarding risk. One of these panelists did not think that the HHRA findings support biomonitoring, but thought it could be useful if people want it; however, the program would need to include surveys on activities and diets to help determine the sources of exposure and the relative contribution of non-site exposure.

Response: As described previously, the TAC decided that the recommendation for arsenic biomonitoring was justified and would be a beneficial component of the overall study. A very detailed questionnaire was completed for all households with individuals participating in the biomonitoring study.

Lead

Comment: The authors recommended a blood lead survey up to age 7. All but one panel member supported this recommendation, although most of the supporters had some reservations and offered caveats. One panelist noted that if the authors step away from the IEUBK model in the text, their conclusions may be somewhat different, with hazard screening resulting in fewer problem properties. Another panelist thought that blood lead survey is a good idea for any community with an aged housing stock, near a source of lead emissions, and with some properties with high soil concentrations. Blood lead can help identify the need for intervention sooner rather than later. Another thought that the recommendation is supportable, but that the community should be involved in the design, and the limitations and study design should be carefully explained to the community members. Another also cautioned that those conducting the blood lead testing will have to be very savvy with risk communication and what they tell people about risk.

Response: The TAC decided that the recommendation for lead biomonitoring was justified and would be a beneficial component of the overall study.

Cadmium

Comment: The panel noted that with the previous discussions, the authors need to recalculate and revise the text for cadmium. They noted that the statement regarding the ILCR being elevated should be checked closely, as the risk is no greater than that for arsenic. The authors need to be careful to be consistent with these types of qualitative judgments and statements

between COCs. They also noted that the reference to future smelter emissions should be revised.

Response: Qualitative judgements and statements (*e.g.*, ILCRs were quite elevated) have been removed from the revised report as suggested.

Methyl Mercury

Comment: An author clarified that they have revised this section and no longer recommend biomonitoring for methyl mercury. The authors did recommend that a fish consumption advisory be considered, particularly for sensitive populations, to reduce exposures to methyl mercury. One panelist thought that the levels were just marginally different from market foods and was not sure a fish advisory would do any good. The panel agreed with the recommendation to consider a fish advisory for methyl mercury and suggested ongoing monitoring of methyl mercury in fish.

Response: The recommendation to include methyl mercury in the biomonitoring study has been removed from the revised report.

Inorganic Mercury

Comment: The panel discussed the authors' recommendation for biomonitoring for inorganic mercury. They discussed that the TRV for inorganic mercury is based on a NTP chronic rat study with renal effects (tubular necrosis). A 14-day study in rats has a no effect level at the same critical dose and so it appears that duration of exposure is a factor involved for inorganic mercury's toxicity. A panel member pointed out that the only receptor with a hazard quotient over 1 was the toddler and asked who they would suggest for biomonitoring. Another panelist asked what would be used for the trigger level and pointed out that the hazard quotient for inorganic mercury averaged only 2. Panelists objected to biomonitoring for inorganic mercury because the TRV is based on a chronic study, whereas the HQ for toddlers is 2, and there is a large uncertainty factor associated with the TRV; therefore, exposures slightly greater than the TRV may not be associated with any appreciable risk of adverse effects. Another reviewer noted that the HHRA authors used 100% bioavailability in their calculations and in this panelist's opinion, 50% would be more appropriate.

All but one panel member thought that the data did not demonstrate a need for biomonitoring given the concerns discussed above. The one panel member who disagreed thought that if arsenic biomonitoring in urine was being conducted, they might as well include urinary mercury; they can both readily be done at the same time.

Response: It is agreed that the use of a 100% bioavailability factor for mercury in soils is likely a significant overestimation of the actual bioavailability. However, Health Canada does not endorse the use of *in vitro* bioaccessibility studies to determine site-specific bioavailability of mercury in soil. Therefore, as recommended by Health Canada representatives on the TAC, a 100% bioavailability was used in the HHRA.

