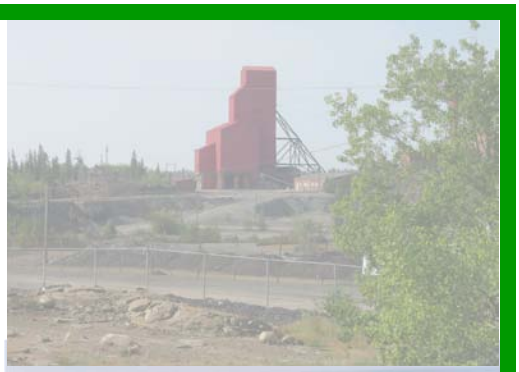
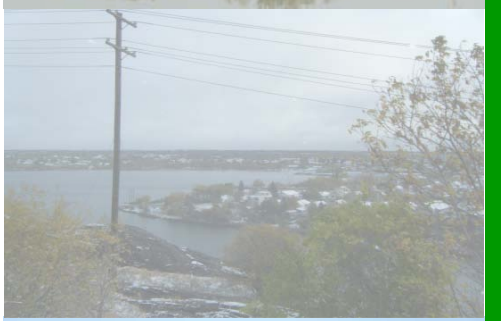


CHAPTER 7

LIMITATIONS AND UNCERTAINTIES



CHAPTER 7:

LIMITATIONS AND UNCERTAINTIES

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7.0 LIMITATIONS AND UNCERTAINTIES IN THE HUMAN HEALTH RISK ASSESSMENT

7.1 Introduction

There is no prescribed "off the shelf" model or single approach to conduct a comprehensive HHRA such as the current assessment developed to evaluate health risks in the Flin Flon area. As such, many decisions are made along the way that can influence the outcome of the assessment. The quantitative, or numerical, risk assessment requires the input of large amounts of data and numerical variables. Some of these input variables can be obtained from the general published literature, while other information must be Flin Flon-specific and were obtained from the various surveys conducted under the auspices of the Study. It must be realized that the goal of quantitative exposure assessment is to produce a conservative model to ensure that risks are never underestimated.

Each of the decisions and input variables contain some element of variability and uncertainty and can affect the outcome of the assessment to some degree. This leads to some amount of "uncertainty" with the final results and conclusions. Risk managers need to know the uncertainties surrounding the study conclusions so that they can make recommendations accordingly (*e.g.*, recommend additional experimentation or monitoring). An uncertainty analysis can pinpoint the priorities for obtaining new information, so that uncertainty can be reduced and the decision-maker can have increased confidence in the decision ultimately taken. This chapter discusses the topic of uncertainty analysis, and the related issue of sensitivity analysis. Uncertainty and sensitivity analysis both focus on the output of the HHRA and are, therefore, closely related. The purposes of the two types of analyses, however, are different. An uncertainty analysis assesses the uncertainty in model outputs that derives from uncertainty (and variability) in the inputs. A sensitivity analysis assesses the contributions of the inputs to the total uncertainty in the output, and can evaluate the relative influence a given variable may have on the overall assessment results. The general concept of uncertainty analysis is described first in this chapter, followed by a discussion of specific areas of uncertainty attached to the HHRA.

7.2 Uncertainty Analysis

A quantitative HHRA involves assigning numerical values to input parameters in an appropriate exposure or risk model to obtain a quantitative estimate of risk. Numerical values are required for parameters describing chemical concentrations in environmental media, chemical fate and transport, human exposure and toxic response. These values may be measured, assumed, prescribed or based on published literature. Variability and uncertainty in the input parameters or risk model result in variability and uncertainty in the estimate of risk. The U.S. EPA (2000) suggests that the risk characterization process maintain transparency, clarity, consistency, and reasonableness. The goal of risk characterization is to clearly communicate the key findings of the assessment and to provide a clear and balanced assessment of the strengths and limitations of the process. Risk characterization involves both scientific and policy based decision making, thereby resulting in a decision making process that blends both elements.

When assumptions are made during the risk assessment process, either because of data gaps or knowledge gaps, each can result in some degree of uncertainty in the overall conclusions. In order to understand the uncertainties within the HHRA and to ensure that the impact of these uncertainties is understood and addressed, it is important to document and characterize them. To ensure that the risk assessment does not underestimate the potential for the occurrence of adverse effects, it is necessary to make assumptions that are conservative (protective). In other

words, assumptions should be made that tend to overestimate exposure, toxicity and risk, rather than underestimate these parameters.

The following sections describe areas of uncertainty within the HHRA, and discuss the potential impacts of these uncertainties on the conclusions drawn from the assessment. Given the tendency for the assumptions described below to overestimate both exposure and toxicity, it is likely that the risk characterization errs on the side of caution and over predicts risk.

7.3 Uncertainties in the Flin Flon HHRA

When assumptions are made during the risk assessment process, either because of data gaps or knowledge gaps, each assumption results in some degree of uncertainty in the overall conclusions of the assessment. To understand the uncertainties within the HHRA and to ensure that the impact of these uncertainties is understood, it is important to document and characterize each of these.

The following sections describe areas of uncertainty within the current HHRA, and discuss the potential impacts of these uncertainties on the conclusions drawn from the assessment. Given the tendency for the assumptions used in this HHRA to overestimate both exposure and toxicity, it is considered extremely unlikely that the overall risk characterization resulted in underestimated potential health risks. The following discussion identifies uncertainties in the exposure assessment (Section 7.3.1) followed by uncertainties in the toxicological assessment and endpoints (Section 7.3.2).

Throughout the exposure assessment and risk characterization, values were generally reported with two significant figures. 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. The authors recognize that the appropriate level of precision afforded a science-based evaluation is established by the least precise component in the evaluation; however, the authors have made the science-policy decision to give all derived values a precision of two digits.

7.3.1 Uncertainties in the Exposure Assessment

The following section outlines a number of the key uncertainties related to the exposure assessment portion of the HHRA.

Area-Wide Risk Assessment Approach versus Site-Specific Approach

It was discussed earlier in this report that no specific regulatory guidance exists in Canada for undertaking an area-wide risk assessment of this scope. However, the process followed for the Flin Flon HHRA embraces the basic principles used in site-specific risk assessments (SSRAs) and area-wide risk assessments (AWRAs) conducted elsewhere in Canada. In addition, the Flin Flon Technical Advisory Committee (TAC) is comprised of stakeholders knowledgeable about the local environment, health issues and risk assessment, and the Community Advisory Committee (CAC) is comprised of interested members of the public, including representatives of

local organizations, that served to provide input and comment from the community. This provides considerable confidence that the process followed by this area-wide risk assessment and the issues addressed were appropriate and provided an accurate assessment of exposure and risks to residents of the COI.

Foundation of the HHRA is Data Generated from the 2006 Manitoba Conservation Soils Study and the Jacques Whitford Residential Soil Sampling Study

Manitoba Conservation and Jacques Whitford conducted surface soil sampling on randomly selected residential, commercial, parkland, and community properties throughout the study area. Manitoba Conservation conducted a surface soil sampling program in August, 2006 which involved the collection of soil from 93 sites in Flin Flon and 13 sites in Creighton. The majority of these sites were within 3 km of the HBMS complex. Samples were collected from the top 2.5 cm of soil within publicly accessible lands such as boulevards, parks/playgrounds, schoolyards, vacant lots and undeveloped areas. Although the Manitoba Conservation soils study effectively characterized concentrations of metals in the soils of public areas, an HHRA will commonly assume that chronic exposure events, for children in particular, will occur at the home. Exposure to metals in soil *via* incidental ingestion and dermal contact is most accurately characterized using values measured from children's play areas on residential properties.

