

**APPENDIX F**

**COMMENT RESPONSE TABLE**



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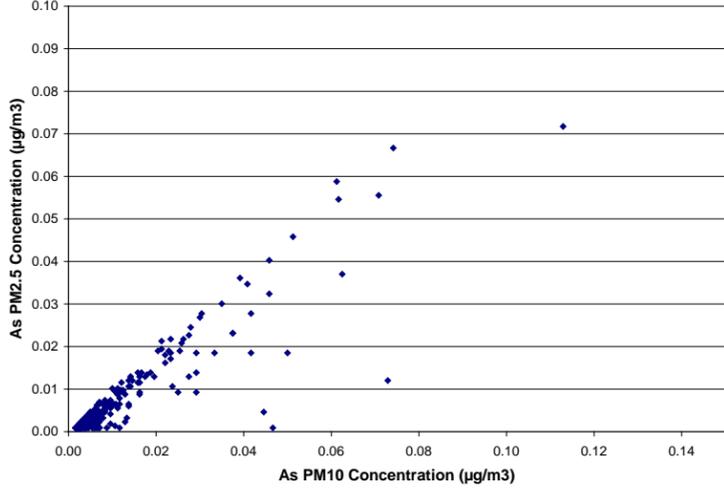
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## APPENDIX F: COMMENT RESPONSE TABLE

Comments Received on the HHRA Terms of Reference (ToR)					
Comment #	Reviewer	Page/Section	Issue/Comment	Intrinsic Response	Change Made in Report
1	Manitoba Water Stewardship	Section 1.0, introduction, page 2	Add Manitoba Water Stewardship to the TAC committee	Manitoba Water Stewardship will be added to the list of members of the TAC	Revision made
2		Section 2.2, Study Scope, Selection of Exposure Pathways and Supporting Rationale, Local Fish, page 9	From the conference call last week, I think Elliot noted they are doing (or planning to do) a survey of the community to determine fishing habits (where, when, how much is kept) and consumption amounts to assess what fish sampling needs be done. I think this task should be noted in the ToR.	Page 10 of the ToR briefly discusses recommendations for an additional fish sampling program to be completed to assist in the assessment of exposure from the consumption of local fish. This discussion will be expanded to include further details of the recommended program and types of information it is anticipated to provide.	Revision made
3			I presume mercury will be one of the COPCs included in the HHRA based upon determinations made in Section 4.2.2. Mercury should be a definite COPC when considering fish muscle tissue. Mercury is a well-known element that bio-accumulates up the food-chain and this potential pathway will likely be a concern for those in the community that eat locally caught fish.	Mercury will be retained as a COPC for evaluation in the HHRA as a result of elevated levels measured in soil. It is acknowledged that consumption of local fish can be a significant exposure pathway for methyl mercury. Assessment of this pathway will be included within the HHRA.	No change required
4			I do not think details of sample collection protocols would not be needed in the ToR, but maybe it should be noted that a set of protocols/instructions (e.g., tagging each specimen with information such as where and when caught, how to store sample, etc.) would be provided to those donating fish samples.	This type of information will be collected if a local fish study is completed.	No change required
5			It probably should also be noted that if adequate fish samples cannot be obtained through donations from recreational anglers, a sampling program may be required.	It is agreed that if adequate fish samples are not obtained through donations, a sampling program may be required to provide concentrations of COPCs in fish tissue for inclusion in the HHRA. Should this be required, a sampling protocol will be developed and circulated to the TAC for review.	No change required
6			Not for inclusion in the ToR, but a comment for considerations when determining what fish samples will be collected. Because mercury bio-accumulates up the food-chain, predator fish such as walleye and northern pike usually have higher concentrations in their muscle tissues than insect feeding fish such as whitefish or suckers. Mercury concentrations also tend to be higher in larger and older than in smaller and younger individuals of these species. For this reason, as wide a size range of individual fish in walleye and pike as possible is best for sampling. Results from a good size range helps generate better regression analyses that can be used to predict concentrations at given size. This is usually less important for whitefish or sucker species because their mercury muscle tissue concentrations are usually much lower. Generally, this also appears to be less important for other metalloids or metals such as arsenic, cadmium, chromium, copper, lead, nickel, and zinc since they do not appear to bio-accumulate in the muscle tissues.	Information provided in this comment is appreciated and will be considered when evaluating the adequacy of samples obtained to characterize concentrations of mercury in local fish.	No change required
7		Section 2.2, Study Scope, Selection of Exposure Pathways and Supporting Rationale, Drinking Water, page 10:	It is suggested to add a sample collection point after treatment at each town drinking water distribution plant to understand the base supply to all households.	It is agreed that collection of water samples following treatment but prior to distribution would provide an important piece of information for the HHRA. This issue has been discussed with HBMS and members of the TAC and it is anticipated that these samples will be collected. The ToR will be revised to reflect this once this has been confirmed.	Revision pending confirmation of study
8			Increase the number of residences within each community for sampling from their taps to increase sample robustness;	This issue has been discussed with HBMS and members of the TAC and it is anticipated that additional samples will be collected. It has been proposed to complete a large one day sampling event in which water samples are collected from multiple homes in Flin Flon and Creighton, as well as the distribution centres. The ToR will be revised to reflect this once this has been confirmed.	Revision pending confirmation of study
9			Newer and older homes should be included in the selection from each community (i.e., Flin Flon and Creighton) to better determine if plumbing materials in older homes may create increased exposure than newer homes.	A detailed assessment of the influence of plumbing on drinking water content is outside of the scope of this assessment.	No change required
10			It is not clear how long the weekly household sampling will occur. Sampling from the distribution centres on a weekly basis may be more than needed if household sampling is planned for more than a month. Often, water sampling on a quarterly basis for a treatment plant is recommended to account for seasonal variation. However, a few water samples from each community facility should be collected at the same times as some household sampling to increase sample accuracy and reduce sample bias.	Information provided in this comment is appreciated and will be considered when evaluating the adequacy of this data set.	No change required

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11	Manitoba Conservation (reviewer 1)	page 5, 2nd paragraph	Though historical exposures in its risk assessment will not be assessed, this point may not have much significance for the conduct of the risk assessment itself. However, the historical emissions data are of relevance for putting the risk associated with future releases into perspective (i.e., the risk in previous years was higher). Data submitted by HBM&S to Manitoba and the Clean Environment Commission hearings in the early 1980's showed that particulate matter releases from HBM&S prior to 1981 were around 14,000 tonnes per year. These data on particulate releases is different from the stated particulate matter releases cited in this Terms of Reference of 7,150 tons prior to 1974 and 6,834 tons per year after 1974.	<p>The risk assessment will address risks under current conditions and moving forward assuming conditions remain generally similar. As discussed during TAC meetings, risks associated with historic conditions will not be addressed in the current HHRA.</p> <p>HBMS has indicated that while the data submitted to the Clean Environment Commission reports a peak in emissions during the 1970's and 1980's of approximately 14,000 tons per year, the annual average during the period of 1975 to 1995 is approximately 6,800 tons per year. The ToR has been revised for clarity.</p>	Revision made
12		2,1 Background Information, page 5, last paragraph:	The statement is made that "it was not until the completion of the surface soil sampling program conducted by Manitoba Conservation in August, 2006 that the issue of risk to human health became an issue of concern." We would prefer that this statement be modified to indicate that metals in soil survey resulted in a process to further investigate and assess the potential to human health from soil and other exposures.	This statement will be revised as requested.	Revision made
13		2.2 Study Scope: Time Scale of Risk Assessment, page 7:	This HHRA will not evaluate "historical impacts" due to "historical metal exposures". It is recommended that the future exposure assessment consider, as appropriate, past exposures that might affect the basis or baseline to which the incremental exposures are added to. This would recognize that there have been past (potentially higher exposures) and that the assessment is not assuming "normal" exposures as one possibly might do with a "green fields" development.	The risk assessment will address risks under current conditions and moving forward assuming conditions remain generally similar. Exposure limits used within the HHRA to predict risks will be selected to be protective of sensitive receptors including those that may have pre-existing health conditions. As discussed during TAC meetings, risks associated with historic conditions are better addressed through alternative studies.	No change required
14		Ambient Outdoor Air, page 10:	<p>While the HBMS monitors may provide data for heavy metals in PM<sub>10</sub>, these monitoring stations are located much farther from the HBMS smelter and so record lower heavy metal concentrations. As well, the heavy metal levels measured at the Provincial building would be most representative of the downtown area of Flin Flon.</p> <p>Using only data from the Ruth Betts and Creighton stations will underestimate the risk from the air inhalation pathway. The resulting risk will therefore be understated for those members of the Flin Flon and Creighton communities who spend any significant amount of time in the downtown area of Flin Flon for recreation, work or other reasons.</p> <p>It is possible to use the heavy metal in TSP data from the Provincial Building. From previous work (Draft report "Assessment of Ambient Air Concentrations of Arsenic in the Flin Flon Area", April 11, 2006), the arsenic levels in PM<sub>10</sub> were found to be strongly correlated to the arsenic levels in TSP. The average ratio of arsenic levels in PM<sub>10</sub> to TSP was about 0.74. (See Table A-10 and Figure A-6 from the draft report). While this analysis had been done only for arsenic, analyses for the other heavy metals will probably also reveal similar strong correlations.</p>	<p>The relationship between concentrations of metals within the PM<sub>10</sub> component and TSP will be further analyzed. Regression equations derived based on concurrent data for PM<sub>10</sub> and TSP may be used to estimate concentrations of COPCs in PM<sub>10</sub> at the Provincial building based on measured TSP concentrations to allow for the use of Provincial building air monitoring data in the HHRA. The proposed methodology will be provided to the TAC for review prior to incorporation in the HHRA. The TOR will be revised to reflect this approach.</p> <p>It is recognized that exposure to COPCs in ambient air may be a significant exposure pathway. The HHRA will address all potential exposure pathways including the inhalation of particulates in outdoor and indoor air.</p>	Revision made