Although the HQs for inorganic mercury were below 1.0 for all receptors other than the toddler in West Flin Flon, these values were based on the 95% UCLM soil concentrations for each COI. Concentrations of mercury in soil, particularly in West Flin Flon, were highly variable, and a

significant number of properties in this community contained concentrations in excess of the PTC. Therefore, as a conservative measure, the TAC decided that the recommendation to include inorganic mercury in the biomonitoring study was justified and would be a beneficial component of the overall study.

Selenium

Comment: Panel members questioned selenium's inclusion as a COC, no one on the panel disagreed with the HHRA conclusion that there are no risks from selenium in the COIs.

Response: Although selenium was only found in excess of the soil criterion in 2% of samples, there were a number of samples that were notably higher than the criterion as well as the Provincial background concentrations. In addition, Manitoba Conservation (2007) indicated a strong correlation between selenium and other known constituents of smelter emissions and recommended that further consideration be given to the potential impacts on human health. Therefore, to ensure that public interests related to the potential effects of selenium in soil are addressed in the HHRA, selenium will remain as a COC within the final HHRA.

9.2.2 Charge Question 25

Charge Question 25. Have the key objectives of the HHRA been addressed by this assessment? (Section 1.3)

> *Objective 1: To assess risks to human receptors residing in Flin Flon, Manitoba and Creighton, Saskatchewan as a result of exposure to metals in soil and other environmental media impacted by the activities of the HBMS complex. The HHRA will estimate the contribution from individual exposure pathways and environmental media to assist in the development of risk management objectives; and,*
>
> *Objective 2: Develop risk management objectives and/or mitigation plans if unacceptable risk levels are identified in the HHRA. These risk management plans will be based on scientific approaches in consultation with the Technical Advisory Committee and the community.*

Comment: The panel discussed the objectives earlier under Charge Question 5. For the most part, they thought the HHRA had met Objective 1, but pointed out difficulties posed by some of the wording. One panel member noted that Objective 1 (as written in Section 1.3 of the HHRA) is to assess risks to human receptors as a result of exposure to metals in soil and other environmental media impacted by the activities of the HBMS complex. Inclusion of the market basket dietary intake into the risk estimates is not consistent with this objective and makes sound risk decision making more difficult. Another countered that this type of approach is valuable because it provides a picture of total exposure from all sources for the people of the community.

Response: Although the objective of the HHRA is to assess risks to human receptors as a result of exposure to metals in soil and other media impacted by the activities of the HBMS complex, to be consistent with Federal RA guidance in Canada, the HHRA included exposure *via* pathways unrelated to the complex (*e.g.*, market basket foods) to allow for the use of an acceptable HQ of 1.0. If background sources are not included in the assessment, an

acceptable HQ would be lower than 1.0. Including background sources of exposure also helps to put site-related exposure into context and is an important factor to consider for assessing overall exposure and risk levels.

9.2.3 Charge Question 26

Charge Question 26. Was the approach used for this community assessment consistent with commonly accepted methods and procedures by government agencies (such as Environment Canada, Health Canada, the Canadian Council of Ministers of the Environment, and the U.S. EPA)?

Comment: The panel members generally agreed that overall the assessment followed the commonly accepted methods. However, there are some instances where the commonly accepted methods differ from one another or with the current scientific literature.

Response: The HHRA report was prepared to be consistent with commonly accepted RA methodologies endorsed by Canadian and international agencies and to reflect the recommendations and concerns raised by members of the TAC and CAC.

9.2.4 Charge Question 27

Charge Question 27. Overall, were the input data and assumptions valid and appropriate for the Flin Flon and Creighton communities?

Comment: Overall the panel agreed that the input data and assumptions were valid and appropriate, except for those that were discussed at the meeting.

Response: Revisions have been made to the HHRA based on the IERP comments where necessary. Detailed responses to comments have been provided to justify the use of data and assumptions in response to additional comments.

9.2.5 Charge Question 28

Charge Question 28. Is the Human Health Risk Assessment presented clearly and completely?