To address uncertainties associated with characterizing concentrations of metals in soil on residential properties, Jacques Whitford completed a soil-sampling program focused on residential properties. In October, 2007, Jacques Whitford collected 369 soil samples (107 in West Flin Flon, 141 in East Flin Flon, 68 in Creighton, 18 in Channing, and 35 at undisturbed locations at varying distances from Flin Flon). The residential properties sampled as part of the Jacques Whitford program represented approximately 10% of the residential properties in the study area. Since not all properties were sampled, there is the potential that higher concentrations of COC may exist within the soils of residential properties that were not included within the study. In addition, although composite samples were collected from each property sampled, higher concentrations of COC may exist on other portions of those properties that were included in the sampling program.

Although higher concentrations than those identified within these sampling programs may exist, the available data was considered to be sufficient to provide an assessment of human health risks on a community-wide basis. The derivation of residential PTCs is considered to provide a more accurate assessment of risks on a property-by-property basis. It should be noted that identifying properties that are of concern based on a comparison of the maximum concentration to the PTC may over- or under-estimate potential risk levels. If the measured maximum concentration is significantly lower than the actual on-site maximum concentration, the potential for adverse health effects may be greater than that anticipated based on sampling data. If the sampling was completed on an isolated portion of the property that contained significantly higher concentrations of COC than the remainder of the property, then actual risk levels may be lower than those anticipated based on sampling data. This is particularly true if areas of soils with elevated COC concentrations are not within commonly used child play areas.

Projected Chemical Concentrations in Media used in the Exposure Modeling were Assumed to Remain Unchanged Over Time

Due to the continuing rate of decrease in smelter emissions arising from ongoing efforts by HBMS and improving technologies, the rate of accumulation of COC in environmental media is anticipated to progressively decrease over time. It is anticipated that the use of recent

measured data to complete an assessment of exposure and risk over the lifetime of a receptor under current conditions and moving forward may slightly underestimate exposure from some media (*e.g.*, outdoor soil) while significantly over predicting exposure from others (*e.g.,* inhalation of air). Due to a number of uncertainties associated with future production at the HBMS complex, variability in meteorological patterns, and future development in the study area, no attempt was made to predict future levels based on current emission rates and characteristics.

Metal refining has been on-going in the Flin Flon area for many years. Emissions have decreased dramatically in recent years and increased stack heights have resulted in wider regional dispersion patterns. The assumption that concentrations in soil will remain at current levels moving 80 years (*i.e.*, the assumed lifetime of a receptor) into the future may result in a moderate underestimation of exposure *via* direct soil pathways (*i.e.*, incidental ingestion and dermal contact). The direct reduction in smelter emissions will have a significant impact on the risks associated with the inhalation of ambient air. For chemicals such as arsenic and cadmium where the inhalation pathway is a major contributor to the overall risk level, emission reductions will significantly reduce exposure and risks *via* the inhalation pathway. It may also influence exposure associated with the consumption of drinking water supplied from surface water as well as the consumption of local fish. Since it is anticipated that indoor dust is influenced by concentrations of COC in ambient air, exposure from direct contact with indoor dust may also be significantly reduced. The effect on other pathways, such as the consumption of local foods (*i.e.*, home garden vegetables, local blueberries and local wild game) are less certain. While increasing concentrations in soil may increase the root uptake of COC, reduced emissions will lower the deposition of particulates onto edible aboveground portions of vegetation that are used as a food source to humans and wild game.

Any unanticipated increases in production at the HBMS complex or other factors that may result in an increase of atmospheric emissions and deposition within the study area would result in exposure and risk levels that are elevated relative to those predicted within the current HHRA. A re-evaluation of risks is recommended if there is a prolonged significant increase in concentrations of COC in environmental media.

Chemical Concentrations Reported at "Below Detection Level"

There is some uncertainty regarding the "actual" concentration of a COC for which laboratory analysis indicates a concentration below detection. Theoretically, the value of that concentration could be any value between zero and the detection limit. To ensure that concentrations were not underestimated, a value equal to the detection limit was selected to represent concentrations in environmental media in the study area (*e.g.*, concentrations in soil, blueberries, surface water, home garden vegetables, *etc*). Given that for some COC, concentrations in local fish tissue were below detection in a large proportion of samples, the 95% UCLM was derived by identifying which samples were non-detect within the ProUCL analyses. In the derivation of typical concentrations of COC in market basket food items, the authors of some of the individual studies considered it to be realistically conservative to employ a value of one-half the detection limit (*e.g.*, U.S. FDA (2004) Total Diet Study).

Chemical Concentrations in Outdoor Air

As a result of the absence of air monitoring stations located in the communities of East Flin Flon and Channing, the HHRA utilized data collected from Ruth Betts school, located in the community of West Flin Flon, to predict exposure to residents of these communities. Given the

proximity of Ruth Betts to the HBMS complex and its location relative to the predominate wind direction, representatives from Manitoba Conservation and HBMS have indicated that use of this data to predict inhalation exposure to residents of East Flin Flon and Channing is considered to be a conservative approach. Although there is uncertainty regarding localized air movements and the influence of local topography on the deposition of particulates, this assumption is anticipated to over predict exposure and risk to residents of East Flin Flon and Channing.

Although the air monitoring station located on Ruth Betts is located in West Flin Flon, the monitor located on the Provincial Building, also within West Flin Flon, consistently reports higher concentrations of various COC (*i.e.*, arsenic, cadmium, copper, and lead) associated with TSP in ambient air. As a result, data collected from the Provincial Building was utilized in the HHRA to predict exposure and risk to residents of West Flin Flon. Samples currently collected at this location are only used to report concentrations of COC associated with TSP, whereas the HHRA evaluated exposure to COC associated with the PM_{10} fraction. Although only TSP data is currently available for this location, chemical-specific correlating factors based on historical paired TSP and PM_{10} data were used to predict concentrations of arsenic, cadmium, copper, and lead associated with the respirable PM_{10} fraction (as described in detail in Appendix I). The analysis of this data indicates that this approach is an effective tool to predict metal concentrations in PM_{10} assuming that the current relationship between PM_{10} and TSP is relatively consistent with that of the 1990s. If factors such as environmental conditions or facility-related emissions have resulted in a significant change in this relationship, use of these correlating factors may result in an over- or underestimation of inhalation risks to residents of West Flin Flon.

Samples collected at the Provincial Building were not analyzed for mercury or selenium. To predict exposure and risk to residents of West Flin Flon, exposure concentrations for these COC were estimated based on measured concentrations in PM_{10} from Creighton School, adjusted according to the relationship between air concentrations derived for arsenic, cadmium, copper, and lead for West Flin Flon and Creighton (*i.e.*, an adjustment factor of 12.4 was applied to the EPCs for Creighton). If factors such as environmental conditions or facility-related emissions have resulted in a significant change in this relationship, use of this adjustment factor may result in an over- or underestimation of inhalation risks to residents of West Flin Flon.

Exposure *via* the inhalation pathway assumed that COC associated with particulates greater than PM_{10} would not reach the respiratory tissues of the lungs. While it is recognized that larger particles would be captured within the upper respiratory tract and be swallowed within mucous, the additional oral exposure resulting from this mechanism is anticipated to be very minor relative to other oral pathways. As a result, this pathway was not evaluated within the HHRA but is identified as an additional potential source of exposure.

Use of Outdoor Air Concentrations to Represent Indoor Levels

As a result of the combined efforts of HBMS, Saskatchewan Environment and Manitoba Conservation, the ambient air monitoring program provided a robust data set to characterize concentrations of COC in outdoor ambient air with data collected over an extended time period at locations in Flin Flon and Creighton. However, since air sampling has not been completed to characterize concentrations of COC in indoor air, the HHRA conservatively assumed that indoor air concentrations would be equal to those measured outdoors. The assumption that indoor concentrations are equivalent to outdoor PM_{10} concentrations is considered to be very conservative based on other studies which typically indicate lower indoor air levels as compared

to outdoor levels. For example, within the IEUBK model, the U.S. EPA (2002) recommends that concentrations of lead in indoor air are equal to 30% of the concentration measured in outdoor air. Given that Health Canada recommends that residential receptors would on average spend 22.5 hours per day indoors, this assumption may have resulted in a significant overestimation of exposure *via* the inhalation pathway. It is therefore anticipated that this approach resulted in an over-prediction of exposure and potential risk associated with the inhalation of COC in indoor air.