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			<p align="center"><b>Table A-10 Ratios of Arsenic Particulate Size Fractions</b></p> <table border="1"> <thead> <tr> <th>Data Summary</th> <th>Ratio</th> <th>Statistical Parameters for Ratio</th> </tr> </thead> <tbody> <tr> <td>Provincial site</td> <td rowspan="5">PM<sub>10</sub>/TSP</td> <td>Geometric mean: 0.68</td> </tr> <tr> <td>December 13, 1990 – December 28, 1998</td> <td>Median: 0.77</td> </tr> <tr> <td>(Days in which PM<sub>10</sub> &gt; TSP were excluded from analysis since these data were ambiguous)</td> <td>Arithmetic mean: 0.74</td> </tr> <tr> <td></td> <td>Correlation coefficient: 0.91 (strong)</td> </tr> <tr> <td></td> <td>Number of samples: 336</td> </tr> </tbody> </table>  <p align="center"><b>Figure A-6. Correlation of Arsenic in PM<sub>2.5</sub> with Arsenic in PM<sub>10</sub></b></p> <p>In reviewing this HHRA, the overall impression is that the emphasis is primarily on the soil pathway, with the significance of the other pathways being downplayed. Given the high levels of heavy metals associated with particulates in the air, the risk from the air pathway should not be minimized.</p>	Data Summary	Ratio	Statistical Parameters for Ratio	Provincial site	PM <sub>10</sub> /TSP	Geometric mean: 0.68	December 13, 1990 – December 28, 1998	Median: 0.77	(Days in which PM <sub>10</sub> > TSP were excluded from analysis since these data were ambiguous)	Arithmetic mean: 0.74		Correlation coefficient: 0.91 (strong)		Number of samples: 336		No change required
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15		4.2.4 Identification of Exposure Pathways, page 29	<p>One of the inhalation exposure pathways identified was “direct inhalation of COPCs predicted in indoor air”. Other than a statement on page 34 that “EPCs in some environmental media (e.g., indoor air) may need to be estimated”, this HHRA does not provide any information to address how the concentrations of the metals in the indoor air will be estimated. Previously, on page 10, there was no mention of the indoor air pathway as one of the exposure pathways being considered.</p> <p>The exposure calculation for metals from the ingestion of indoor dust (Exhibit 2.0) appears not to be related to the quantity of dust present, rather just the concentration of metal in the dust. One would expect that the level of indoor dust to vary with season and accordingly impact the potential amount for ingestion?? An explanation of Exhibit 2.0 would be helpful. If quantity of dust ingested did vary by its availability, there would be merit in determining indoor dust level quantified by season.</p> <p>It is noted that a number of “constants” are being contemplated for use such as the “DIRA” (the Canadian per capita dust intake rate in g/day); what value will be used for “DIRA” and what is the basis for this value. Similarly, other “constants” are referenced (e.g., SIRA - annualized soil intake rate, AFsoil - adherence factor for soil, RVIR - home-produced belowground produce (g/kg/day), etc.) – it would be helpful that the values to be used and the rationale in support of these values be eventually documented.</p>	<p>The discussion on page 10 will be revised to include the inhalation of COPCs in indoor air as well as the methodology used to predict indoor air concentrations. Concentrations of COPCs associated with airborne particulates in indoor air may be a factor of concentrations in ambient outdoor air, outdoor soil, indoor dust, as well as household items and furnishings. Since measurements of COPC concentrations in indoor air are not available, a methodology for estimating these concentrations must be developed. Within the IEUBK model (Integrated Exposure, Uptake, and Biokinetic model), the U.S. EPA assumes that the concentration of lead in indoor air is 30% of the concentration measured in outdoor air. Since the origin of this assumption is not well documented, the applicability of this value to predict indoor air concentrations of other COPCs has not been established. As a result, use of alternate values to relate indoor air concentrations to measurements in outdoor air may be considered in the HHRA. Alternatively, concentrations in indoor air may be predicted using concentrations measured as part of the indoor dust survey and literature-based measurements of total PM<sub>10</sub> found in residential buildings.</p> <p>The assessment of exposure <i>via</i> the ingestion of soil or dust uses a very conservative estimate of the typical amount of soil or dust ingested on a daily basis for receptors of different ages. This value is used regardless of the amount of dust present in a given environment and is multiplied by the site-specific concentration of COPCs in soil or dust to predict the amount of a COPC ingested. This approach is endorsed by numerous regulatory agencies such as Health Canada and the U.S. Environmental Protection Agency. It is considered to be a conservative reflection of the amount of soil or dust ingested throughout the year.</p> <p>The soil and dust intake rates along with numerous other exposure parameters are presented in Tables 4-2 to 4-7 along with references of their origin. The HHRA will provide additional discussion regarding the selection and use of these values.</p>	<p>Revision made</p> <p>No change required</p> <p>No change required</p>
16		3.4 Exposure Estimation Methods, pages 36-44:	This HHRA does not provide any details on the sources of data that will be used for many of the parameters listed in the equations (e.g., RAF – relative absorption factor for inhalation, food, etc.; FPLF – food preparation loss factor, FVIR – home produced fruit consumption rate (g/kg/day).	Consumption rates are provided in Tables 4-2 to 4-7 along with references of their origin. Details regarding all exposure parameters and assumptions such as chemical specific absorption factors and food preparation loss factors will be provided in the HHRA.	No change required
17		4.5 Risk Characterization, page 48	The HHRA seems to consider only long-term carcinogenic (ILCR) and non-carcinogenic risks (hazard quotient). There does not seem to be any mention of the risk associated with acute exposures. The acute health risks are an issue since the heavy metals data from the Provincial building have identified instances where the heavy metal concentrations exceed short-term exposure criteria.	The focus of the study is intended to be soil-borne exposures, which are typically evaluated on a chronic basis, since the outcome will be ‘safe’ soil levels for the Flin Flon/Creighton area. Inhalation exposure will be considered and as such acute risks related to short-term events will be evaluated. The TOR will be modified to reflect this.	Revision made
18		Appendix B. Bioaccessibility Testing Protocol	There seems to be a significant amount of uncertainty associated with the proposal to do bioaccessibility testing. For example, there are no standard tests and some of the methods have only been validated for some metals using <i>in vivo</i> data. This degree of uncertainty increases the level of uncertainty associated with the soil component of this HHRA.	Agreed, prior to conducting a bioaccessibility study, the methods will be discussed with Health Canada and other technical experts on the TAC to ensure the study is conducted in a manner that is acceptable to the TAC.	No change required
19	Reviewer 2	2.2 – Time Scale of Risk Assessment	This Section states the Human Health Risk Assessment (HHRA) will not evaluate historical impacts or risks. The reasons why this is not to be included as part of this HHRA should be explained in detail to ensure that all stakeholders (including the public) fully understand the scope of the HHRA. People who have potentially been exposed to metals and other elements of concern for the past several decades may still be concerned about their personal health, even if the HHRA shows that present and future levels do not present a risk.	The risk assessment will address risks under current conditions and moving forward assuming conditions remain generally similar. As discussed during TAC meetings, risks associated with historic conditions will not be addressed in the current HHRA.	No change required
20		3.2.1 – Soil Sampling Program	This section will have to be amended to reflect changes in methodology and to include details of the laboratory methods used to prepare and analyze the samples.	The HHRA will provide a detailed description of the sampling methodology and the laboratory methods used in the residential soil sampling program. It is also anticipated that this will be provided in the report prepared by Jacques Whitford following the completion of the sampling program.	No change required

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21		4.2.2.1 – Chemical Screening	<p>This section indicates that Canadian Council of Ministers of the Environment (CCME) guidelines for human health will be used to screen out and identify the chemicals of potential concern (COPC), and that OMOE guidelines and United States EPA preliminary remediation goals will be used to screen elements for which there are no applicable CCME guidelines. With this in mind, assurance should be made that soil concentration data for the elements beryllium (Be), iron (Fe), zinc (Zn), and perhaps antimony (Sb) (see Table 4-1) will also be collected during the residential soil sampling program even though these elements do not have a CCME guideline for human health.</p> <p>CCME and OMOE guidelines are based on 20% of the toxicological reference value, while U.S. EPA preliminary remediation goals (PRGs) are based on 100% of the toxicological reference value. Health Canada (2004, <i>Federal Contaminated Sites Risk Assessment in Canada Part III: Guidance on Peer Review of Human Health Risk Assessments for Federal Contaminated Sites in Canada</i>) recommends that when the U.S. EPA PRGs are used they should be divided by a factor of 5 (<i>i.e.</i>, reduced so that they are based on 20% of the toxicological reference value) in order to be comparable to CCME and OMOE guideline derivation policies. Was this taken into account in the EPA PRGs presented in Table 4-1?</p>	<p>Soil samples collected as part of the residential soil sampling program will undergo an ICP scan which will provide an analysis for all metals listed in Table 4-1.</p> <p>The EPA PRGs were not divided by a factor of 5 in Table 4-1 of the ToR. It is agreed that this should be done to allow them to be comparable to CCME and OMOE standards. The ToR will be revised.</p>	<p>No change required</p> <p>Revision made</p>
22		4.3.1 – Background Exposure	<p>This section indicates that background exposures and risks will be considered in the HHRA. Were background samples collected as part of the residential soil sampling and dust sampling programs? If not, how will the HHRA determine background exposure?</p>	<p>It is anticipated that the residential soil sampling program will provide additional background soil concentrations to supplement the data collected by Manitoba Conservation. The protocol for the indoor dust sampling program did not include sampling from locations that would be reflective of background concentrations.</p>	<p>No change required</p>
23		4.3.4.1 – Outdoor Soil/Indoor Dust Exposure	<p>This section indicates that a relative absorption factor (RAF) will be used in estimating the exposure to ingestion of metals in soil and dust, and that this will be largely based on the <i>in vitro</i> analysis outlined in Appendix B. Since the methodologies cited in Appendix B appear to have been developed fairly recently and have not been proven for all COPCs, it might be prudent to carry out this analysis using the most conservative methodologies available (<i>i.e.</i>, worst-case scenario).</p>	<p><i>In vitro</i> bioaccessibility results will only be used as appropriate. Default literature based absorption factors will be used where bioaccessibility results are deemed inappropriate.</p>	<p>No change required</p>
24		4.3.4.2 - Exposure Via Home Garden Produce and Wild Berry Consumption	<p>Assuming that garden produce and berries are usually consumed fresh (<i>i.e.</i>, not in a dried state), then the concentration of contaminants in the equations in this section should be expressed on a fresh weight rather than a dry weight basis? Keep in mind that, depending on the species, water can account for as much as 95% of the total fresh weight of garden vegetables.</p> <p>As with the estimates of exposure from soil/dust, a relative absorption factor (RAF) for food will also be incorporated into the equations to estimate exposure from garden produce and berries. How will this RAF be determined?</p>	<p>Concentrations of chemicals measured in produce and berries are typically provided on a dry weight basis. This is accounted for in the exposure assessment through the use of a dry weight consumption rate that is derived using ingestion rates measured on a fresh weight (or wet weight) basis which are then adjusted by accounting for the water content of the food item. Since food categories such as “home-grown root vegetables” include a number of different types of produce, a conservative value reflective of the range of water content in root vegetables is used.</p>	<p>No change required</p>
25		4.3.4.3 – Background Market Food Basket Exposure	<p>Will any local grocery store food items be analyzed for COPCs or will the HHRA rely solely on published literature (<i>e.g.</i>, Health Canada (2005) <i>Total Diet Study</i>).</p>	<p>It is anticipated that the HHRA will rely on values within published literature such as the Health Canada Total Diet Study.</p>	<p>No change required</p>
26		4.4.1 – Selection of Toxicity Reference Values for the Assessment	<p>It would have been helpful if this section had provided a more definite (final) list, with supporting text, of the exposure limits being considered for use in this HHRA, rather than just mentioning several potential sources of this information.</p>	<p>More detailed information was not provided partly because the final list of COPCs has not yet been determined. The final list will be determined following the review of the residential soil sampling program. A list of the selected exposure limits can be provided to the TAC once the COPCs have been selected.</p>	<p>No change required</p>
27		4.4.2. – Bioavailability	<p>The document indicates that bioavailability tests will be conducted on a subset of samples collected from the residential soil sampling program. Bioavailability can be influenced by the soil texture (<i>i.e.</i>, proportional amounts of sand, silt, and clay), particle size, and the amount of organic matter. These and other characteristics were likely quite variable in the residential soil samples, which may make it difficult to extrapolate the bioavailability</p>	<p>The bioaccessibility study will be conducted on a large number of soil samples, with the intention of capturing varying soil textures from the community.</p>	<p>No change required</p>

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			results from a subset of samples to the entire set of soil samples.		
28		4.5.1 - Calculation of Risk Levels	Health Canada (2004) suggests that Hazard Quotients be summed for chemicals that have an affect (non-cancerous) on the same organ, and that risks for chemicals that produce the same form of cancer also be summed. Will the HHRA incorporate this recommendation into the calculation of overall risk?	This recommendation will be incorporated into the TOR. The HHRA will consider summing individual risks for chemicals with similar critical effects (i.e., those that act via a similar toxicological mechanism on a similar target tissue).	Revision made
29	Saskatchewan Health	3.2.1- Soil Sampling Program (page 14)	<u>Removal of grass cover for soil sampling:</u> No mention was made if a record would be kept regarding what type of soil cover there is at the sampling site. It could be useful to keep a detailed record indicating whether or not grass cover was present at the site and if it was removed prior to soil sampling. If a grass cover is present on the site, then the levels of contaminants measured in the soil sample is likely higher than the actual exposure.	Detailed field notes were collected along with all residential soil and dust data detailing this type of information (see example attached below).	No change required
30		3.2.1- Soil Sampling Program (page 14)	<u>Selection of homes for residential soil sampling:</u> It is recommended that 200 residential homes be used for soil sampling. We are interested to know how the residential homes will be selected.	Soil samples were collected for 188 locations (66 locations in West of Ross Lake, 62 locations East of Ross Lake, 30 locations in Creighton, 10 locations in Channing, and 20 remote locations). 12 duplicate samples were also collected for QA/QC purposes. Homes were primarily selected on a volunteer basis.	No change required
31		3.2.2 – Indoor Dust Sampling Program (page 14)	<u>Indoor dust sampling:</u> It appears that the indoor dust sampling will take place at the same homes that are used for residential soil samples, but this is not specified in the TOR nor is how many homes will be sampled for indoor dust and how they will be selected.	Dust samples were collected from a total of 54 locations (45 houses, 5 schools and 4 daycares). 44 of these locations were also sampled for soil. Houses for dust sampling were selected from volunteers of those involved in the soil component of the study.	No change required
32		4.2.3 – Receptor Identification and Characterization (page 24)	<u>Assumptions in receptor characteristics:</u> Assumptions were made for swim events per year-recreational and duration of swimming event -recreational. How were these assumptions derived? This may be a point that could be brought up with the CAC to see if these assumptions are reasonable. In addition, it is not clear what EF <sub>r</sub> (exposure frequency – recreational) is, nor how it was obtained or where it will be used.	The number of swim events per year and the duration of these events have been slightly modified from those values presented in the original draft of the ToR. The number of swim events per year is now 30 for all age classes with the exception of the infant. This value was taken from the U.S. EPA (2003) User's Manual for the Swimmer Exposure Assessment Model (SWIMODEL) Version 3.0. It is based on the 90 <sup>th</sup> percentile value for the number of swimming events per month (10) for a freshwater swimming pool taken from the U.S. EPA Exposure Factors Handbook (1997). Assuming that receptors would be able to swim in lakes in the Flin Flon-Creighton area for 3 months per year (mid-June to mid-September), the number of swim events per year is 30 (10 events/month x 3 months/year). The duration of these events was also taken from U.S. EPA (2003) and represents the recommended values for long-term exposures (2.3 hours/event for the toddler and child, 1.7 hours/event for the teen, and 1.3 hours/event for the adult). The exposure frequency-recreational value represents the amount of time a receptor would spend on a recreational property such as a park or playground. The value presented in the terms of reference was based on those values recommended in Table 6 of Health Canada's (2006) <i>Federal Contaminated Site Risk Assessment in Canada Part 1: Guidance on Human Health Preliminary Quantitative Risk Assessment</i> . Based on a receptor spending 2 hours/day, 2 days/week, 35 weeks/year on a recreational site, a value of 5.8 days/year is presented in the terms of reference (2 hrs/day x 2 days/week x 35 weeks/year divided by 24 hours to produce the number of 24 hour days per year). This value will be used to predict exposure to receptors while spending time on recreational properties.	Revision made
33		4.2.4 – Identification of Exposure Pathways (page 29)	<u>Inhalation exposure pathways:</u> Mention is made that direct inhalation of COPCs in indoor air will be predicted, but it is not clear how this will be predicted.	Please refer to the response to comment #15.	No change required
34		4.3.4 – Exposure Estimation Methods (page 35)	<u>Winter snow coverage:</u> In the winter the exposure to soil will be greatly reduced which will impact the exposure via incidental ingestion of soil, but no mention is made of the possibility of measuring metals in the snow in Creighton/Flin Flon, which may be an ingestion exposure pathway that could be considered.	Based on concerns raised by members of the TAC regarding exposure to COCs via the ingestion of snow, it is anticipated that a sampling program will be completed in the month of February in which concentrations of COCs in snow will be measured. It should be noted that this pathway is not typically assessed in RAs and as a result, there is significant uncertainty associated with the selection of exposure factors required to assess this pathway such as the amount of snow ingested on a daily basis.	No change required