Comment: The panel agreed that in general, the HHRA was presented clearly and completely, and that it was a very comprehensive effort. One panelist thought the authors did a good job organizing the document and the panelist was able to quickly find everything. Panel members recognized the challenges in conducting such a comprehensive assessment and thought that their suggestions and recommendations could be incorporated fairly readily.

Response: No response required.

9.2.6 Charge Question 29

Charge Question 29. Are there additional important issues that should have been addressed?

Comment: None.

References

Apostoli, P., Alessio, L., Romeo, L., Buchet, J.P., and Leone, R. 1997. Metabolism of arsenic after acute occupational arsine intoxication. J Toxicol Environ Health 52(4): 331-342.

ATSDR. 2007. Toxicological Profile for Lead. US Department of Human and Health Services, Public Health Service, Atlanta, GA. Agency for Toxic Substances and Disease Registry.

ATSDR. 2008. Toxicological Profile for Cadmium. Draft for public comment (Update). US Department of Health and Human Services, Public Health Service, Atlanta, GA. Agency for Toxic Substances and Disease Registry.

Brown, M.J., Meehan, P.J. 2004. Health Effects of Blood Lead Levels Lower than 10 mcg/dL in Children. Am J Public Health 94(1): 8-9.

Brown, M.J., Rhoads, G.G. 2008. Guest editorial: Responding to blood lead levels < 10 microg/dL. Environ Health Perspect 116(2): A60-1.

CCME. 1996a. Canadian Soil Quality Guidelines for Contaminated Sites. Human Health Effects: Inorganic Mercury. Canadian Council of Ministers of the Environment. Final Report. March 1996.

CCME. 1996b. A Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines. Canadian Council of Ministers of the Environment. The National Contaminated Sites Remediation Program.

CDC. 1991. Preventing Lead Poisoning in Young Children. Chapter 2. Background. Childhood Lead Poisoning Prevention Program. Center for Disease Control and Prevention. Atlanta, GA: U.S. Department of Health and Human Services. Available at: http://wonder.cdc.gov/wonder/prevguid/p0000029/p0000029.asp. [August 16, 2007].

CEOH (Federal-Provincial Committee on Environmental and Occupational Health). 1994. Update of evidence for low-level effects of lead and blood lead intervention levels and strategies- Final report of the Working Group. Environmental Health Directorate, Health Canada, Ottawa, Ontario.

Gupta, U.C. 1979. Boron nutrition of Crops. In: Advances in Agronomy. Vol. 31. Brady, N.C. (Ed). Academic Press Inc. New York. pp 273-307. Cited In: OMOE, 1996.

Health Canada. 2006. Guidelines for Canadian Drinking Water Quality, Supporting Documentation. Ottawa, Ontario. Available at: http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/arsenic/arsenic-eng.pdf.

Health Canada. 2007. Human Health Risk Assessment of Mercury in Fish and Health Benefits of Fish Consumption. Ottawa, ON, Health Canada, Health Products and Food Branch, Food Directorate, Bureau of Chemical Safety.

Health Canada. 2008. Health Canada 2008. Summary of Health Canada Toxicological Reference Values. May 2008 Draft. Personal Communication Louise White. Regional Health Risk Assessor and Toxicology Specialist, Healthy Environments and Consumer Safety Branch, Health Canada.

IOM. 2000. Dietary reference intakes for Vitamin A, Vitamin K, Arsenic, Boron, Chromium, Copper, Iodine, Iron, Manganese, Molybdenum, Nickel, Silicon, Vanadium, and Zinc (2000). A Report of the Panel on Micronutrients, Subcommittees on Upper Reference Levels of Nutrients and of Interpretation and Uses of Dietary Reference Intakes, and the Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Food and Nutrition Board, Institute of Medicine, NATIONAL ACADEMY PRESS. Washington, D.C.

Jacques Whitford. 2008. Metals in Soil. Flin Flon, Manitoba. Prepared for Hudsob Bay Mining and Smelting. Jacques Whitford. February, 2008.