Selection of Appropriate Soil Ingestion Rates

There is significant uncertainty associated with the selection of an appropriate soil and dust ingestion rate, particularly for toddlers, within the completion of the HHRA. Intrinsik selected a value of 80 mg/day for toddlers as recommended in the Federal guidance on human health preliminary quantitative risk assessment (PQ_{RA}) recently published by Health Canada (2006). However, other jurisdictions, including the OMOE and the U.S. EPA, recommend the use of a soil ingestion rate of 100 mg/day for toddlers and have incorporated this value into their guidance and regulations. Both values are rooted in a similar dataset of soil tracer studies in children, and largely differ due to differing statistical analyses and methodologies used in the development of the soil ingestion rate. Neither value is incorrect and both involve appropriate interpretations of the underlying scientific data. Intrinsik selected the Health Canada regulatory recommended value as it was based upon a more recent evaluation of the scientific literature. However, it is important to note that the conclusions and recommendations of the current assessment would not have changed significantly had the slightly more conservative soil ingestion rate of 100 mg/day been used in the Flin Flon HHRA.

Indoor Dust Concentrations (Soil to Dust Relationship)

As part of an indoor dust study conducted on behalf of HBMS, concentrations of COC in indoor dust were measured in approximately 45 homes in the study area. Using co-located measured concentrations of COC in outdoor soil and indoor dust, it was shown that significant correlations exist for all COC except lead. Chemical-specific regression equations were derived to relate the concentration of a COC in indoor dust to the concentration measured in outdoor soil. These equations were used to predict concentrations of COC in indoor dust based on the outdoor soil EPC. In addition, these equations were used in the derivation of the PTCs.
Although the observed relationships between outdoor soil and indoor dust are statistically significant, the variability in indoor dust concentrations is only partially attributed to outdoor soil. Other sources such as ambient outdoor air and consumer products may also have a significant influence on indoor dust concentrations. As a result, use of the regression equations to derive indoor dust concentrations based on outdoor soil may underestimate concentrations if there are other significant sources of COC that were not encountered in homes used to derive the equations. Although a community-based HHRA cannot assess risks based on the unique conditions of every individual home, it is anticipated that the method selected to characterize exposure and risk associated with indoor dust provided a reasonably accurate assessment for the overall population.

Exposure via Wild Game Consumption

The current study did not include the collection and analysis of local wild game tissue. As a result, concentrations were predicted based on a combination of measured environmental data (*i.e.*, forest soils, wild plants, and surface water) and conservative uptake and biotransfer factors. Although this process involves a significant level of uncertainty, the assumptions

utilized are anticipated to have resulted in a large overestimation of tissue concentrations. It was assumed that all local wild game consumed by area residents would forage within a 15 km radius of the HBMS smelter. Measured concentrations of COC in forest soils and plants within this area are much higher than concentrations measured at greater distances. Given that large game such as moose and deer are likely to forage in areas further removed from the populated area, predicting tissues concentrations based on this data and assuming that residents will feed exclusively on game hunted from this area is likely to significantly over predict chronic exposure *via* the consumption of local wild game.

The exposure assessment assumed that residents would consume 52 meals per year of local wild game and that 75% (39 meals) would be large game and 25% (13 meals) would be wild birds. Given that predicted concentrations of selenium in mallard are higher than in other wild game, those receptors that have a much higher consumption rate of mallard meat may be subject to elevated exposure and risks relative to those predicted for the general population. However, given the conservatisms associated with the predicted tissue concentrations as well as exposure from other sources, it is not anticipated that unacceptable risks would occur to residents as a result of exposure to selenium in wild game meat.

Food Consumption Patterns of Flin Flon Residents

The HHRA assumed that residents of the COI have similar market basket consumption patterns as those described by Richardson (1997) to represent the general Canadian population. The results of the study completed by Richardson (1997) have been used in many HHRAs and have been referenced by regulatory agencies such as Health Canada. Although it is recognized that there is a high degree of variability in food consumption patterns among individuals, use of this data to predict exposure to COC in market basket foods is widely accepted among risk assessors in Canada.

While it was considered to be appropriate to assume that residents of the COI would consume market basket foods at similar rates as other Canadian populations, the unique characteristics of the study area required the use of a number of conservative assumptions to estimate exposure from the consumption of local foods. A local food survey was completed in which residents were requested to indicate the frequency of consuming local fish, local wild game, and local blueberries. To best represent the general population, the most common responses were selected to characterize consumption patterns. Based on the survey results, it was assumed that residents would on average consume 1.5 meals of local fish per week throughout the year, and 1 meal per week of local wild game. While these are anticipated to be conservative and realistic approximations for a typical resident, it is recognized that these assumptions will under-estimate exposure for individuals with higher consumption frequencies and over-estimate exposure to individuals that infrequently consume local fish and wild game. In addition, since there is room for a large degree of personal judgement and overall uncertainty in requesting individuals to estimate the mass of fish and wild game consumed per meal, the HHRA assumed a standard serving size of 227 g (or 8 oz) for adults and body weight adjusted values for all other age groups. This serving size is recommended by a number of agencies for predicting exposure through local fish and/or wild game consumption including Saskatchewan Environment (2004) and the Great Lakes Sport Fish Consumption Advisory Task Force (GLSFCAT, 1993). While it is recognized that many individuals will consume larger or smaller portions of local fish and/or wild game meat, it is anticipated that this value represents a typical serving size for the majority of the population.

Although some individuals may have consumption rates that are double or triple the rates assumed for the general study area population, and the resulting exposure to COC *via* these pathways may be double or triple those predicted in the HHRA, the contribution of local fish and wild game to total exposure and risk is not anticipated to be significant for any COC other than mercury. The consumption of local fish and wild game had a relatively low contribution to the total exposure to arsenic, cadmium, copper, lead, and selenium. In addition, individuals that consume local fish and wild game more frequently are anticipated to consume less market basket meats, therefore although the exposure to COC from local foods would be greater than that predicted in the HHRA, exposure from the consumption of market basket foods would likely be lower than that predicted in the HHRA.

Since concentrations of mercury were not measured in wild game, highly conservative assumptions were used to predict concentrations in edible tissues based on concentrations of mercury measured in soil, wild plants, surface water, and sediment. As a result, predicted concentrations of mercury in local wild game (EPC of 0.0068 µg/g ww) were notably higher than the literature-based concentrations used to represent market basket meats (EPC of 0.0011 µg/g ww). Despite these conservative assumptions, the contribution of wild game to total mercury exposure was still minor, generally representing approximately 2% of the total inorganic mercury exposure. It should also be noted that individuals that consume large amounts of local moose meat (with a predicted tissue concentration of 0.015 µg/g ww) may be subject to higher levels of mercury exposure than those who consume deer or game birds more frequently (refer to Chapter 4, Table 4-16).

Level of COC in Consumer Products

Background concentrations of the COC in consumer products were not evaluated in the current assessment. While some of the COC are found in several consumer products (*e.g.*, lead in some hair dye and cosmetics, and mercury in herbal remedies and skin lightening cream), the relative contribution to total exposure are anticipated to be minor compared to exposure contributions arising from other pathways, such as oral ingestion of food, soil and water. However, it should be noted that the predicted exposure and risk for mercury included exposure related to dental amalgam. Although the forward exposure and risk-calculations did not include exposure from consumer products, the derivation of the PTC for lead allocated a portion of the RTDI to generic consumer products. Additional discussion of potential exposure to COC through contact with consumer products is provided for each COC below.