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35		4.3.4 – Exposure Estimation Methods (page 36)	<u>Winter accessibility factor:</u> In the calculation for soil intake rate in the winter a winter accessibility factor (WA) is used, but it is not clear what this factor is or how it is obtained.	In the calculation of exposure to COCs via incidental ingestion of outdoor soil and indoor dust, based on U.S. EPA guidance, it is assumed that 45% of the recommended daily intake is attributed to ingestion of outdoor soil and 55% to the ingestion of indoor dust. For example, the ingestion rate for a toddler is 80 mg/day; therefore, 44 mg/day is assumed to be indoor dust and 36 mg/day is assumed to be outdoor soil. The ingestion rate for indoor dust will remain the same for entire year. The ingestion rate for outdoor soil will be 36 mg/day for the summer months, however, during the winter months it is assumed that there will be limited exposure to outdoor soil. Therefore, a winter accessibility factor of 10% is applied to reduce the ingestion rate for outdoor soil only. As a result, during the winter months, the toddler will ingest 44 mg/day of indoor dust and 3.6 mg/day of outdoor soil. The winter accessibility factor was previously assumed for a community-based risk assessment for a northern Ontario community. This value was reviewed and determined to be acceptable by an independent expert peer review panel (TERA – Toxicology Excellence in Risk Assessment).	No change required
36		4.3.4 – Exposure Estimation Methods (page 37)	<u>FR<sub>prorated</sub> equation:</u> the paragraph directly following the FR <sub>prorated</sub> equation has been truncated by the equation.	This will be corrected in the revised ToR.	Revision made
37		4.3.4 – Exposure Estimation Methods (page 42)	<u>Drinking water ingestion:</u> Typical Flin Flon and Creighton drinking water concentrations are to be used in all scenarios, but according to the literature review and data gap analysis (p. 13), only one water sample from a residential home in Creighton and one water sample from a residential home in Flin Flon will be obtained on a weekly basis. It may be prudent to collect water samples from additional residential homes in both communities to help ensure appropriate drinking water concentrations as there may be confounding factors present at the current sampling homes that may not be readily apparent.	This issue has been discussed with HBMS and members of the TAC and it is anticipated that additional samples will be collected. It has been proposed to complete a large one day sampling event in which water samples are collected from multiple homes in Flin Flon and Creighton, as well as the distribution centres. The ToR will be revised to reflect this once this has been confirmed.	Revision made
38		4.4.3 – Speciation (page 48)	<u>Urinary arsenic studies:</u> It is mentioned that urinary arsenic studies may be beyond the scope of the current project. We feel it is important to determine what the subsequent steps may be for different outcomes of this HHRA to understand what direction HBMS will be headed based on different scenarios. We should discuss this at the meeting. It may be too early to determine the scope of the study depending on what the HHRA results are showing.	This is a topic that will likely warrant further discussion amongst members of the TAC as the study progresses. The completion of a urinary arsenic study should be considered following analysis of the results of the residential soil and dust sampling program and/or following the review of the initial results of the HHRA.	No change required
39		4.5.1 – Calculation of Risk Levels (page 48)	<u>Hazard Quotient calculations:</u> To what ratio will the hazard quotient be compared to determine if a risk exists? Will it be 0.2 or 1.0 or another ratio, and what is the justification for the selected comparison ratio? Likely best to determine this a priority.	Given that the HHRA will include a multi-media exposure assessment, it is anticipated that an acceptable hazard quotient (HQ) will be 1.0. However, for those chemicals where an additional medium (e.g., consumer products) may act as a relevant source of exposure that was not assessed in the HHRA, an acceptable HQ of <1.0 may be used.	No change required
40		Appendix C (page C-1)	<u>Estimation of indoor dust concentrations of COPCs:</u> It is not clear what determining the relationship between metal concentrations in soil and dust will achieve in terms of this HHRA. Will the relationship be used in calculations within the HHRA, or is it more to understand the impact of industry on dust COPCs in the home?	Establishing the relationship between metal concentrations in outdoor soil and indoor dust will allow risk assessors to consider exposure and risks on a property-specific basis for those properties where only one media was sampled (i.e., outdoor soil or indoor dust). For example, since outdoor soil concentrations are/will be available for significantly more properties than indoor dust concentrations will be, the indoor dust concentration for a particular property can be calculated based on the measured outdoor soil concentration and the relationship between these two media established at sites where both were measured. This will also be useful for risk management decisions which may involve an assessment on a property-by-property basis.	No change required

Comments Received on the HHRA Terms of Reference (ToR)					
Comment #	Reviewer	Page/Section	Issue/Comment	Intrinsic Response	Change Made in Report
41	Manitoba Health	4.1 – Human Health Risk Assessment Framework (page 17)	What is the purpose of developing property-specific soil standards? How is the “acceptable level of risk” defined, or will the TAC be doing that?	<p>Property-specific soil standards are soil concentrations that have been back-calculated using all of the HHRA exposure parameters and assumptions that are protective of receptors under specific property uses. For example, a property-specific soil standard will be derived for each COC that represents the maximum concentration that can occur on a residential property without the occurrence of an unacceptable risk. This is valuable to risk assessors and risk managers because the HHRA will typically predict risks on a community or population level using an exposure point concentration. However, this will not directly consider risks to receptors that will be chronically exposed to concentrations in excess of the exposure point concentration (e.g., residential receptors exposed to elevated concentrations in their backyard). Therefore, by providing PSSSs, a risk manager can determine which properties contain concentrations above the PSSSs which may indicate that risk management may be required.</p> <p>The acceptable level of risk is based on regulatory policy. Given that the HHRA will address risks for an area spanning the Manitoba-Saskatchewan border, the assessment will follow Federal policies provided by Health Canada and the CCME. Intrinsic has requested confirmation from Health Canada and the Provinces on this issue, but anticipates that the acceptable ILCR will be <math>1.0 \times 10^{-5}</math>. For non-carcinogenic compounds, the HHRA will include a multi-media assessment, therefore, an acceptable HQ will be 1.0 provided that there are no relevant additional sources of exposure. For those chemicals where consumer products or other media not included in the assessment may significantly contribute to exposure, an acceptable HQ of &lt;1.0 may be adopted.</p>	<p>No change required</p> <p>No change required</p>
42		4.3.3 – Deterministic versus Probabilistic Exposure Analysis (page 33)	The proposed approach (using a deterministic analysis) and then considering a probabilistic analysis if elevated risks are found doesn't provide an estimation of the likelihood of under-rating the risk. What is the rationale and is this a standard approach? I have some concern about this given the number of assumptions that will go into building the models to estimate the risk.	Currently there are no plans to perform a probabilistic assessment; however, following the completion of the deterministic HHRA this issue will be re-addressed	No change required
43		4.3.4.1 – Outdoor Soil/Indoor Dust Exposure (page 36)	Will equation elements (such as the period of time considered summer/winter, and the winter accessibility factor) be derived with the participation of the TAC? Are they site-specific?	Exposure parameters will be selected by Intrinsic from RA procedures documents provided by recognized agencies such as Health Canada, the CCME, and the U.S. EPA, or peer reviewed scientific literature. When available, reliable site-specific information will be used in place of generic values. When neither site-specific information or literature-based information is available, professional judgment may be used to derive values. The TAC will have the opportunity to review these values and provide comments/suggestions for the selection of alternate values.	No change required
44		4.3.4 – Exposure Estimation Methods (page 35-44)	Although these are labeled as exposure calculations, they seem to represent attempts to estimate doses from the various exposure pathways.	In keeping with the general terminology used in risk assessment, the calculations described in this section are part of the step described as the exposure assessment which estimates the chemical-specific dose resulting from exposure to impacted environmental media.	No change required
45		Page 48	Is the exposure ratio more accurately a dose ratio? For threshold response chemicals my understanding is that the “threshold level” is a level of 0 risk. If we have a ratio 5 times that, what does this mean in terms of quantifiable risk, or is the best we can say from this that there is an elevated risk of adverse health impacts?	<p>Yes, the exposure ratio, or hazard quotient, is the ratio between the predicted dose and the reference dose (RfD).</p> <p>An exposure ratio, or hazard quotient, of 1.0 or less indicates that the predicted dose is not sufficient to result in the occurrence of the toxicological endpoint upon which the RfD is based. The relative exceedance over the value of 1.0 provides little information on the magnitude of risk; although the likelihood that an adverse effects will occur increases as the ratio increases. It is best to consider an HQ &gt; 1.0 as indicative of the potential for an elevated risk of adverse health impacts, not the occurrence of health effects.</p>	<p>No change required</p> <p>No change required</p>

Comments Received on the HHRA Terms of Reference (ToR)					
<b>Comment #</b>	<b>Reviewer</b>	<b>Page/Section</b>	<b>Issue/Comment</b>	<b>Intrinsic Response</b>	<b>Change Made in Report</b>
46		Page 49	Is a single incremental lifetime cancer risk calculated for all types of cancer an element might cause or is it done for each type of cancer (for example arsenic has been associated with higher risk of skin, bladder and lung cancer)? How is the cancer slope factor derived, and again is there a unique factor for each cancer/each element?	<p>For carcinogenic COCs that have been associated with multiple forms of cancer, an ILCR is typically calculated for the occurrence of the most sensitive form of cancer.</p> <p>The cancer slope factor is derived using statistical models and generally will consider each cancer endpoint independently, generating factors for each endpoint. The selected slope factor is the most conservative value calculated. This selection process is done by regulatory agencies such as U.S. EPA and Health Canada.</p>	No change required

Field Sheet - Metals in Soil, Flin Flon Sampling Program								
Sample ID	UTM Coordinates	House Address	Sample Depths (cm)				Comments (Condition of Yard, Sod Condition, Soil Texture)	Interest in Dust Sampling? (Name,Phone#)
			Front Lawn	Back Lawn	Sand box	Garden		
Ff363	316198 6074193	183 Dominion	0-2.5 2.5-5.0 5.0-10 10-15 25-30	0-2.5 2.5-5.0 5.0-10 10-15 25-30	0-2.5 2.5-5.0 5.0-10 10-15 25-30	0-2.5 2.5-5.0 5.0-10 10-15 25-30	1 <sup>st</sup> home on street, 1953, original lawn, Back 3% slope, 1/2lawn, 1/2 garden. 0-2.5 thatch, 2.5-4 organic, 4-20 clay min. Mature spruce tree in garden, light gray soil colour in garden, little compost.	Jane Doe Xxx-xxxx

**Sample Field Sheet Completed for Each Location Selected Within the Supplemental Soil Sampling Program**

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Response to Comments on the Draft Problem Formulation

**A Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan**

Comments from Manitoba Conservation

**Comment:** For the most part, the approach and exposure scenarios proposed for this Human Health Risk Assessment (HHRA) follows standard methodologies used by other agencies (e.g., U.S. EPA) and we believe is acceptable for this assessment. One suggestion:

1. **6.0 Exposure Scenarios:** All of the exposure scenarios assume that each sub-groups (e.g., children, office workers, etc.) are inside for most of the day. For all of the receptors, the annual average time spent outdoors (parameter TSO) is 90 minutes/day. This assumption ignores the subgroup of workers who work predominantly outside (e.g., city work crews, landscapers, garbage collectors, etc.). While the occupational exposure of workers at the HBMS smelter is excluded from this HHRA, the exposure of these outdoor workers might still be considered. Is there merit to develop an exposure scenario that addresses this additional outside exposure?

**Response:** An outdoor worker scenario will be added to the Problem Formulation to be assessed in the HHRA. This scenario will evaluate exposure and risks to workers that spend 100% of their workday outdoors, exposed to COCs in soil *via* incidental ingestion and dermal contact, as well as through the inhalation of outdoor air.

Comments from Health Canada

**Comment:** Section 2.2, p.4: when referring to the use of air monitoring data to predict exposure point concentrations, is this implying formal modeling will be used to obtain those predictions?