Landrigan, P.J., Kimmel, C.A., Correa, A.., and Eskenazi, B. 2004. Children's health and the environment: public health issues and challenges for risk assessment. Environ Health Perspect 112(2): 257-265.

Lowney, Y.W., Wester, R.C., Schoof, R.A., Cushing, C.A., Edwards, M., Ruby, M.V. 2007. Dermal Absorption of Arsenic from Soils as Measured in the Rhesus Monkey. Toxicological Sciences 100(2):381-392.

Manitoba Conservation. 2007. Concentration of Metals and Other Elements in Surface Soils of Flin Flon, Manitoba, and Creighton, Saskatchewan, 2006. Prepared by Geoff Jones. Manitoba Conservation. July, 2007. Report No. 2007-01.

NAS. 1993. Pesticides in the Diets of Infants and Children. National Academy of Science. National Academy Press, Washington, D.C. Cited In: Landrigan *et al.,* 2004.

OMOE. 1996. Rationale for the Development and Application of Generic Soil, Groundwater and Sediment Criteria for Use at Contaminated Sites in Ontario. Ontario Ministry of the Environment and Energy. ISBN 0-7778-4504-5.

RAIS. 2008. Chemical-Specific Toxicity and Properties. Risk Assessment Information System (RAIS). http://rais.ornl.gov/cgi-bin/tools/TOX_search?select=chem#.

Richardson, M., Mitchell, M., Coad, S., Raphael, R. 1995. Exposure to mercury in Canada: a Multimedia analysis. Air, Water Soil Pollut 80(1-4):21-30. Cited In: CCME, 1996b.

Richardson, G.M. 1997. Compendium of Canadian Human Exposure Factors for Risk Assessment. O'Connor Associates 1155-2720 Queensview Dr., Ottawa, Ontario.

Schintu, M., Kauri, T., and Kudo, A. 1989. Inorganic and Methyl Mercury in Inland Waters. Wat. Res. 6: 699-704.

Schroeder, W.H. and R.A. Jackson. 1987. Environmental Measurements with Atmospheric Mercury Monitor having Speciation Capabilities. Chemosphere 16: 183-189. Cited In: CCME, 1996.

Schoof, R.A., Yost, L.J., Eickhoff, J., Crecelius, A., Cragin, D.W., Meacher, D.M., and Menzel, D.B. 1999. A Market Basket Survey of Inorganic Arsenic in Food. Food and Chemical Toxicology 37:839-846.

Schoof, R.A., and J.W. Yager. 2007. Variation of Total and Speciated Arsenic in Commonly Consumed Fish and Seafood. Human and Ecological Risk Assessment, 13: 946-965.

Stantec. 2008. Metals in Surface Water, Sediment, Fish and Blueberry Samples Collected near Flin Flon, Manitoba and Creighton, Saskatchewan. Stantec Consulting Ltd.

U.S. EPA. 1994. Guidance Manual for the Integrated Exposure Uptake Biokinetic Model for Lead in Children. United States Environmental Protection Agency. EPA/540/R-93/081.

U.S. EPA. 2002. Estimated per Capita Fish Consumption in the United States. EPA-821-C02-003. Office of Science and Technology, Washington D.C., U.S. Cited In: Schoof and Yager, 2007.

U.S. EPA IRIS. 1998. Arsenic – Carcinogenic Assessment for Lifetime Exposure. Integrated Risk Information System (IRIS). United States Environmental Protection Agency. http://www.epa.gov/iris/subst/0278.htm.

Wester, R. C., Maibach, H. I., Sedik, L., Melendres, J., and Wade, M. 1993. In vivo and in vitro percutaneous absorption and skin decontamination of arsenic from water and soil. Fundam. Appl. Toxicol. 20:336–340.

Yost, L.J., Schoof, R.A., and Aucoin, R. 1998. Intake of inorganic arsenic in the North American diet. Hum Ecol Risk Assess 4(1):137-152.