<u>Arsenic</u>

Consumers may be exposed to arsenic through contact with chromate copper arsenate (CCA) treated wood, also referred to as “pressure-treated” wood (ATSDR, 2007a). Adults who saw or sand CCA- treated wood may expose themselves to arsenic through inhalation of dust particles if the appropriate safety mask is not worn. Exposure to arsenic in children may occur while playing on playgrounds constructed from CCA-treated wood. Through hand-to-mouth activities, children may inadvertently ingest particles containing arsenic. For both adults and children, additional exposure may occur through the burning of CCA-treated wood which releases arsenic into the atmosphere (ATSDR, 2007a). As of 2003, arsenic in the U.S. and Canada as a wood preservative has been phased out for residential uses such as playgrounds, picnic tables, decks, fencing and boardwalks. But, in many cases, there are many residential consumer products that still remain that contain arsenic as a wood preservative (ATSDR, 2007a; Health Canada, 2005). However, to date, Health Canada does not believe that CCA-treated wood poses any additional health risks to the public (Health Canada, 2005).

Cadmium

Exposure to cadmium through consumer products primarily occurs through cigarette smoke (ATSDR, 2008). On average, a cigarette contains approximately 1.7 µg of cadmium of which 10% is inhaled when smoked. In total, 1 to 3 µg of cadmium can be absorbed through inhalation from smoking one-pack of cigarettes per day. Additional exposure may occur through the use of cadmium plated utensils and galvanized equipment for food processing and through the use of plastics in food preparation containing cadmium stabilizers. Exposure to cadmium may also occur through the use of enamel and pottery glazes containing cadmium-based pigments (ATSDR, 2008).

Copper

Some garden products that are used as fungicides may contain copper which could be a source of copper exposure to the user through accidental exposure *via* the skin or ingestion (ATSDR, 2004).

Lead

A well known source of lead is the presence of lead-based paints in homes. The intentional addition of lead to consumer paints was prohibited in the United States in 1977, and the maximum total lead level was set at 0.06% (600 mg/kg) (Government of Canada, 2005). In January 1991, the Canadian Paint and Coatings Association voluntarily adopted this value for all Canadian produced consumer paints. Under Canadian regulation, the maximum total lead content for surface coating materials used in or around the home was not reduced from 0.5 % (5,000 mg/kg) to 0.06% (600 mg/kg) until 2005 (Government of Canada, 2005). There are many homes in which this paint is still present and may become a significant source of lead if it begins to flake or weather and then be consumed by children. Another well documented source of lead is through cigarette smoke. Cigarettes contain between 2.5 and 12.2 µg per cigarette of which 2 to 6% of lead is inhaled by the smoker (ATSDR, 2007b).

Exposure to lead may arise from various consumer products (ATSDR, 2007b). One source of exposure to lead may be through the oral consumption of non-western folk remedies such as Alarcon, Ghasard, Alkohl, Greta, Azarcon, Liga, Bali Goli, Pay-loo-ah, Coral, Koo Sar and Rueda. In some of these remedies, lead has been added into the formulations to provide colour or to increase the weight, thereby increasing the sales price. On occasion, some of these remedies have been found to increase the daily dose of lead in excess of 300 mg (ATSDR, 2007b). The use of hair dyes and some cosmetics containing lead acetate may also contribute to lead exposure. Since lead acetate is soluble in water it is easily transferred to the hand and other objects/surfaces. This can lead to oral consumption of particles containing lead through hand-to-mouth activity. Other consumer products that may increase oral exposure to lead include the use of lead-glazed ceramics and lead crystal decanters and glasses. As well, some plastic food wrappers may be printed with pigments containing lead chromates which may leach into food items (ATSDR, 2007b).

An activity such as hunting may increase lead exposure. If lead ammunition is used, then each time that the gun or rifle is discharged the dust generated will contain lead (up to 1,000 µg/m^3). Also, in those cases where animals are used as food sources, lead pellets that the animals have ingested or imbedded will increase lead concentrations. Hobbies that may increase lead exposure include the use of molten lead for casting ammunition and making fishing weights as well as toy soldiers. The use of lead solder for stained glass; use of lead glazes in pottery; use of lead-based paint; use of lead compounds for colouring agents in glassblowing; and, the

presence of lead in platinum printing and screen printing materials may also increase exposure to lead (ATSDR, 2007b).

Additionally, children may have other sources of exposure to lead including the use of inexpensive metallic jewellery containing varying levels of lead and *via i*mported vinyl mini-blinds that have used lead to stabilize the plastic. As the plastic deteriorates, lead dust may be generated which can be ingested by children who touch the blinds and then put their hands in their mouths (ATSDR, 2007b).

Mercury

Exposure to metallic mercury may occur through silver-coloured dental amalgam fillings. These fillings consist of 50% metallic mercury which may be released in small amounts over time from decay. Exposure to metallic mercury may also occur through religious practices involving the use of Azogue. Azogue is the commercial name for metallic mercury and is sold as herbal remedy or for the use in spiritual practices. It can found in capsules, glass containers or in sealed pouches and can be sprinkled in the home or mixed in water, perfume or placed into candles. Once, it evaporates, it poses a risk as it is easily inhaled. Exposure to metallic mercury vapour may also arise from damaged thermostats, fluorescent light bulbs, barometers, glass thermometers and some blood pressure devices. Lastly, metallic mercury vapors may result from the use of fungicides containing mercury (ATSDR, 1999). Other sources of mercury including mercurous chloride may be from swallowing or applying outdated medicinal products such as laxatives and through the improper use of skin lightening creams and some topical antiseptic and disinfectant agents (ATSDR, 1999).

Selenium

Selenium exposure through consumer products may occur *via* dietary supplements such as vitamins and minerals, shampoos used to treat dandruff, and creams used to treat eczema (ATSDR, 2003).

Level of COC in Cigarettes

Similar to mammals, plants require essential minerals to survive. Through evolution, they have adapted the ability to acquire these nutrients directly from soil. *Nicotiana tabacum* (tobacco) is known to be used effectively in biotechnology for the removal of metals from contaminated soils (Bernhard *et al*., 2005). Subsequently, when tobacco is dried and processed for cigarettes, it may potentially become a source of daily metal intakes for regular smokers. It has been reported that arsenic, cadmium, copper, lead, mercury, and selenium can be found in either tobacco, cigarette paper, filters and/or cigarette smoke (Arista, 2003a; Bernhard *et al.*, 2005). Arista Laboratories conducted a study to determine metals' yields in cigarette smoke from 25 brands of cigarettes (Arista, 2003a). All data obtained from their study has been validated and compared to historical values (Arista, 2003b). The findings from this study relating to the COC are summarized in Table 7-1.

Table 7-1 Metal Content in Cigarettes				
Chemicals from Cigarette Smoke[a]	***Metal Content (ng/cigarette)***[b]	***Standard Deviation (ng/cigarette)***	***Detection limit (ng/cigarette)***	***Limit of Quantitation (ng/cigarette)***
Arsenic	3.6	0.4	1.0	2.7
Cadmium	53.9	6.8	0.5	1.2
Lead	13.6	1.4	0.7	2.0
Mercury	2.4	0.2	0.15	0.25
Selenium	1.2	0.2	0.9	2.3

[a] Adapted from Arista, 2003b.
[b] Values attained from automated machinery.

Bernhard *et al*. (2005) conducted a critical review on metals in cigarette smoke in 2005. Their results pertaining to the COC are summarized in Table 7-2

Table 7-2 Metal Content in Tobacco			
Chemicals from Tobacco[a]	***Metal Content (µg/g tobacco)***	***Serum Concentration of Smokers > 10 cigarettes/day***	***Serum Concentration of Non Smokers***
Cadmium	0.5 to 1.5 µg/cigarette	0.92 µg/L	0.55 µg/L
Copper	156	1.31 mg/L	1.10 mg/L
Lead	1.2 µg/cigarette	9.0 µg/L	4.2 µg/L
Mercury	5 to 7 ng/cigarette	Not reported	Not reported
Selenium	Not reported	Not reported	Not reported

[a] Adapted from Bernhard *et al*., 2005.