**Response:** No, it is not anticipated that formal modeling will be used to derive exposure point concentrations for COCs in ambient outdoor air. Data collected from monitoring stations will be used to assess acute and chronic inhalation risks. For the chronic exposure scenarios, the annual average air concentrations will be used. For the acute exposure scenario, the maximum 24-hour concentrations will be used. Data collected from the monitoring station on Ruth Betts School will be used to assess exposure to residents in East Flin Flon and Channing. Data collected from the Creighton School will be used to assess exposure to residents in Creighton; and data collected from the Provincial building will be used to assess exposure to residents of West Flin Flon.

**Comment:** Section 3.2, p. 8: sulphur will not be addressed directly as a COPC, but will the influence of sulphur on metal solubility/bioavailability to edible plants/people be discussed (perhaps within the bioavailability study)?

**Response:** The influence of sulphur on metal solubility/bioavailability will be discussed in detail within the HHRA. This will include a discussion of the uncertainties associated with the predicted exposure and risk levels.

**Comment:** Table 3-3: criteria for screening soil concentrations were based upon residential/parkland scenario, although data were gathered from backyard gardens (some of which were used for growing food). This may cause some confusion for the public -- given that in some cases (e.g., lead) the agricultural guideline is lower than the residential. Does the use of the residential vs. the agricultural guideline affect the selection of the COPCs -- and could the

issue of the appropriateness of the residential vs. the agricultural guideline be addressed in the report?

**Response:** For a number of chemicals, the screening criterion used to select the COCs for the HHRA is the human health component of the CCME residential soil quality guideline. While it is recognized that the final agricultural guideline may be lower than the final residential guideline for a number of chemicals, only cadmium has a human health component value for agricultural land use (1.4 µg/g) that is lower than the human health component value for residential land use (14 µg/g). For the remaining chemicals, including lead, the difference between the two final guidelines can be attributed to ecological effects. In the case of lead, although the overall guideline is lower for the agricultural land use (70 µg/g) than for residential land use (140 µg/g), this difference is due to a lower environmental health component value. The human health component value for both land uses is 140 µg/g.

Since cadmium has already been retained as a COC for the HHRA, use of the human health component values for agricultural land use over the human health component values for residential land use does not impact the overall selection of COCs for the HHRA.

**Comment:** Section 3-4: Where were the exceedances for Zn, Cr, Th, Mn and Fe located? Is there any other possible reason for these elevated levels other than the assumption of naturally-occurring anomalies? And were the areas sampled in the USGS survey similar geologically to the Flin Flon area?

**Response:** Zinc was found above the health-based screening criterion (16,000 µg/g) in 2 of 652 samples (21,200 µg/g at FF223F in West Flin Flon, and 16,500 µg/g at TQ0508 in West Flin Flon). \*Please note that the Problem Formulation originally indicated that 3 samples contained zinc at concentrations in excess of the screening criterion. A transcription error was identified therefore the revised Problem Formulation now indicates that only 2 samples were in excess of the criterion.

Chromium was found above the health-based screening criterion (220 µg/g) in 2 of 652 samples (245 µg/g at TQ0586 in Channing, and 230 µg/g at TQ0578 in East Flin Flon) \*Please note that the Problem Formulation originally indicated that 3 samples contained chromium at concentrations in excess of the screening criterion. A transcription error was identified therefore the revised Problem Formulation now indicates that only 2 samples were in excess of the criterion.

Thallium was found above the health-based screening criterion (1 µg/g) in 3 of 652 samples (1.51 µg/g at FF301F in East Flin Flon, 2.7 µg/g at TQ0539 in East Flin Flon, and 1.1 µg/g TQ0505 in West Flin Flon).

Manganese was found above the health-based screening criterion (320 µg/g) in 51 of 318 samples. These exceedances were found in East and West Flin Flon, Creighton, and Channing.

Iron was found above the health-based screening criterion (11,000 µg/g) in 200 of 652 samples. These exceedances were found in East and West Flin Flon, Creighton, and Channing.

Given that only 2 samples exceeded the criteria for zinc and chromium, and 3 samples exceeded the criterion for thallium, it is anticipated that elevated concentrations are either the result of naturally occurring anomalies, isolated anthropogenic influences, or laboratory uncertainty. Since a larger number of samples exceeded the criteria for manganese and iron, it

is anticipated that these concentrations are the result of the natural geology of the area. This is supported by the spearman correlation completed by Manitoba Conservation (2007) which indicated that neither manganese or iron in soil were correlated with any of the known or suspected elements of smelter emissions (*i.e.*, antimony, arsenic, cadmium, copper, lead, mercury, molybdenum, selenium, silver, sulphur, thallium, and zinc). This suggests that levels of manganese and iron in soils are not related to atmospheric emissions from the smelter.

The areas sampled from Manitoba in the USGS survey spanned from Southern Manitoba (latitude 49°, longitude -97°) (*e.g.*, Winnipeg) to Northern Manitoba (latitude 58°, longitude -94°) (*e.g.*, Churchill). There is likely significant variability in the geology spanning this distance. Flin Flon is located at latitude 54° and longitude -101°. The average concentrations of manganese and iron were both below the average concentrations for all of Manitoba reported in the USGS study. In addition, concentrations of these chemicals were similar to those measured in Baker's Narrows and Cranberry Portage which are anticipated to have similar geology to the Flin Flon-Creighton area.

**Comment:** Section 6-0: The residential scenario could also include time spent in public areas (like playgrounds) for the toddler -- which I think would be a fairly realistic concern for the public.

**Response:** The HHRA will include an assessment of exposure and risk for residential and commercial land. Since Health Canada (2006) recommends that the residential exposure scenario assumes that receptors are on-site for 24 hours per day, 365 days per year, there is no time left to be allotted to time spent in recreational areas. For recreational land, although Health Canada (2006) recommends an exposure frequency of 2 hours per day, 2 days per week, 35 weeks per year, this is not considered to be appropriate for community-based assessments when it is assumed that exposure to COCs will also occur during time spent away from the recreational land. In the derivation of soil standards based on property-use, to be consistent with the CCME, the back-calculated value protective of residential land use will also be applied to recreational/parkland properties.

**Comment:** I have concerns about the exclusion of occupational exposures -- for *e.g.*, theoretically both occupational and residential exposures could be below regulatory thresholds, but the combined total could exceed such limits. If the decision to exclude occupational exposures is carried forward, then this must be discussed as a significant area of uncertainty in the results of the HHRA.

**Response:** A commercial land use scenario will be included which will evaluate exposure and risk to outdoor workers exposed to impacted soil while at work. This will not include occupational exposure related to mining or smelting activities.

**Comment:** Finally, please be aware that Health Canada has updated the guidance for risk assessment of contaminated sites (these updates may not be publicly available, yet, please let me know if you don't have access) – in particular, adoption of the U.S. EPA age-specific adjustment of slope factors for cancer risk assessment may influence the results of this HHRA.

**Response:** This document has been reviewed and will be considered in the preparation of the HHRA.

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### Comments from Manitoba Health

**Comment:** On page 4 the last paragraph talks about the air quality data that will be used. Will there be a correction factor based on the distance of Channing vs. Eastern Flin Flon since the data for both comes from the Ruth Betts school?

**Response:** Due to a number of variables and uncertainties, it is not anticipated that an accurate correction factor could be derived to adjust air data collected from Ruth Betts to be reflective of site-specific conditions in Channing and Eastern Flin Flon. Therefore, the data from Ruth Betts will be used as is for both areas.

**Comment:** On page 7 the 3rd paragraph from the bottom mentions that the concentrations may create risks to human health. I wonder if for consistency that could either be rephrased or have an additional comment added to the effect that “preliminary review suggests that the risk to human health is most likely low.” This phrase is from page iv of the MB Conservation report, and has been reinforced to the CAC, TAC and media.

**Response:** The suggested comment has been added to the revised Problem Formulation to be consistent with the Manitoba Conservation report.

**Comment:** The incremental lifetime cancer risk is identified in the report. I assume that these will be malignancy specific (e.g., lung cancer, bladder cancer, etc.) when compared to the Flin Flon/ Creighton population. What population data will be used as the baseline for comparison (e.g., Canada average, MB average)? Is the lung cancer risk going to be calculated as a sum risk from the multiple exposures (e.g., risk from arsenic and risk from Cadmium)? Is this risk going to be calculated separately for the 4 communities identified for analysis?

**Response:** The incremental lifetime cancer risks (ILCRs) will be endpoint specific and will be compared to the Health Canada acceptable ILCR of  $1.0 \times 10^{-5}$ . If there is evidence to suggest that COCs have similar toxicological mechanisms and elicit similar carcinogenic endpoints, the ILCRs will be summed. ILCRs will be calculated separately for each of the four communities described in the Problem Formulation.

**Comment:** Can you clarify if how the dose to the receptors is to be calculated is normally part of the problem formulation? I'm referring to the modeling parameters that have previously been presented as well as the bioaccessibility testing.

**Response:** The problem formulation typically includes a description of the exposure scenarios, exposure pathways, and receptor parameters. Specific equations used to calculate dose is not necessarily included within the Problem Formulation, however, for the current HHRA, these equations were provided in the Terms of Reference to allow for feedback from members of the TAC.

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Response to Government Comments Regarding Bioaccessibility	
Comment	Intrinsic Response to Comment
<p>1) <i>Bioaccessibility methodology:</i>                      a. <i>Elliot has provided some evidence regarding the validity of the bioaccessibility testing especially for arsenic, lead and nickel. Can there be more complete documentation / summarization of the validity of using and justifying the use of bioaccessibility testing for the other COPCs?</i></p>	<p>While some researchers and some regulators (U.S. EPA) consider bioaccessibility studies to only be validated for Arsenic and Lead, the consensus among others is that it is acceptable to use for other elements. For example, bioaccessibility has been accepted and used in Ontario for nickel, copper and cadmium, although there is a lack of regulatory guidance on this subject. The following are a few studies that address bioaccessibility with other elements:</p> <ul style="list-style-type: none"> <li>• <i>+Bioavailability of Hg (review, and making the point that &lt;100% is available):</i> Rosalind A. Schoof and Jesper Bo Nielsen, 1997. Evaluation of Methods for Assessing the Oral Bioavailability of Inorganic Mercury in Soil, Risk Analysis, 17 (5), 545-555.</li> <li>• <i>Bioavailability and bioaccessibility of Hg:</i> Dennis J. Paustenbach, Gretchen M. Bruce, and Paul Chrostowski, 1997. Current Views on the Oral Bioavailability of Inorganic Mercury in Soil: Implications for Health Risk Assessments, Risk Analysis, 17 (5), 533-544.</li> <li>• <i>Bioaccessibility of various elements in dust, with lots on ratios and particle size.</i> Pat E. Rasmussen, Suzanne Beauchemin, Michelle Nugent, Rose Dugandzic, Monique Lanouette, and Marc Chenier, 2008. Influence of Matrix Composition on the Bioaccessibility of Copper, Zinc, and Nickel in Urban Residential Dust and Soil, Human and Ecological Risk Assessment, 14: 351–371.</li> </ul> <p>Scientifically, we believe that there is no basis to reject its use for non-validated elements, since there is no scientific reason that these elements will behave differently from arsenic and lead. That said, this is ultimately a policy based decision that will have to satisfy regulatory concerns.</p>
<p>b. <i>Not all the recommendations were followed that were contained in Lindsay’s original comments on bioaccessibility and in the reference cited in the recent document by ASU and ESG (Health Canada 2007. Federal contaminated site risk assessment in Canada, Part V: guidance on complex site specific human health risk assessment of chemicals.) Can you provide further comment / clarity on why other dilutions were not used in those situations were detection limits were not exceeded and why there weren’t more combinations of various particle sizes &lt; 45 µ and &lt; 250 µ used (e.g., &lt; 45 µ with 2,000:1 dilution)?</i></p>	<p>Consideration of all combination and permutation of solid to fluid ratio particle size was not feasible. A pilot study was designed up to examine some of the combinations suggested. Two soils were used that had high concentrations of elements, but one was used with lower concentrations, in an effort to test a soil from a different location in Flin Flon (<i>i.e.</i>, to obtain 3 soils that were representative of all the soils). Using a high estimate of bioaccessibility (from all soil values averaged together), the ratios 250:1 and 500:1 could have been used to obtain detectable values for the two soils FF208B and FF276F (the higher concentration soils). The ratio 1000:1 would have had estimated detectable values only for soil FF208B. The third soil, CS102B would have not had any detectable values (or very few) at the ratios higher than 100:1. Therefore it would have been difficult to accurately (and statistically) determine the effect of ratio on this representative set of samples. However, note that Health Canada (2007) also suggests addressing soil to solution issues (<i>i.e.</i>, avoiding the measurement of saturation rather than bioaccessibility) as follows: “If bioaccessibility is independent of solubility limitations, then a) the curve associating bioaccessibility with soil-borne concentration should not be negative; and b) the curve associating the</p>

Response to Government Comments Regarding Bioaccessibility	
Comment	Intrinsik Response to Comment
	mass of extracted substance and soil-borne concentration should have a statistically significant positive slope. Both of these relationships will indicate that the proportion of the substance extracted from the soil is independent of soil-borne concentration and is, thereby, independent of assay design." For a), the % bioaccessibility vs. total concentration curves are for the most part scattered (around a horizontal line), except for Hg in Phase 1. For b), all bioaccessible concentrations vs. total concentrations (log transformed since the data sets are log distributed) are correlated (most r values >0.9). With respect to the question about conducting more ratio tests on the <45 um fraction (presumably following a factorial design), two reasons account for only doing one set of experiments with this fraction: the soil sample sizes were insufficient to obtain enough mass of the smaller fraction (one of our criteria for choosing soils for this experiment was high soil masses), and the detection limit concerns expressed above.
c. In the documentation (Table A-1) it is useful to see the denominator for each of the calculations for the % bioaccessibility calculation (i.e., Concentration in the various solids) rather than having to refer elsewhere.	Noted; however the format for table A-1 will remain unchanged.
2) Use of the bioaccessibility results: a. Sensitivity analysis: It is not clearly identified in the Terms of Reference how sensitivity testing will occur for those metals/metalloids for which bioaccessibility testing has been validated: Will mean and 95 percentile outlimit bioaccessibility values be used as well as 100% in the determination of exposure?	95UCLM values will be calculated for each phase and each element (the 95 UCLM represents an upper bound estimate of the mean value). The 95UCLM for the gastric phase will be utilized in all calculations (if 95UCLM values > 100% than 100% will be used for the calculation purposes). Intestinal phase results will be considered in the discussion of parameter uncertainties.
b. For the determination of Relative Bioaccessibility, there is some documentation of the methodologies to determine the relative bioaccessibility of arsenic (using arsenic in water for the determination of RfD), nickel (using food and even human water consumption testing), and lead (Drexler / Brattin) but there is little mention on the methods to calculate Relative Bioavailability for the other COPC	Similar methods will be considered for other elements. This will involve a review of the studies utilized in the derivation of TRVs

Please note that the following update (provided by Queen's/RMC) was provided to the TAC in mid-July and no comments were received. In addition, I offered to set up a conference call to discuss these preliminary results so that the main study could proceed and I was informed that further discussion was not necessary. As a result, the study proceeded as outlined below.