From the studies above, it was shown that cigarettes do contribute arsenic, cadmium, copper, lead, mercury and selenium to the diet of regular smokers. The daily contribution of metals from cigarettes was further evaluated in an Austrian study conducted by Wolfsperger *et al*. (1994). This study showed that higher levels of cadmium and lead were found in the hair of cigarette smokers when compared to their non smoking counterparts. The findings of their study relating to COC are summarized in Table 7-3.

Table 7-3 Metal Content in Hair of Cigarette Smokers *vs.* Non Smokers		
Chemicals from Cigarette Smoke[a]	***Metal Content in Hair (µg/g hair) (P < 0.05)***	
	Cigarette Smokers	***Non Smokers***
Cadmium	0.075	0.038
Lead	3.42	1.47

[a] Adapted from Wolfsperger *et al*., 1994.

Health Canada has indicated that smoking cigarettes may contribute to an additional 0.01 to 0.04 µg/kg bw/day of arsenic exposure (Health Canada, 1993), and to an additional 0.053 to 0.066 µg/kg bw/day of cadmium exposure (Health Canada, 1994). As for selenium, Olson and Frost (1970) found an average of 0.08 mg selenium/kg (range 0.03 to 0.13 mg/kg) in a variety of cigarette tobaccos. If it is assumed that a cigarette contains 1 g tobacco and that all the selenium in tobacco is volatilized and inhaled during smoking, it can be calculated that a person smoking one pack of 20 cigarettes per day would inhale an average of 1.6 µg from this source (WHO, 1987).

While the data indicates that smoking cigarettes appears to be an additional source of all of the COC, the degree of contribution would be highly dependent on the number of cigarettes smoked per day, and the conditions under which they are consumed. As such, the potential contribution

from cigarette smoke to COC exposure could not be accurately quantified in the current HHRA, but does add an additional degree of uncertainty for those individuals who are smokers (or are routinely exposed to second-hand smoke).

The Resuspended Dust Pathway

Within the HHRA, the predicted exposure to COC *via* inhalation was assessed using ambient air concentrations measured at air monitoring stations. While it is assumed that measurements from these monitoring stations would capture dust-borne contaminants originating from the tailing and or slag piles in addition to atmospheric releases from the HBMS complex and other regional sources, they may not be reflective of conditions in which there is a significant level of dust re-suspended by wind or human activities. The U.S. EPA recommends that inhalation of resuspended dust be evaluated only if site-specific exposure setting characteristics indicate that this is potentially a significant pathway. Since it is not anticipated that this is necessary to assess exposure to the general population, the HHRA did not specifically evaluate the resuspended dust pathway. Receptors that are involved in activities that result in chronic pro-longed exposure to resuspended dust (*e.g.*, construction activities) may be subject to higher levels of exposure than those described in the HHRA.

Exposure *via* Consumption of Breast Milk

Nursing infants may be exposed to chemicals through the consumption of breast milk. It is acknowledged that consideration of the potential risks to infants and toddlers from exposure to chemical residues in breast milk should be incorporated into current risk assessment methods (U.S. EPA, 2006); however, there is a gap in the knowledge of how best to predict levels of metals in breast milk (Sharma and Pervez, 2005). Generally, this pathway is most significant for organic chemicals that show a strong tendency for bioaccumulation in biological tissues and is not assessed for inorganic chemicals. A review of the literature indicated no published methodology for consideration of inorganics in mother's milk. Metals do not bind to fat and so do not usually accumulate to higher concentrations in breast milk than in blood (Golding, 1997). Mercury concentrations in maternal blood have been reported to be about three times higher than levels in breast milk (Solomon and Weiss, 2002). Transfer coefficients from maternal blood to breast milk are low for both mercury and lead (<1), the restriction of transfer likely due the mammary gland barrier (Dorea, 2004). Levels of exposure to metals *in utero* are generally expected to be higher than during breast-feeding (Solomon and Weiss, 2002).

Physiologically-based pharmacokinetic (PBPK) models have been developed to help quantify the transfer of a chemical to the infant during breast-feeding (Clewell and Gearhart, 2002; Corley *et al*., 2003). In the case of exposure to lead, the U.S. EPA IEUBK model used in the current assessment considered maternal transfer exposures using PBPK modelling techniques. A PBPK model was developed by Byczkowski and Lipscomb (2001) to predict the kinetics of methyl mercury excretion in breast milk under different rates of maternal ingestion, and validated by comparing model predictions against available clinical data for methyl mercury distribution and elimination in mothers and their nursing infants. Unlike the IEUBK model, the PBPK model for methyl mercury has not yet been recommended or recognized by suitable regulatory agencies (*i.e.*, Health Canada, U.S. EPA) and is not widely available for use. Thus, it was not deemed appropriate to apply in the current assessment.

Conventional calculation methods for predicting uptake of organics to breast milk using a linear biotransfer factor (BTF) (Travis *et al*., 1988) have failed to predict the concentration of methyl mercury in milk (Byczkowski and Lipscomb, 2001).

Breast milk biotransfer factors (BM_{BTF}) used to predict concentrations of COC in an exposed mother`s breast milk are commonly based on the chemical`s octanol-water partition coefficient (K_{ow}). Octanol-water partition coefficients are not applicable for inorganic chemicals such as arsenic, cadmium, copper, lead, and inorganic mercury. Given that methyl mercury displays some characteristics similar to organic compounds, such as its tendency for bioaccumulation in lipids, exposure of the infant *via* consumption of breast milk was considered.

The log K_{ow} for methyl mercury has been reported to range from 1.7 to 2.54 (*i.e.*, a K_{ow} of 50 to 347) (Environment Canada, 2002). Conservatively using the high end of this range, a BM_{BTF} is calculated as follows using a standard equation (Chiao and McKone, 1995):

$$BM_{BTF} = 2.0x10^{-7} \times K_{ow}$$

where:

BM_{BTF}	=	Breast milk biotransfer factor (µg/kg milk) / (µg/day intake)
K_{ow}	=	Octanol-water partition coefficient for methyl mercury (347)

Therefore, based on the above equation, a BM_{BTF} for methyl mercury was calculated to be $6.9x10^{-5}$ (µg/kg milk) / (µg/day intake).

Using this BM_{BTF}, the concentration of methyl mercury in breast milk is calculated based on the mother`s daily exposure and body weight as follows for the community of West Flin Flon:

$$C_{BM} = \frac{EXP_{MOTHER} \times BW_{MOTHER} \times BM_{BTF}}{1000}$$

where:

C_{BM}	=	Concentration of methyl mercury in breast milk (µg/g milk)
EXP_{MOTHER}	=	Mother's total daily exposure to methyl mercury *via* all routes (0.36 µg/kg-day)
BW_{MOTHER}	=	Mother's body weight (70.7 kg)
BM_{BTF}	=	Breast milk biotransfer factor [$6.9x10^{-5}$ (µg/kg milk) / (µg/day intake)]
1000	=	Unit conversion factor (g/kg)

Therefore, the concentration of methyl mercury in the breast milk of a mother living in West Flin Flon was calculated to be $1.8x10^{-6}$ µg/g.

The U.S. EPA (1997) recommends a mean breast milk consumption rate of 742 mL/day for infants (age 1 to 6 months). Assuming that 1 mL of breast milk weighs approximately 1 gram, a daily breast milk consumption rate of 742 g/day was used to predict the exposure of an infant to methyl mercury *via* the consumption of breast milk as follows:

$$EXP_{INFANT} = \frac{C_{BM} \times BMC \times BIO_{ORAL}}{BW}$$

where:

EXP_{INFANT}	=	Infant's daily exposure to methyl mercury (µg/kg/day)
C_{BM}	=	Concentration of methyl mercury in breast milk ($1.8x10^{-6}$ µg/g milk)
BMC	=	Breast milk consumption rate (742 g/day)
BIO_{ORAL}	=	Oral bioavailability of methyl mercury (1.0; unitless)
BW_{INFANT}	=	Infant's body weight (8.2 kg)

Therefore, the infant's daily exposure to methyl mercury *via* the consumption of breast milk was calculated to be $1.6x10^{-4}$ µg/kg/day.