We have completed the first part of the study that compares the three methods for three soils. The three methods are: (1) <45 um, 100:1; (2) <250 um, 100:1 (3) <250, 2,000:1. Two phases (gastric and intestinal) were done for each of them.

We are still working on the stats and summarizing everything in a report but here is what we find:

1. Hg bioaccessibility is always higher in Phase 2 (gastric+intestinal). All other elements are either similar (As, some Cu) or lower in Phase 2. Se bioaccessibility was mostly non-detectable. We think the Hg higher bioaccessibility is because of increased

- organics (bile extract and pancreatin) in Phase 2. The take-away message is that we need to do both phases for this combination of elements.
2. The order of bioaccessibility (both % and mg/kg) is: Method 3 is generally higher than method 1, which is higher than Method 2.
  3. Following from observation 2 above, we cannot say that these differences are statistically significant at this point because we have only done ANOVA, and there are not enough detectable numbers for method 3 to make the stats reliable. We will be doing t-tests between method 1 and 2 today.
  4. Following from observation 3 above, all results for methods 1 and 2 were detectable for Hg, As, Cd, Cu, Pb and Zn, but not for Se. For method 3, the following percent of the results were non-detectable: 67% for Hg, 50% for As and Cd, 100% for Se, and 17% for Zn. All Cu and Pb results were detectable for method 3.

Based on these results, and assuming we want to have as many detectable results as possible, we recommend method 1: <45 um, 100:1 for the most conservative results, but with the highest detection rate. Even though method 3 gives generally higher numbers, we feel that the detection is compromised. We will also continue doing 2 phases and providing both numbers.

This study was done with FF208B, CS102B and FF276F. We'll be getting ASU to send a report shortly.

M. Yole Additional comments, (addendum to comments by Lindsay Smith):

### **Comments on Bioaccessibility Testing Protocol For Soil at Flin Flon Manitoba - Final Draft for TAC**

**Prepared by Margaret Yole, Safe Environments Programme, Health Canada  
Feb 14, 2008**

While this version of the document is noted to have incorporated the comments provided previously by Lindsay Smith (Health Canada), additional comments are provided below:

#### **1. Lack of Bioavailability Data for TRVs for the COPC's at Flin Flon:**

The current Health Canada TRVs for arsenic, lead, cadmium, copper and zinc are based upon studies for which no bioavailability information is available.

It will therefore likely be difficult to make meaningful assessments of relative absorption for most metals found in Flin Flon soils based on the bioaccessibility data obtained from this report.

TRV's with bioavailability data could be obtained from other major regulatory agencies, but may prove difficult to find. The Health Canada TRVs for cadmium and lead are based upon WHO values; most major regulatory authorities have based their arsenic TRVs on the same studies as those used by Health Canada; and the zinc and copper TRVs are based upon values provided the Institute of Medicine (see summary information regarding the derivations of each of these TRVs attached separately – **not for general circulation**).

## 2. Discussion of Uncertainty

The discussion of uncertainty is very limited – and refers mainly to differences in bioaccessibility estimates between various experimental protocols. It is noted that no model is ideal, but no data regarding the performance of the model to be used are provided, *i.e.*, how does it compare to *in vivo* bioavailability measurements?

The more pertinent areas of uncertainty to discuss relate to potential differences between experimental bioaccessibility values and real-world bioavailability (*i.e.*, limitations on the usefulness of data obtained).

In addition to the factors identified by Lindsay Smith, and the presence of food in the GI tract (as noted in the document), there are multiple additional factors that influence metals uptake *in vivo*, including (though not limited to):

- Effects of microbial activity *in vivo* (the model used will simulate only the gastric and small intestinal environments – the lower intestine is not an insignificant player in absorption from the GI, for *e.g.*, <http://pubs.acs.org/cgi-bin/abstract.cgi/esthag/2007/41/i15/abs/es062410e.html> ),
- Effects of dynamic equilibrium *in vivo* (with more or less continuous removal of solubilized metals from the ingesta at the intestinal brush border, as compared to the static equilibrium *in vitro*);
  - Influence of metals transport at the intestinal brush border (passive diffusion, transporter/carrier proteins, co-transport, formation of absorbable complexes with proteins/amino acids, membrane binding and internalization, competitive and non-competitive interactions between the various metals, *etc.*);
  - Influence of diet and nutritional status (especially calcium) on rates up uptake of metals from the intestine;
  - effects of pH on ionization and lipid solubility;
  - Biliary excretion of heavy metals (including arsenic, lead, mercury, cadmium and zinc) and influence of enterohepatic recirculation; and,
  - Age and development of GI physiology.

## 3. Effects of Soil Type

There is no discussion of the how effects of soil type will be handled in the interpretation of the results, or whether only certain samples will be included in the analysis – sandboxes, playgrounds (possible including sandy areas), garden soils (usually high organic matter) and street area soils (usually less organic matter), *etc.*, were all sampled in the Flin Flon area.

## 4. Effects of Initial Metal Concentration in Soil on Equilibrium Conditions *in vitro*

There is no discussion of the how the potential differences in bioaccessibility at different bulk metal concentrations will be handled in the interpretation of the results.  
(see again <http://pubs.acs.org/cgi-bin/abstract.cgi/esthag/2007/41/i15/abs/es062410e.html> )

## 5. The Names of the Members of the SBRC are Not Relevant to this Document.

## 6. A Subset of Soil Samples will be Sieved to Less Than 250 $\mu\text{m}$ , But it is Not Noted to What Size.

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Response to Manitoba Conservation Comments on the Draft Toxicological Profiles	
Comment	Intrinsic Response to Comment
<p>All of the toxicological reference values (TRVs) chosen for the Human Health Risk Assessment (HHRA) were selected from existing criteria established by different regulatory agencies (e.g., U.S. EPA, Health Canada, etc.). While this approach does mean that the TRVs have undergone thorough development and review, it also means that a key exposure pathway may be ignored if a criterion is not available (e.g., cadmium does not have a carcinogenic-based criterion for oral exposures).</p> <p>How does Intrinsic intend to include those metals in the HHRA for which there is a reasonable exposure pathway but no available criteria?</p>	<p>Please see responses pertaining to particular chemicals. However, all of the COCs retained have available criteria for all relevant pathways that will be used to assess risk for the exposure pathways of interest. For example, cadmium does not have a carcinogenic-based criterion for oral exposures since regulatory agencies have not developed quantitative estimates of carcinogenic risk following oral exposure. The reason for this is a lack of evidence of carcinogenic effects following oral exposure. The U.S. EPA IRIS database indicates the following: <i>Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.</i></p>
<b>Arsenic (Table A1-1)</b>	
The use of existing criteria leads to an inconsistency in the arsenic criteria in that the acute 1-hour inhalation exposure limit ( $0.19 \mu\text{g}/\text{m}^3$ ) is less than the 24-hour inhalation exposure limit ( $0.3 \mu\text{g}/\text{m}^3$ ).	It should be noted that air quality data is only available on a 24-hour basis and as a result the 1-hour criteria was not utilized in the risk assessment.
There is no short-term acute exposure limit for oral exposures to arsenic, presumably because of the use of existing criteria. How will this exposure pathway be assessed?	Agreed, an appropriate short-term TRV for oral arsenic exposure will be included in the final draft.
<b>Arsenic (Page A1-6)</b>	
The reason given for accepting both the U.S. EPA oral and inhalation criteria for carcinogenicity was that each was "the most widely accepted and technically defensible toxicity reference value available for arsenic". While this statement is probably correct, some justification for these opinions (i.e., "most widely accepted" and "most technically defensible") should be provided.	All TRVs have been reviewed by a qualified toxicologist and it has been concluded that the U.S. EPA values are the most defensible. It should be noted that the U.S. EPA and Health Canada unit risks and slope factors are similar. The U.S. EPA unit risk is slightly more recent and includes consideration of additional studies beyond those considered by Health Canada.
The discussion states that the U.S. EPA inhalation unit risk factor of $4.3(10^{-3}) (\mu\text{g}/\text{m}^3)^{-1}$ should not be used if the air concentration exceeds $2 \mu\text{g}/\text{m}^3$ . A note to that effect should be included in Table A1-1.	Agreed; a footnote will be included in the table to indicate this.
<b>Cadmium (Table A4-1)</b>	
There are no criteria provided for oral exposures to cadmium for acute risk or chronic –carcinogenic risk. Since cadmium is a known carcinogen from an inhalation perspective, cadmium could also be an oral carcinogen. How will Intrinsic incorporate the risk from cadmium for an oral exposure pathway into the HHRA?	Evidence for the carcinogenic risk of cadmium <i>via</i> oral exposure through consumption of cadmium present in the soil is insufficient as the studies have not provided the necessary data. Therefore, both the U.S. EPA IRIS and RIVM do not have slope factors for the oral route and thus it will not be assessed in the HHRA. The reason for this is a lack of evidence of carcinogenic effects following oral exposure. The U.S. EPA IRIS database indicates the following: <i>Seven studies in rats and mice wherein cadmium salts (acetate, sulfate, chloride) were administered orally have shown no evidence of carcinogenic response.</i>
There is an inconsistency between the value for the chronic, non-cancer inhalation exposure limit listed in Table A4-1 and Table A4-3. In Table A4-1, the value is given as $0.025 \mu\text{g}/\text{m}^3$ , while in Table A4-3 the value is $0.005 \mu\text{g}/\text{m}^3$ . The value in Table A4-3 is also consistent with the accompanying text on the TRVs.	Agreed. The correct value should be $0.005 \mu\text{g}/\text{m}^3$ for the chronic, non-cancer inhalation exposure limit. The correction will be made in table A4-1.

<p><b>Copper (Table A3-1)</b></p>	
<p>There is no acute oral exposure limit for copper, even though one is presented in Table A3-3 (i.e., 10 µg/kg/day). Although this TRV is discussed in the text on page A3-5, no explanation is given as to why it was not chosen for use in the HHRA.</p>	<p>Agreed; an appropriate short term TRV will be selected in the final draft.</p>
<p><b>Lead</b></p>	
<p>As with the previous draft profiles, all of the toxicological reference values (TRVs) chosen for the Human Health Risk Assessment (HHRA) were selected from existing criteria established by different regulatory agencies (e.g., U.S. EPA, Health Canada, etc.). As noted previously, this approach does mean that the TRVs have undergone thorough development and review, but it also means that a key exposure pathway may be ignored if a criterion is not available (e.g., there is no acute oral criterion for lead). How does Intrinsic intend to include lead in the HHRA for exposure pathways which have no available criteria?</p>	<p>Agreed, however, no appropriate short term TRV was identified for lead.</p>
<p><b>A5-1.7 Toxicological Reference Doses (TRVs): Carcinogenic TRVs:</b> Both the U.S. EPA and IARC have classified inorganic lead as a probable human carcinogen, but none of the carcinogenic TRVs available from the California EPA were accepted. How does Intrinsic intend to account for lead's carcinogenicity in this risk assessment?</p>	<p>As indicated, several agencies have classified lead as a probably human carcinogen; however, U.S. EPA, in the IRIS database has indicated the following:  <i>Quantifying lead's cancer risk involves many uncertainties, some of which may be unique to lead. Age, health, nutritional state, body burden, and exposure duration influence the absorption, release, and excretion of lead. In addition, current knowledge of lead pharmacokinetics indicates that an estimate derived by standard procedures would not truly describe the potential risk. Thus, it is recommended that a numerical estimate not be used.</i>                  As a result, the HHRA will not attempt to quantify lead related cancer risk.</p>
<p><b>A5-1.7 Toxicological Reference Doses (TRVs): Non-Carcinogenic TRVs:</b> The Health Canada provisional total daily intake (pTDI) of 3.6 µg/kg-day was selected for this risk assessment. The consultant states that this pTDI will be used to evaluate both oral and inhalation exposure pathways. Since this pTDI is expressed in units more commonly used for oral exposures, how will the "inhalation exposure pathway" be calculated for comparison with this pTDI?</p>	<p>Systemic exposure will be considered in the HHRA, in that both oral and inhalation exposures will be expressed as a dose (µg/kg/day) and evaluated as such. The U.S. EPA IEUBK model, used for the evaluation of children lead exposures, does this conversion in-line. For other receptor groups, inhalation exposure (expressed as an air concentration) will be converted to an inhalation dose using the following equation:</p> $Dose \left( \frac{\mu g}{kg \cdot day} \right) = \frac{EXP \left( \frac{\mu g}{m^3} \right) \times Inh \ Rate \left( \frac{m^3}{day} \right)}{Body \ Weight \ (kg)}$
<p><b>Mercury</b></p>	
<p><b>Elemental Mercury (Table A6-1)</b></p>	
<p>The chronic, non-cancer exposure limit for oral exposures is given as 0.06 µg/m<sup>3</sup>. This TRV is actually for inhalation exposures, as indicated on Table A6-3, but the derivation of 0.06 µg/m<sup>3</sup> was not discussed in the text at all. On page A6-12, however, the text states that the U.S. EPA IRIS Reference Concentration (RfC) of 0.3 µg/m<sup>3</sup> was chosen for the assessment, but this RfC is not bolded in Table A6-3 (indicating it was selected), nor was it included in Table A6-1.</p>	<p>Agreed, the value of 0.06 µg/m<sup>3</sup> corresponds to the TRV for inhalation exposure as per Health Canada as there is no oral exposure limit for elemental mercury. This correction will be made in Table A6-1. Also, a brief summary will be included to indicate how the Health Canada value was derived.</p>

<b>Elemental Mercury (Page A6-10)</b>	
The statement is made that only the inhalation exposure pathway for elemental mercury would be assessed. A short statement should be provided giving a rationale for this decision.	RIVM has stated that absorption of metallic mercury in the body after oral consumption is quite minimal compared to inhaled metallic vapour whereby, 70 to 80% is absorbed into the body. Therefore, since absorption into the body is minimal after oral consumption, this pathway will not be assessed in the HHRA. Additionally, mercury is most common found in rocks and soil as mercuric sulphide (an inorganic form).
<b>Inorganic Mercury (Table A6-1)</b>	
As for arsenic, the use of existing criteria leads to the inconsistency that the 1 hour inhalation exposure limit of $1.8 \mu\text{g}/\text{m}^3$ was slightly less than the 24 hour exposure limit of $2.0 \mu\text{g}/\text{m}^3$ .	It should be noted that air quality data is only available on a 24 hour basis and as a result the 1 hour criteria was not utilized in the risk assessment.
The units for the chronic, non-cancer inhalation exposure limit should be " $\mu\text{g}/\text{m}^3$ " rather than " $\mu\text{g}/\text{kg}/\text{day}$ ".	Agreed, the correction will be made.
<b>Inorganic Mercury (Table A6-3)</b>	
A value for an acute, oral exposure limit of $7.0 \mu\text{g}/\text{kg}/\text{day}$ for inorganic mercury is provided in Table A6-3, but no explanation is given as to why an acute, oral exposure limit value was not chosen for the HHRA.	Agreed; an appropriate short term TRV will be selected in the final draft.
<b>Methyl Mercury (Page A6-12)</b>	
The statement is made that only the oral exposure pathway for methyl mercury would be assessed. A short statement should be provided giving a rationale for this decision.	Methyl mercury is primarily produced by microorganisms, which can convert inorganic mercury to an organic form. In the past, organic forms of mercury were used as fungicides, but these uses are presently banned in North America and Europe due to evidence of adverse human health effects. WHO has indicated that methyl mercury does not largely occur in the atmosphere; therefore, inhalation exposure will contribute minimally to the overall intake of methyl mercury. The inhalation pathway for methyl mercury is not commonly assessed in HHRA.
<b>Selenium (Table A2-1)</b>	
As has already been noted for several of the other metals, there is no acute oral exposure limit for selenium. Will the acute, oral exposure pathway be included in the HHRA for selenium?	Agreed, however, no appropriate short term TRV was identified for selenium.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
-	-	-	All revisions submitted in pdf form	All corresponding revisions completed
<b>Dave Green (Manitoba Water Stewardship) - submitted Feb.26/27/2009</b>				
General Comments	-	-	Upfront Notes: While I have done a general review for the majority of the document, my focus has been on the water quality and fish information. I have tried to avoid repeating comment on what has been already provided in the breadth of good comments from the other reviewers.	No revisions required.
General Comments	-	-	General comments: While some of the supplemental reports have maps showing site locations, a good map/s in the introduction or as an appendix showing the different areas and lake names discussed in the text would be very helpful. If maps were put into an appendix it should be noted in the introduction so readers know at the beginning where to find them	Additional maps have been added to the main report where appropriate.
1	Page 1-12	first bullet	It notes that Flin Flon's drinking water is Cliff Lake that is actively supplied water from Trout Lake. I take this means water is physically pumped from Trout Lake to Cliff Lake. If that is what is intended to be said then it would be clearer to say that than "actively supplied." This phrasing also occurs on page 2-9, Section 2.4.1 and page 4-6, section 4.1.1.4. As well, while people around Flin Flon call it Trout Lake, officially (on topographic maps) it is called Embury Lake. It would be good to put Embury Lake in parentheses after Trout Lake, or at least do this on a general reference map.	As suggested, clarification has been provided in Sections 1.5, 2.4.1, and 4.1.1.4.
2	Page 2-15	Section 2.5.3, top of page	It should be Manitoba Water Stewardship instead of Manitoba Conservation	Revision has been made as suggested.
2	Page 2-32	Section 2.10, first sentence	Suggest to change phrase in the parentheses to (see Appendix P for a copy of the survey and a map showing Areas 1 to 7).	Revision has been made as suggested.
2	Page 2-3	first sentence	change from (provided at the end of this document) to (see Appendix	Revision has been made as suggested.

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<b>Chapter</b>	<b>Page</b>	<b>Section</b>	<b>Comment</b>	<b>Response/Resolution</b>
3	Page 3.1.2	four bullets	These areas should be clearly delineated and labeled on a map and then the map referenced. A general rule is that features identified or referred to in the text should be also marked on maps.	East and West Flin Flon have now been clearly indicated on a revised Figure 3-1. A reference to this figure has been added to this section.
3	Page 3-3	Figure 3-1	quality is not that good and some text should be enlarged if resolution cannot be increased.	This Figure has been replaced with a similar Figure with greater resolution.
4	Page 4-10	Section 4.1.1.6, last sentence, last paragraph	It noted fish tissue concentrations were provided on a dry weight basis and converted to wet weight. Based upon this, subsequent calculations for results in Tables 4-8 and 4-9 and possibly others will need to be re-done. Fish tissue concentrations are normally provided on a wet weight basis. This has been corrected in the draft fish and berry report to reflect they were in wet weight.	The report and all exposure and risk calculations related to the consumption of fish have been adjusted to reflect that concentrations of COC in fish were provided as wet weight concentrations rather than dry weight concentrations.
4	Page 4-12	Table 10	The mercury concentrations should be $\mu\text{g/g}$ wet weight.	Revision has been made as suggested.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses																																																																			
Chapter	Page	Section	Comment	Response/Resolution																																																															
4	Page 4-12	Table 4-10	<p>The fraction of methylmercury in fish seems somewhat low from what I generally understood it to be. There is also a wide variation in the fractions from these nine samples. Unfortunately, I only have some Freshwater Institute (501 University Crescent, Winnipeg) data from Southern Indian Lake during 1979 and 1980. The organic mercury to total mercury ratios for this data were generally around 85 % as shown in the following table: Year Species Tissue Type n Sum Total Hg (mg/g) Sum MeHg (mg/g) Ratio MeHg:Total Ratio when an average is calculated using individual ratios</p> <table border="1"> <tbody> <tr> <td>1979</td> <td>N. Pike Liver</td> <td>99</td> <td>37.53</td> <td>32.56</td> <td>0.87</td> <td>0.86</td> </tr> <tr> <td></td> <td>Walleye</td> <td>59</td> <td>14.99</td> <td>13.38</td> <td>0.89</td> <td>0.89</td> </tr> <tr> <td></td> <td>Whitefish</td> <td>60</td> <td>19.95</td> <td>18.81</td> <td>0.94</td> <td>0.91</td> </tr> <tr> <td>1980</td> <td>N. Pike Muscle</td> <td>86</td> <td>57.12</td> <td>48.39</td> <td>0.85</td> <td>0.84</td> </tr> <tr> <td></td> <td>Walleye</td> <td>62</td> <td>35.53</td> <td>29.68</td> <td>0.84</td> <td>0.84</td> </tr> <tr> <td></td> <td>Whitefish</td> <td>84</td> <td>12.9</td> <td>10.79</td> <td>0.84</td> <td>0.84</td> </tr> <tr> <td></td> <td>N. Pike Liver</td> <td>84</td> <td>35.4</td> <td>29.9</td> <td>0.84</td> <td>0.84</td> </tr> <tr> <td></td> <td>Walleye</td> <td>58</td> <td>14.41</td> <td>12.38</td> <td>0.86</td> <td>0.90</td> </tr> <tr> <td></td> <td>Whitefish</td> <td>84</td> <td>20.43</td> <td>17.46</td> <td>0.85</td> <td>0.83</td> </tr> </tbody> </table> <p>My suggestion is that exposure should probably consider a scenario of 100 % as well to be conservative. The federal guideline of 0.5 mg/g for unrestricted sale of fish in retail outlets is based upon assessing total mercury concentrations in tissues that also gives a conservative approach.</p>	1979	N. Pike Liver	99	37.53	32.56	0.87	0.86		Walleye	59	14.99	13.38	0.89	0.89		Whitefish	60	19.95	18.81	0.94	0.91	1980	N. Pike Muscle	86	57.12	48.39	0.85	0.84		Walleye	62	35.53	29.68	0.84	0.84		Whitefish	84	12.9	10.79	0.84	0.84		N. Pike Liver	84	35.4	29.9	0.84	0.84		Walleye	58	14.41	12.38	0.86	0.90		Whitefish	84	20.43	17.46	0.85	0.83	<p>The report has been revised to use the 95% UCLM fraction of methyl mercury content in local fish (96%) as opposed to the average (79%) originally used.</p>
1979	N. Pike Liver	99	37.53	32.56	0.87	0.86																																																													
	Walleye	59	14.99	13.38	0.89	0.89																																																													
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8	Page 8-11	Page 8-11	<p>I don't understand what is being said in this paragraph and I think it needs re-working. Second, the 0.5 µg/g</p>	<p>This discussion has been revised to improve clarity and to indicate that the Health Canada guideline is for commercially</p>																																																															

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
			guideline is a total mercury wet weight value targeted for unrestricted commercial sale of fish in retail stores. It is preferable to not refer to it as “the” human consumption guideline value because it is not the upper definitive value the Federal Government or we use for consumption advisories.	sold fish. Additional references for sportfish consumption guidelines in Manitoba and Saskatchewan have been added.
Appendix C	page C-18 last paragraph and Table C-17 and C-18		A footnote should be included to note that the MSWQC were based upon the former Manitoba Surface Water Quality Objectives (1988) that have been replaced by the Manitoba Water Quality, Standards, Objectives, and Guidelines (2002).	A footnote has been added to Tables C-17 and C-18 as requested.
Appendix C	Page C-23		The Ross-Schist 1994 report summary and tables for water chemistry are repeated here from page C-18. Perhaps remove the water data information from pages C-18 and C-19 since this section deals with fish and sediment. They could then be left in this section on surface water data. However, if that is the case, the footnote suggestion in the previous bullet will need to go with Tables C-25 and C-26.	As suggested, Tables C-17 and C-18 have been removed from Section C-1.5 since the surface water data is more appropriately included in Section C-1.6. The suggested footnote has been included within these tables.
Appendix C	Page C-13, Table C-11		I think the arsenic value should be 5.7 µg/L instead of 57 µg/L based upon data I have that seems to match this information.	Revision has been made as suggested.
Appendix C	Page C-14, Table C-12		Similar to the previous comment, data I have that seems to match this information shows the 2004 copper concentration to be 15 µg/L instead of 48 µg/L and the zinc to be 100 µg/L instead of 100 µg/L. I will check with the Water Stewardship Office of Drinking Water to try and confirm this data.	Comment has been withdrawn by Dave Green. No revision required.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
<b>Lindsay Smith</b> (Health Canada, Safe Environments, MB/SK Region, for Manitoba Conservation)				
2	Page 2-5		<p>The tenant survey was not found in Appendix D with the rest of the report on the dust study. Please include a summary of the findings of this report, particularly in regards to the homes that had high levels of lead and/or other metals.</p> <p>It is not clear in future chapters of the risk assessment how the lead-in-paint data was incorporated into the study. Data on the percentage of homes with lead paint and the levels found in homes would be pertinent to both the risk assessment and future risk management decisions. Please provide this data with the rest of the information on the dust study in Appendix D.</p>	<p>A draft of the original Jacques Whitford Dust Study has been included in Appendix D of the revised report. This includes wipe sample results, lead in paint analysis and the tenant survey. At this stage, the lead in paint data has not been utilized in the HHRA; it is anticipated that this data will be useful risk management phases of the project.</p>
2	Page 2-6		<p>While it is true that PM10 is most relevant to the inhalation pathway, please note that people may still be exposed to total suspended particles. Particles larger than 10 microns are typically ingested rather than inhaled. Exposures that can not be quantified directly can still be discussed in the uncertainty section of the report.</p> <p>What prevented the collection of PM10 data from near the provincial building, or a similar downwind location that would yield a conservative estimate of air pollution and prevent the necessity of extrapolating PM10 data from the TSP data?</p>	<p>This pathway has been noted as an uncertainty in Chapter 7.</p> <p>The limitations of the data collected by Manitoba Conservation were highlighted in a number of reports prepared during the initial stages of the HHRA which were reviewed by the TAC. Manitoba Conservation made no indications that their current sampling program could be revised to include the collection of PM10 samples at the Provincial Building and the analysis of additional chemicals.</p>
2	Page 2-11		<p>There is a potential for sediment results to be affected by sampling location/method. Typically sediment samples from several points in a line perpendicular to the shore line so assure representative samples. Please explain if some other method was used.</p>	<p>The collection and analysis of sediment samples was completed by Stantec. Please refer to the Stantec report provided in Appendix E of the HHRA for details on the sampling method.</p>