The predicted HQ associated with the consumption of breast milk is calculated as follows:

$$RISK_{BM} = \frac{EXP_{INFANT}}{OralRfD}$$

where:

$RISK_{BM}$	=	Risk to an infant from consumption of breast milk
EXP_{INFANT}	=	Exposure to methyl mercury from consumption of breast milk ($1.6x10^{-4}$ µg/kg/day)
Oral RfD	=	Oral exposure limit for methyl mercury (0.2 µg/kg/day)

Therefore, the HQ associated with exposure to methyl mercury *via* the consumption of breast milk for a toddler in West Flin Flon is estimated to be 0.00080. Given that concentrations of mercury in West Flin Flon soils are considerably higher than those found in other communities, HQs are likely even lower for infants living in East Flin Flon, Creighton, and Channing. Overall, exposure to COC as a result of the consumption of breast milk is considered to be minor.

Use of the Site-Specific Bioaccessibility Study

A site-specific bioaccessibility study was conducted as part of the HHRA. Although there is less uncertainty than assuming 100% bioaccessibility or using non-site-specific literature-based values, the use of bioaccessibility studies within HHRA is an emerging area that introduces several elements of uncertainty into the assessment.

There is no universally accepted method for conducting a study of this nature and as such professional judgment was used in the development of the methods and the interpretation of results. Methodological changes are emerging in the literature on an ongoing basis and the methods have not been validated for all COC. The methods and results of this study are further discussed elsewhere.

The purpose of the bioaccessibility study must be kept in context. The purpose of the study was to estimate the relative difference in bioaccessibility between metals in outdoor soil from the study area and those used in the toxicological studies used to derive the TRVs utilized in the HHRA. The study was NOT intended to measure the absolute bioavailability of metals in soil and dust from the study area.

Use of Biomonitoring as Part of the HHRA

The current HHRA should not be considered a health study in which biomonitoring was used to predict potential health risks within the Flin Flon population. Biomonitoring is often viewed as the gold standard for exposure assessment, and ultimately risk management. However, there are many pros and cons to the use of biomonitoring in such health studies. One of the largest challenges is how one interprets the results of biomonitoring work. One needs an accurate benchmark by which to compare measured biomonitoring data. Some biomonitoring data is easier to collect than others (*e.g.*, breast milk, urine, and hair *versus* blood or adipose tissue). Due to ethical reasons, one cannot typically conduct such biomonitoring activity without a clear demonstration of potential risk within the community under study. However, given that the HHRA demonstrated potential risk to receptors due to exposure to one or more of the COC, one of the recommendations for follow up work is the gathering of biomonitoring data to ground truth assessment results.

Other Key Assumptions and Related Uncertainties Include:

Receptors and their characteristics were selected in an attempt to purposely overestimate potential exposures (*e.g*., it was assumed that the residential receptor would spend 100% of their time in the Flin Flon area while consuming significant amounts of food from the local area). The residential receptor was assumed to be present in the Flin Flon area for 24 hours/day, 7 days/week, 52 weeks/year for their entire lifetime (*i.e*., 80 years).

Uncertainty in the estimation of exposure in risk assessment is generally related to a lack of specific knowledge about the site itself, the receptors of concern, or the scenarios in which those receptors may be exposed. In order to address these data gaps, data from the literature was employed as a basis for scientific judgment of values which would represent the realistic exposures. This approach was used in cases where data were lacking.

Transplacental transfer of COC was not considered in the HHRA model. While it is likely that some *in utero* exposure does occur, no method of assessment for this exposure was identified in the literature. As such, this pathway was not considered in the HHRA model but is a component in the assessment of exposure to lead within the IEUBK model.

The individual variability in physiological and behavioural parameters may be a source of uncertainty in risk assessment. Where site-specific data were lacking, receptors and their characteristics were selected in an attempt to purposely overestimate potential exposures. An example of this might be soil ingestion by children; while there were no site-specific data describing soil ingestion, or activities leading to soil ingestion, data from various literature sources such as Health Canada were employed. These data were considered comprehensive and conservative; as they were based on fecal soil content, soil and dust ingestion from all sources was included, and it is unlikely that this value would underestimate typical soil ingestion.

Pica children were not singled out as unique receptor groups, nor were these unique behaviours specifically assessed in the HHRA. If a child is known to exhibit pica behaviour, then special attention is generally paid to the child's activities. As a result, it is expected that pica related exposures, while likely in some instances, will only occur on short-term, intermittent occasions. Further discussion on children exhibiting Pica behaviours is provided in Chapter 6.

7.3.2 Uncertainties in the Hazard Assessment

The following assumptions were used in development of toxicological criteria for the COC, all of which contribute to the uncertainties inherent in the HHRA.

Animal models are used as surrogates for humans in the development of many TRVs, thereby introducing uncertainties into the risk factors due to the interspecies variability in sensitivity. For genotoxic carcinogens, it was assumed that no repair of genetic lesions occurs, and therefore, no threshold can exist for chemicals that produce self-replicating lesions. However, the existence of enzymes that routinely repair damage to DNA is well documented in the scientific literature, and the potential adverse effects arising from damage to DNA is only observed if the ability of these repair enzymes to "fix" the damage is exceeded.

In the derivation of limits by regulatory agencies, large uncertainty factors (*i.e*., 100-fold or greater) were used in the estimation of the reference dose (RfD) for threshold-type chemicals. These uncertainty factors were applied to exposure levels from studies where no adverse effects are observed (*i.e*., to the NOAEL). Thus, exceeding the toxicological criterion does not mean that adverse effects would occur. Exposures greater than the calculated toxicological criterion may also be without risk (*i.e*., below the threshold for adverse effects in humans), but this could not be, or was not, determined by the agency which derived the toxicological criterion. Humans were assumed to be the most sensitive species with respect to toxic effects of chemical. However, for obvious reasons, toxicity assays are not generally conducted on humans, so toxicological data from the most sensitive laboratory species were used in the estimation of toxicological criteria for humans.

Different age categories were used as part of the exposure and hazard assessment components of the risk assessment to permit the evaluation of potential risks to sensitive subcategories (such as the toddler). As specific toxicity data is typically not available for specific life-stages, this adds an additional layer of uncertainty to the results. In fact, the results of the assessment may distinguish a difference between life-stages which can not be validated based upon existing toxicity data for most chemicals. However, it is considered a conservative approach to use chronic lifetime risk reference values with less-than-lifetime exposures.

TRVs, because of their inherent conservatism, are widely considered protective of sensitive subgroups and lifestages. However, risk assessment, and TRV's and environmental quality guidelines for that matter, can only protect most of the people, most of the time. There can always be those individuals that are hypersensitive, and those situations require special consideration. But, risk assessments do not investigate these situations unless there is clear evidence that such a situation exists in the study area. There is no such evidence of this in the study area.

Chemical specific uncertainties are discussed in the individual chemical toxicological profiles provided in Appendix A.

In the case of arsenic, there is agreement in the published literature that the methods used to estimate the oral toxic potency of arsenic based on exposures of Taiwanese populations to arsenic in drinking water would significantly overestimate cancer risks at lower levels of exposures, such as that experienced by the general North American population. The use of such data would thus result in an overestimation of cancer risk for the populations within the study area.

In addition, the basis for the inhalation cancer potency factor for arsenic was an air concentration derived from occupational epidemiological studies. It has been suggested that because exposures to airborne arsenic would be mediated by inhalation of particulate matter, and since a higher proportion of particulate matter would be respirable in occupational settings as compared to environmental exposures, the inhalation potency of arsenic is likely overestimated for exposures associated with environmental contamination.