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
2	Page 2-12		Please explain what information was obtained to assure that fish tissue other than muscle tissue was not frequently consumed. Consumption habits may vary with communities and ethnicities.	Residents of the Flin Flon/Creighton area were assumed to have consumption patterns similar to the general Canadian population. Although it is recognized that receptor groups such as First Nations may consume portions of fish and wild game other than muscle tissue, the TAC agreed that the closest First Nations group was located outside of the primary area of impact and that the HHRA did not need to address exposure scenarios specific to these receptors.
2	Page 2-14		The nature of the evaluation of the relevance of fish taken from Ross and Schist Lake should be explained. It seems the risk assessment would want to include fish from lakes impacted by the HBM&S facility.	The fish study conducted by Stantec and Manitoba Conservation included the collection and analysis of numerous fish samples from Schist lake. This data was included in the derivation of EPCs for local fish in the HHRA. Neither Stantec nor Manitoba Conservation attempted to collect fish from Ross lake. According to Stantec, Ross lake does not support any populations of sportfish.
2	Page 2-15		<p>How were exposure areas defined? This the first time they are mentioned, but no methodology is given for their delineation.</p> <p>Please explain why Douglas Lake was not resampled given that it is the raw water source for Creighton and data is 15 year old. It seems that sampling this lake will fill a large data gap in the risk assessment regarding water sources.</p>	Based on the discussion provided on page 2-15 of the draft report, it is unclear what the reviewer is referring to as exposure areas? The area that has been impacted by smelter emissions is discussed in Section 1.4. The communities of interest (COI) are defined in section 3.1.2. The communities of Channing and Creighton were delineated based on the actual municipal boundaries. The communities of East and West Flin Flon were defined based on physical separation by Ross lake. Douglas lake was not re-sampled because the results of the local food survey indicated that it was not commonly used for recreational activities. In addition, recent treated drinking water concentrations for distribution to Creighton residents are available and provide a more accurate representation of the concentrations of COC that residents will be exposed to via the consumption of drinking water.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
2		Table 2-13 and 2-14	Currently it is hard to reconcile the data in the table with the verbal summary. The presentation might be clearly if the table title included the source, that is too say which study it represents.	Revision has been made as suggested.
2	Page 2-18		The final sentence of the final paragraph gives the impression that blueberry data is not available, yet the proceeding section has outlined several sources of such information. Please clarify.	This sentence has been removed from the revised report.
2	Page 2-31		<p>The issue with using the regression equation in Drexler and Brattin (2007) is the accuracy of the regression equation for high values of IVBA. The regression equation under predicts RBA when IVBA is greater than 0.6. Since the IVBA estimates for lead in soil in Flin Flon were 75%, something must be done to improve the RBA estimate.</p> <p>From Figure 4 it appears that the equation underestimates the RBA by about 10%, so the simplest solution would be to boost the RBA value by 10% to 73%.</p> <p>Other solutions include calculating the 95% UCL on the predicted RBAs and using it for the bioavailability adjustment; recalculating the regression equation for points where IVBA is greater than 0.6 or refitting all the points in Drexler and Brattin to a curvilinear equation that fit the data better.</p> <p>There is a paucity of data supporting the RAF for arsenic (Table 2-20). In the comments on the bioaccessibility study, it was noted that further justification/ validation of the bioaccessibility test for arsenic was desirable. This has not been provided. In is unclear from the text whether 34% represents a 95% UCL.</p>	<p>Following review of Drexler and Brattin (2007), the authors make no reference to uncertainty associated with the application of the regression equation when an IVBA exceeds 0.6 and do not recommend the use of any adjustment factors when this occurs. Based on review of Figure 4 of this reference, it appears that use of the regression equation may be just as likely to over-predict as it is to under-predict the RBA when the IVBA is greater than 0.6. No adjustment was made to the RBA used in the HHRA.</p> <p>The basis of this comment is unclear. Bioaccessibility results for arsenic are commonly utilized in HHRA (such as the Sudbury HHRA) and we are aware that Health Canada has accepted this data in the past.</p> <p>The RAF of 34% for arsenic represents the 95% UCLM.</p>

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2	Page 2-32		The food study could be more informative as too what summary statistic consumption was used for fish, berries, mushrooms. Sufficient information should be presented to determine that the values used protective of those members of the community with an above average consumption.	A detailed explanation of the methodologies used to derive consumption rates for local foods is provided in Chapter 4. A summary of the local food survey results is provided in Section 2.10.
4	Page 4-2		Please provide further details on how the divisions between the regions of Flin Flon were determined. Is it possible to provide a map that has sampling points and concentrations? As placement of results in one community or another may make the difference in the risk estimates for each community of interest, it is important that this information be presented in a manner that is easily accessed by reviewers.	A more detailed description the communities of interest (COI) is provided in section 3.1.2. The communities of Channing and Creighton were delineated based on the actual municipal boundaries. The communities of East and West Flin Flon were defined based on physical separation by Ross lake. The residential soil sampling study is provided as Appendix B and includes numerous maps illustrating the sample locations as well as colours to indicate which samples exceeded the CCME guidelines and to what extent.

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4	Page 4-4		<p>An explanation of why no additional air monitoring was undertaken, given the large data gaps that exist surrounding the contaminant concentrations in this media at various locations in Flin Flon would be a useful addition to the risk assessment report.</p> <p>Since the provincial school data represents a downwind community in close proximity to the smelter, it is desirable that the methods used to assess their risk are defensible. A regression equation should only be used if it can be shown to be valid and conservative.</p> <p>It appears the least squares method was used to calculate the regression equations. This method is sensitive to extreme values, and each of the graphs in Appendix I seems to have data points that could be considered outliers, which means the equations are subject to a considerable level of uncertainty. Two possible approaches may help express some of the uncertainty and assure that estimate of contaminant concentration in air in West Flin Flon are conservative: the first is to use a delete-one approach to calculate upper limit estimates for the coefficients in the model. The other is to compare the least squares method with the least absolute deviation regression and select the model which results in the most conservative estimate of PM<sub>10</sub>.</p>	<p>The limitations of the data collected by Manitoba Conservation were highlighted in a number of reports prepared during the initial stages of the HHRA which were reviewed by the TAC. Manitoba Conservation made no indications that their current sampling program could be revised to include the collection of PM<sub>10</sub> samples at the Provincial Building and the analysis of additional chemicals. The correlation of COC concentrations in PM<sub>10</sub> relative to TSP has been revised and a more conservative approach was used. Regression equations are no longer used and instead an upper estimate of the ratio of COC concentrations between PM<sub>10</sub> and TSP has been adopted. Please refer to the revised Appendix I and the discussion in Chapter 4.</p>

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4	Page 4-7/4-8		Given that there the MDL for mercury is 1.0 micrograms/L, it seems that a prudent course of action is to use ½ of this value for the Exposure Point Concentration, or at least select the highest maximum value (0.13 micrograms/L) rather than the UCL.	Table 4-5 of the draft report incorrectly indicated that the detection limit for mercury in Flin Flon drinking water was 1.0 µg/L. This value should have been listed as 0.1 µg/L. As discussed in the text following Table 4-5, the elevated detection limit for mercury reported in the JW study was notably elevated relative to the results of the HBMS sampling program. Use of this elevated detection limit in the derivation of the EPCs would likely significantly over-estimate the concentrations of mercury that residents would be exposed to in drinking water. Therefore, this data was not considered to be appropriate for use in the HHRA.
4	Page 4-17		Appendix P did not contain the results/findings of the food consumption survey, so it was impossible to validate the assumption that 75% of wild game was large game and 25% was wild bird. More information on the food consumption survey would allow for greater transparency of assumptions used in the risk assessment.  Justification of the assumption of a moisture content of berries would be helpful. Did the lab desiccate the berries or make a generic assumption?	A summary of the local food survey is provided in Section 2.10. Additional discussion has been added to Section 4.1.1.8 describing the assumptions used to derive local wild game consumption rates. A reference has been added to support the use of the 85% moisture content assumed for blueberries.
4	Page 4-32		It is reasonable to assume that people, especially children, receive the entire estimated soil intake while outdoors during those months that snow is not covering the ground. The assumption that 55% of daily ingestion will be from dust is likely to underestimate the risks to toddlers and children, because it is possible, even likely, that soil intake results one or two events, rather than a continuous exposure throughout the day. Assuming that 100% of daily soil ingestion is from outdoor soil during 8 months out of the year is considered a reasonable estimate of upper bound exposure.	The HHRA has been revised to assume that for 8 months of the year, 100% of the daily soil/dust ingestion is outdoor soil, and for 4 months of the year, 100% is indoor dust. The dermal exposure pathway has also been adjusted to follow the same assumption ( <i>i.e.</i> , all dermal exposure during summer months is from outdoor soil, and all dermal exposure during winter months is from indoor dust).

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4	Page 4-53		<p>Please note that the bioaccessibility study undertaken for lead in soil gives a good approximation of absolute bioavailability. That is because the bioaccessibility study is correlated to an absolute bioavailability study. An adjustment is only necessary to get to relative bioavailability (that is for comparison with a TRV). The appropriate value for input into the IEUBK model would be the 95% UCLM from the bioaccessibility study.</p>	<p>Absolute bioavailability (ABA) is the ratio of the amount of lead absorbed relative to the amount ingested (U.S. EPA, 2007). To estimate the site-specific ABA of soil: <math>ABA_{soil} = ABA_{soluble} \times RBA_{soil}</math></p> <p>The U.S. EPA indicates that the ABA of soluble lead in water and diet is 50%, therefore: <math>ABA_{soil} = 50\% \times RBA_{soil}</math></p> <p>In the above equation, <math>RBA_{soil}</math> represents the relative <u>bioavailability</u> of lead in soil. The study completed using soils collected from the Flin Flon area was an <i>in vitro</i> test to measure the rate or extent of lead solubilization from soil in a solvent designed to mimic gastric fluid. The fraction of lead which solubilizes in an <i>in vitro</i> test is referred to as <i>in vitro</i> <u>bioaccessibility</u> (IVBA) (U.S. EPA, 2007). Based on a comparison of <i>in vivo</i> and <i>in vitro</i> testing, the U.S. EPA has derived a linear regression to be used to adjust the IVBA test results to produce an <math>RBA_{soil}</math> value: <math>RBA_{soil} = 0.878 \times IVBA - 0.028</math></p> <p>Since the results of the bioaccessibility study on the Flin Flon soils produced a 95% UCLM IVBA of 69%, an <math>RBA_{soil}</math> of 58% was calculated using the above equation. This value was used in the HHRA model-based calculations because the predicted exposure was compared to an RfD which was derived based on studies of soluble lead.</p> <p>Since the IEUBK model requires the use of an <math>ABA_{soil}</math>, the <math>RBA_{soil}</math> was adjusted by the <math>ABA_{soluble}</math> as follows: <math>ABA_{soil} = 50\% \times 58\%</math></p> <p>This produced a value of 29%. It should be noted that this value is essentially equal to the IEUBK default <math>ABA_{soil}</math> of 30%.</p> <p>It should be noted that this process is described in both U.S. EPA (2007) and in the discussion section of Drexler and Brattin (2007).</p>

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4	Page 4-52		It seems that the IEUBK model assumes that bioavailability is the same for dust and lead, however, given that recent research as established that it may be different for the two compounds, this assumption needs to be justified.	Exposure predicted using the HHRA exposure model and the IEUBK model have been revised to assume a 100% lead bioavailability in indoor dust.
4	Table 4-33		Please note that the table lists the bioavailability in dust for the HHRA model to be 30%, however there was no study of bioavailability in dust performed for the HHRA, and it was agreed that the assumption would be 100%. While we recognize that the IEUBK model refers to absolute bioavailability (something that should be clarified in the model) it is important that the difference between the assumptions used in the model, and those agreed upon for the HHRA.	The HHRA and the assumptions listed in this table have been revised to use an assumed 100% lead bioavailability in indoor dust within the HHRA model. Within the IEUBK model, the default dust bioavailability of 30% was used.
4	Page 4-53		The bioavailability numbers in the text, do not match those presented earlier in the data gap analysis. Comments made on regression between bioaccessibility and bioavailability (page 2-21) are equally applicable here.	Chapter 2 has been revised to reflect the results of the updated bioaccessibility analysis. The 95% UCLM phase 1 lead IVBA of 69% was converted to an RBA <sub>soil</sub> of 58% and an ABA <sub>soil</sub> of 29%.
4	Page 4-54		Please discuss whether the assumptions in the IEUBK model, particularly in regards to air, are appropriate for a site with a continuous point source.	The development of the IEUBK model considered studies on communities with and without point sources of lead. For example, the derivation of the default bioavailability of lead in the lungs was based on the deposition/absorption of lead in respiratory tissues of young children in communities with and without point sources of lead. In addition, the derivation of the Multiple Source Analysis (MSA) module to relate concentrations of lead in indoor dust to outdoor air and soil was based partially on measurements in homes near lead point sources. One concern that has been raised regarding the use of the IEUBK model in communities with ongoing point sources is that the direct deposition of contaminants on hard surfaces may create concentrations of contaminants that are higher than those measured in soil. This may create a soil-related medium that is distinct from “outdoor soil” and “indoor dust”. However, this issue is not unique to the IEUBK model and is a valid point to be considered in any HHRA.