The toxicological profiles provided (Appendix A) are intended as overviews of the available toxicological information and opinions available at the time of their completion. As such, they relied on secondary reviews by major reputable agencies, which is standard practice in preparing toxicological profiles. It is almost always impractical and unnecessary to review all key primary papers when a number of reputable agencies have already done so. There is essentially no added value for the considerable costs and time that would be necessary to obtain and review the primary papers. In any event, the primary literature was reviewed up to what was most current at the time the profiles were prepared, with some minor revisions to certain COC based upon recent review comments and emerging information. The purpose of these reviews is also clearly stated at the front of every profile. For example, the lead profile states: "This profile is not intended to provide a comprehensive review of the available toxicological and epidemiological literature on lead compounds. Rather, the purpose of the lead toxicological profile is to: i) summarize the most relevant toxicological and epidemiological information on this substance; ii) outline any recent information that may challenge previous findings; and iii) provide supporting rationale for the lead exposure limits selected for use in the HHRA of the Flin Flon area. The following toxicological review of lead is based primarily on secondary sources, such as ATSDR toxicological profiles and other detailed regulatory agency reviews, and is supplemented with recent scientific literature."

Furthermore, the toxicological profiles were not used as a means of selecting TRVs; rather, the profiles simply provide supplementary supporting documentation for those readers who may be interested in an overview of the toxicology for each COC, but do not wish to conduct this level of research on their own.

Thus, in our opinion, the level of effort and detail that went into preparation of the profiles is appropriate and adequate for the purpose of the risk assessment. TRVs were selected based on detailed review of several of the most well-known and well-regarded regulatory agencies in the world. A number of considerations went into selecting the TRVs, including the scientific basis, the underlying science policies, the date of last major revision and others.
Several issues have not been included in the quantitative evaluation; rather, these have been discussed in Chapter 6. These include:

- Co-exposure to SO_2 and inhalation of metal fine particulates in air (PM_{10});
- Exposure to mixtures and risk health effects of common non critical endpoints (*e.g.,* cardiovascular system effects, respiratory effects, reproductive and neurological effects); and,
- Pica children and other sensitive sub-populations (seniors, pregnant women, people with compromised health and/or low socio-economic status).

7.4 Sensitivity Analysis

The purpose of a sensitivity analysis is to identify how variation in the output of a model (*e.g.*, total daily intake of a chemical) is influenced by uncertainty in the input variables. If the output variance precludes effective decision making, sensitivity analysis may be used to identify the

input variables that contribute the most to the observed output variance. Subsequently, research efforts may be initiated to reduce uncertainty in those input variables. Sensitivity analysis can also be used to simplify model structure by identifying those input variables that contribute little to the output (*e.g.*, a minor route of exposure) and thus can be removed from the analysis.

To investigate the relative sensitivity of risk predictions as part of the HHRA, the impact of key input variables on the calculated health risk related to exposures of a resident living in West Flin Flon to each of the COC was evaluated. The key variables evaluated included the following:

- Indoor Air Concentration: It was considered to be highly conservative to assume that the concentration of COC in indoor air were equal to the concentration measured in outdoor air. The effect of using the U.S. EPA IEUBK assumption that concentrations in indoor air are equal to 30% of the concentration measured in outdoor air was tested;
- Outdoor Air Concentration: Since it is anticipated that production, and subsequently air emissions, at the HBMS complex will decrease in the near future, the effect of reducing the average lifetime outdoor ambient air concentration by 50% was tested;
- Local Fish Consumption Rate: The HHRA assumed that receptors would consume 1.5 local fish meals per week throughout the year. The effect of doubling this rate to 3 meals per week was tested;
- Local Wild Game Consumption Rate: The HHRA assumed that receptors would consume 1 local wild game meal per week throughout the year. The effect of doubling this rate to 2 meals per week was tested;
- Soil/Dust Consumption Rate: The soil/dust ingestion rate for the toddler was increased from the Health Canada recommended value of 80 mg/day to the OMOE recommended value of 100 mg/day; and,
- Bioaccessibility of COC in Soil: For those COC in which a 100% bioaccessibility in soil was assumed (*i.e.*, cadmium, copper, mercury, and selenium), the effect of using the site-specific bioaccessibility analysis was tested. For arsenic and lead, the HHRA utilized the results of the one-phase bioaccessibility testing. The effect of using the two-phase results was tested as well as an assumption of 100% bioaccessibility.

The results of this analysis for each COC are presented in Tables 7-4 to 7-9.

Table 7-4 Sensitivity Analysis for the Assessment of Risks to a West Flin Flon Lifetime Composite Receptor or Toddler Exposed to Arsenic

Variable	*Value Used in HHRA*	*Adjusted Value*	*% Change in Risk Level*
Indoor Air Concentration	100% of Measured Outdoor Air Concentration	30% of Measured Outdoor Air Concentration	66% decrease in inhalation ILCR
Outdoor Air Concentration	0.084 µg/m^3	0.042 µg/m^3 (50% reduction)	50% decrease in inhalation ILCR
Local Fish Consumption Rate	1.5 local fish meals per week	3 local fish meals per week	3.1% increase in oral+dermal ILCR
Local Wild Game Consumption Rate	1 local wild game meal per week	2 local wild game meals per week	0.068% decrease in oral+dermal ILCR[a]
Soil Ingestion Rate	80 mg/day for toddler	100 mg/day for toddler	1.4% increase in total oral+dermal ILCR for toddler

Table 7-4 Sensitivity Analysis for the Assessment of Risks to a West Flin Flon Lifetime Composite Receptor or Toddler Exposed to Arsenic

Variable	*Value Used in HHRA*	*Adjusted Value*	*% Change in Risk Level*
Bioaccessibility in Soil	33% (single-phase analysis)	37% (two-phase analysis)	0.68% increase in oral+dermal ILCR
Bioaccessibility in Soil	33% (single-phase analysis)	100%	15% increase in oral+dermal ILCR

[a] The predicted ILCR decreased when the wild game consumption rate was doubled because the predicted concentration of arsenic in local wild game is lower than the literature-based concentration for market basket meats and it is assumed that receptors will eat less market basket meat if they consume more local wild game.

Table 7-5 Sensitivity Analysis for the Assessment of Risks to a West Flin Flon Lifetime Composite Receptor or Toddler Exposed to Cadmium

Variable	*Value Used in HHRA*	*Adjusted Value*	*% Change in Risk Level*
Indoor Air Concentration	100% of Measured Outdoor Air Concentration	30% of Measured Outdoor Air Concentration	66% decrease in inhalation ILCR
Outdoor Air Concentration	0.048 µg/m^3	0.024 µg/m^3 (50% reduction)	50% decrease in inhalation ILCR
Local Fish Consumption Rate	1.5 local fish meals per week	3 local fish meals per week	0.71% increase in oral+dermal HQ for toddler
Local Wild Game Consumption Rate	1 local wild game meal per week	2 local wild game meals per week	4.2% increase in oral+dermal HQ for toddler
Soil Ingestion Rate	80 mg/day for toddler	100 mg/day for toddler	3.7% increase in oral+dermal HQ for toddler
Bioaccessibility in Soil	100%	87% (single-phase analysis)	1.4% decrease in oral+dermal HQ for toddler
		46% (two-phase analysis)	5.8% decrease in oral+dermal HQ for toddler

Table 7-6 Sensitivity Analysis for the Assessment of Risks to a West Flin Flon Toddler Exposed to Copper

Variable	*Value Used in HHRA*	*Adjusted Value*	*% Change in Risk Level*
Indoor Air Concentration	100% of Measured Outdoor Air Concentration	30% of Measured Outdoor Air Concentration	66% decrease in inhalation ER
Outdoor Air Concentration	0.84 µg/m^3	0.42 µg/m^3 (50% reduction)	50% decrease in inhalation ER
Local Fish Consumption Rate	1.5 local fish meals per week	3 local fish meals per week	1.0% increase in oral+dermal HQ
Local Wild Game Consumption Rate	1 local wild game meal per week	2 local wild game meals per week	0.55% increase in oral+dermal HQ
Soil Ingestion Rate	80 mg/day for toddler	100 mg/day for toddler	3.8% increase in oral+dermal HQ
Bioaccessibility in Soil	100%	43% (single-phase analysis)	6.3% decrease in oral+dermal HQ
		35% (two-phase analysis)	7.2% decrease in oral+dermal HQ

Table 7-7 Sensitivity Analysis for the Assessment of Risks to a West Flin Flon Toddler Exposed to Lead as Assessed Using the HHRA Model

Variable	*Value Used in HHRA*	*Adjusted Value*	*% Change in Risk Level*
Indoor Air Concentration	100% of Measured Outdoor Air Concentration	30% of Measured Outdoor Air Concentration	5.5% decrease in total HQ
Outdoor Air Concentration	0.34 µg/m^3	0.17 µg/m^3 (50% reduction)	4.2% decrease in total HQ
Local Fish Consumption Rate	1.5 local fish meals per week	3 local fish meals per week	0.79% increase in total HQ
Local Wild Game Consumption Rate	1 local wild game meal per week	2 local wild game meals per week	0.31% increase in total HQ
Soil Ingestion Rate	80 mg/day for toddler	100 mg/day for toddler	12% increase in total HQ
Bioaccessibility in Soil	58% (single-phase analysis)	12% (two-phase analysis)	24% decrease in total HQ
Bioaccessibility in Soil	58% (single-phase analysis)	100%	22% increase in total HQ

Table 7-8 Sensitivity Analysis for the Assessment of Risks to a West Flin Flon Toddler Exposed to Mercury

Variable	*Value Used in HHRA*	*Adjusted Value*	*% Change in Risk Level*
Indoor Air Concentration	100% of Measured Outdoor Air Concentration	30% of Measured Outdoor Air Concentration	0.91% decrease in total HQ
Outdoor Air Concentration	0.016 µg/m^3	0.008 µg/m^3 (50% reduction)	0.70% decrease in total HQ
Local Fish Consumption Rate	1.5 local fish meals per week	3 local fish meals per week	2.1% increase in total HQ for inorganic mercury; 77% increase in total HQ for methyl mercury
Local Wild Game Consumption Rate	1 local wild game meal per week	2 local wild game meals per week	0.48% increase in total HQ
Soil Ingestion Rate	80 mg/day for toddler	100 mg/day for toddler	19% increase in total HQ
Bioaccessibility in Soil	100%	1.5% (single-phase analysis)	74% decrease in total HQ
		3.5% (two-phase analysis)	72% decrease in total HQ

Table 7-9 Sensitivity Analysis for the Assessment of Risks to a West Flin Flon Toddler Exposed to Selenium

Variable	*Value Used in HHRA*	*Adjusted Value*	*% Change in Risk Level*
Indoor Air Concentration	100% of Measured Outdoor Air Concentration	30% of Measured Outdoor Air Concentration	0.32% decrease in total HQ
Outdoor Air Concentration	0.052 µg/m^3	0.026 µg/m^3 (50% reduction)	0.22% decrease in total HQ
Local Fish Consumption Rate	1.5 local fish meals per week	3 local fish meals per week	19% increase in total HQ
Local Wild Game Consumption Rate	1 local wild game meal per week	2 local wild game meals per week	1.6% increase in total HQ
Soil Ingestion Rate	80 mg/day for toddler	100 mg/day for toddler	0.65% increase in total HQ
Bioaccessibility in Soil	100%	55% (single-phase analysis)	0.97% decrease in total HQ
		57% (two-phase analysis)	0.97% decrease in total HQ

Based on the above sensitivity analysis, the variable with the most significant influence on the outcome of the HHRA varied from chemical to chemical. Since arsenic, cadmium and copper

can have direct effects on sensitive respiratory tissues, assuming that indoor air concentrations were 30% of those measured outdoors, and a 50% reduction in outdoor air concentrations, both resulted in a large decrease in risk *via* inhalation. For lead and mercury, direct soil contact was a significant source of overall exposure, therefore, increasing the soil ingestion rate from 80 mg/day to 100 mg/day resulted in a significant increase in risk for the toddler (*i.e*., 12% and 19% increase in total HQ for lead and mercury, respectively). For lead, the use of the two-phase bioaccessibility analysis rather than the single-phase would result in a large reduction in overall risk (*i.e*., 24% decreases in the total HQ). For mercury, the HHRA did not utilize the results of the bioaccessibility analysis and instead assumed 100% bioaccessibility in soil. Use of either the single (1.5%) or two-phase analyses (3.5%) would have a significant reduction in overall risk, dropping from an HQ of 1.9 for the toddler in West Flin Flon to 0.49 or 0.52, respectively. Although the most significant source of exposure and risk for selenium is from the consumption of market basket foods, doubling the local fish consumption rate from 1.5 to 3 meals per week resulted in a 19% increase in the overall selenium HQ (from 0.92 to 1.1 for the toddler in West Flin Flon). Since the primary route of exposure to methyl mercury is through the consumption of local fish, this adjustment resulted in a 77% increase in the total methyl mercury HQ (from 1.9 to 3.3 for the toddler in West Flin Flon).

Sensitivity Analysis for the IEUBK Model

Exposure and risk estimates for lead were also assessed using the IEUBK model. Table 7-10 provides the % change in the predicted geometric mean blood lead concentration (BLL) for a child living in West Flin Flon.

Table 7-10 Sensitivity Analysis for the Assessment of Risks to a West Flin Flon Child Exposed to Lead as Assessed Using the IEUBK Model

Variable	***Value Used in HHRA***	***Adjusted Value***	***% Change in Geometric Mean BLL***
Indoor Air Concentration	100% of Measured Outdoor Air Concentration	30% of Measured Outdoor Air Concentration	2.2% increase in BLL (4.5 to 4.6 µg/dL)
Outdoor Air Concentration	0.34 µg/m^3	0.17 µg/m^3 (50% reduction)	2.3% decrease in BLL (4.5 to 4.4 µg/dL)
Local Fish Consumption Rate	1.5 local fish meals per week	3 local fish meals per week	0.89% increase in BLL (4.5 to 4.5 µg/dL)
Soil Ingestion Rate	IEUBK Default Values 85 mg/day for 0-1 years 135 mg/day for 1-2 years 135 mg/day for 2-3 years 135 mg/day for 3-4 years 100 mg/day for 4-5 years 90 mg/day for 5-6 years 85 mg/day for 6-7 years	Health Canada Values (adjusted for IEUBK age groups) 50 mg/day for 0-1 years 80 mg/day for 1-2 years 80 mg/day for 2-3 years 80 mg/day for 3-4 years 20 mg/day for 4-5 years 20 mg/day for 5-6 years 20 mg/day for 6-7 years	38% decrease in BLL (4.5 to 2.8 µg/dL)
Absolute Bioavailability in Soil	29% (single-phase analysis)	6% (two-phase analysis)	29% decrease in BLL (4.5 to 3.2 µg/dL)
Absolute Bioavailability in Soil	29% (single-phase analysis)	85% ABA (100% IVBA)	64% increase in BLL (4.5 to 7.4 µg/dL)

Based on the sensitivity analysis completed for the IEUBK model, adjustments to outdoor and indoor air concentrations and doubling the local fish consumption rate had minor impacts on the predicted geometric mean BLL. Given that the Health Canada recommended soil ingestion rates are notably lower than the IEUBK default values, use of the Health Canada values resulted in a significant (38%) decrease in the predicted BLL. Use of the two-phase

bioaccessibility analysis as opposed to the single-phase analysis would also result in a significant decrease (29%) in the predicted BLL.

7.5 References

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