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5			General Comment (Chapter 5): The use of PRG to assess the number of properties with metal concentrations that could result in an unacceptable risk is very much appreciated and show a thoughtfulness about protecting each individual in the community. It would, however, be very useful to have more information about how these PRGs were calculated (including sample calculations) so that a more complete review can be performed.	Specific details regarding the derivation of PRGs/STCs are provided in Chapter 5 for each COC.
5	Page 5-5		Table 5-1 compares 24 hour air concentrations to 1 hour toxicity reference values. This implies that the average over 24 hours would be greater than the acceptable level for just one hour of exposure. These estimates, although better than nothing, are not just greatly uncertain, but would greatly underestimate the risk from exposure to peaks in contaminant concentrations	Agreed.
5	5.1.2		This section does not explain how acute ingestion exposure estimates were arrived at, nor was it possible to locate this information in any of the previous sections. Please provide the information or a reference to where the information is to be found. It should be noted that a toxicity reference values such as a tolerable daily intake or reference value does convey any information about the shape or slope of the dose response curve. Qualifying the exceedances in Table 5-3 as marginal may or may not be accurate, but there is not really sufficient information to support the claim.	The basis of this question is unclear since Section 5.1.2 does include equations and assumptions used to estimate acute ingestion exposure. Reference to marginal exceedances has been removed.
5	Page 5-8		Contamination in drinking water is referred to as background, but isn't it possible that smelter activities have contributed to the concentrations in drinking water?	It is unclear why the reviewer has indicated that drinking water is referred to as background. The HHRA has not treated COC content in drinking water as background and acknowledges that it is likely influenced by smelter activities.

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5	Page5-16		Although there are limits in the epidemiological studies of arsenic exposure in Taiwan, these studies have been used in preference to other studies because they show the direct effects of arsenic exposure in human populations. IRAC considered evidence of carcinogenicity in humans to be superior to animal studies when classifying carcinogens.	Agreed, this wording has been modified.
5	Page 5-17		The difference between cancer risk attributable to arsenic in all the regions of Flin Flon, and background level is an order of magnitude. The discussion should exercise care to prevent marginalizing this difference.	Agreed.
5	Page 5-24		Unfortunately the Community Health Assessment was limited by available data and by the small population size, and is not sufficient to determine the true risk of lung cancer in the Flin Flon area.	Agreed, this wording has been modified.
5	Page 5-31		The data gap analysis makes it sound as though samples from properties were composited, yet the assessment of copper exposure gives maximum values for properties. Which is the case? A composited sample is more representative of a mean value.	The reported concentrations are for composite samples. For some properties, a number of samples from different areas (e.g., front yard, backyard, garden) were collected (each as a composite of samples). The comparison of sample concentrations to the PRG involved a comparison to each sample rather than taking an average for those properties with multiple samples.

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5	Page 5.4 /Table 5-44		Dermal and oral exposure should be summed. It should be noted that these risks are over and above the risk predicted for residential exposure, so while they are not consequential in and of themselves, they do reduce the conservative nature of other risk estimates.	Agreed.
<b>Manitoba Conservation - submitted Feb.10/09</b>				
General Comments			OVERALL GENERAL/KEY COMMENTS:- generally, the report is well-written, professional and covers the subject matter in a thorough way- several substantive matters for follow-up have been detailed in the comments below including: a. more information is needed on how the bio-monitoring survey information can be used, b. the toxicological reference doses for air exposure for arsenic, cadmium and copper probably need to be changed (and related risk calculation repeated), c. more advice on mitigation options in fulfillment of study objective #2 is required	Revision has been made as suggested; see below.
Exec Sum			1. Executive Summary: For the final draft report, an Executive Summary will need to be included. A draft of this Executive Summary should be provided to the Technical Advisory Committee (TAC) for review before inclusion in the final report.	An executive summary has been added to the revised report.

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1	p. 1-4, 1-6	1.3 Study Objectives	<p>Page 1-4 states that “this report addresses the assessment of health risk, but does not provide risk management options.” Such options are to be addressed separately. On page 1-6, Objective 2 indicates that an objective of the HHRA is “to develop risk management objectives and/or mitigation plans if unacceptable risk levels are identified in the HHRA.” Section 8.2 summarizes “unacceptable” risk for chronic exposure to arsenic, lead and mercury in certain areas of the communities. Though it is recognized that recommendations are made for bio-monitoring as a follow-up to confirm/adjust the predictions of the HHRA, it would appear that more information/advice might be offered to fulfill this study objective, since one is led to believe that this objective is addressed in the report. This additional information might include other potential mitigation measures to consider for different future time horizons (short, mid and long term). For example, while results of the biomonitoring study are being awaited, it is conceivable that such results might not be available until mid-2011 if the surveys can’t be completed in 2009 and have to be delayed to the late summer of 2010. In the meantime, what is the range of mitigation measures that should and can be undertaken to respond to the predicted risk?</p>	<p>The wording in the section will be clarified. The current schedule anticipates completion of the biomonitoring study in early 2010, to coincide with release of the HHRA report. As such, the need for risk management will be informed by both the HHRA and the biomonitoring study. The nature and extent of required risk management will be assessed at a later stage in the process. Interim risk management measures, several things that residents can do to reduce exposure to metals in the environment while the studies are ongoing, have been added to the recommendations (Chapter 8) section of the report.</p>

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1	p. 1-8	1.5 Study Scope: Time Scale of Risk Assessment	By focusing only on the risk from current and future exposures, the lifetime risk faced by current, long-term residents of Flin Flon and Creighton is likely being underestimated since the air emissions and subsequent exposures in previous years were undoubtedly higher. Consequently, this current study would be more representative of the future risk of newer residents to the area. A statement acknowledging this limitation should be included in the report.	This discussion has been revised to reflect the issue addressed in this comment.
1			Throughout the Introduction, the text is written in a rather confusing mix of future tense (e.g., "sources will be", "conclusions will be evaluated") mixed with past tense (e.g., "information was gathered", "zinc was found in excess"). As an example, in the section on Indoor Air on page 1-13, the text describes what might be done rather than what actually was done in the assessment. It is suggested that the Introduction should consistently provide an overview of what was actually done (i.e., use the past tense).	Chapter one has been revised to consistently use past tense.
2		2.9.1 Overview of Bioaccessibility	Given that bioaccessibility values can differ widely since methodologies to mirror human systems are still in development and that specific direction was provided to Intrinsic by the government members of TAC (with an understanding that the peer panel review would consider this as well), perhaps some better context needs to be placed on the significance of bioaccessibility to the risk findings. Can an analysis be done of the sensitivity of the HHRA to the use of bioaccessibility? What would be the risk levels if bioaccessibility were not incorporated via the relative absorption factor (RAF)? What would the risk levels be if bioaccessibility factors were allowed for other metals?	The sensitivity analysis in Section 7.4 provides a quantitative assessment of the affect of varying the soil bioaccessibility on the predicted risk levels.

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4	page 4-4	4.1.1.2 Ambient Air Concs.	<p>The HHRA's approach to using the PM10 fraction is consistent with Health Canada's position, as outlined in the document "Federal Contaminated Sites Risk Assessment in Canada; Part I: Guidance on Human Health Preliminary Quantitative Risk Assessment" From an inhalation perspective, the only fraction that is relevant is the respirable fraction since larger particles would not be inhaled. These larger particles, however, would still be included in the risk assessment through the soil concentrations (containing deposited lead particles).</p> <p>The recent revised EPA NAAQS for lead recognizes that the airborne lead is not only inhaled but can also be deposited leading to other potential exposure pathways (e.g., ingestion). The NAAQS is therefore based on TSP so that all potential pathways are taken into account. In particular, on page 66988, "EPA received comments expressing concern that because only a fraction of airborne particulate matter is respirable, an air standard based on total air Pb would be unnecessarily stringent and therefore the standard should be limited to respirable size Pb particulate matter. Such a standard might have led to a Pb NAAQS with an indicator of Pb in particulate matter less than or equal to 10 µm in diameter (Pb-PM10) as the indicator. The Agency considered this recommendation, but did not accept it. Rather, EPA reemphasized that larger particles of air-related Pb contribute to Pb exposure through ingestion pathways, and that ingestion pathways, including those associated with deposition of Pb from the air, can be a significant component of Pb exposures. In addition to these ingestion exposure pathways, nonrespirable Pb that has been emitted to the ambient air may, at some point, become respirable through weathering or mechanical action, thus subsequently contributing to</p>	No response or revisions required.

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			<p>inhalation exposures. EPA concluded that total airborne Pb, both respirable and nonrespirable fractions, should be addressed by the air standard (43 FR 46251)."</p> <p>For this risk assessment, though the PM<sub>10</sub> fraction probably best represents the inhalation pathway, this may be a question for the peer review panel to address and to confirm the approach.</p>	
4	p. 4-5	Table 4-4	The date range for the air quality data used in the assessment should be included as a footnote in this table.	A footnote has been added to the table as suggested.
4		4.1.1.4	The text states at the outset of this chapter that all data that were less than the method detection limit (MDL) were conservatively assumed to be present at the MDL value. With this in mind, why was the MDL for Hg (0.1 µg/L) in samples from the JW Water Sampling Program not put forth as the overall EPC for Hg in Section 4.1.1.4. Note that the JW results for Cu are considerably higher than the HBM&S results and yet were used as the EPC.	As discussed in the text following Table 4-5, the elevated detection limit for mercury reported in the JW study was notably elevated relative to the results of the HBMS sampling program. Use of this elevated detection limit in the derivation of the EPCs would likely significantly over-estimate the concentrations of mercury that residents would be exposed to in drinking water. Therefore, this data was not considered to be appropriate for use in the HHRA. The JW results for copper were used in the derivation of the EPCs because these were <u>measured</u> values and were not elevated as a result of an elevated detection limit. These values were considered to be reflective of concentrations of copper in Flin Flin drinking water.
4		4.1.1.4	The Hg results in Table 4-5 appear to be incorrect. The table and immediate text indicate that the MDL for Hg was 1.0 µg/L, when it actually was 0.1 µg/L (see Table 4-6 and Appendix Q).	Revision has been made as suggested.
4	p. 4-9	4.1.1.5 Vegetable Garden Produce Concs.	The statement is made that the "chemical form of arsenic ( <i>i.e.</i> , organic or inorganic)...is a significant factor". A brief one-sentence explanation should be included stating why the form of arsenic is significant.	This discussion has been revised for clarification.
4		4.1.1.5	The reference to "Manitoba Conservation, 2006" in this section is incorrect. The correct reference to the 2002 garden study report is Jones and Henderson (2006).	All references to this study have been revised as suggested.
4		4.1.1.5	The EPCs for the above ground home grown	The report incorrectly indicated that there were 141 washed

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			vegetables may need partial or complete recalculating. The paragraph preceding Table 4-7 states that the number of above ground vegetable samples was 141, when in fact it was 75. Please verify that the calculations were done using the correct samples (washed only) and the correct sample size.	samples. The derivation of the above-ground produce EPC was based on the 75 washed samples. This revision has been made to the report. No corrections to the exposure and risk calculations are required.
4		4.1.1.5	A more accurate estimate of the inorganic fraction of total As in lettuce should be sought if one is available? The edible parts of beans and lettuce are quite different structurally ( <i>i.e.</i> , fruit vs. leaves) and this may influence the fraction of the total As that is in inorganic form in either type of vegetable. Also, since lettuce tended to accumulate the highest concentrations of arsenic (relative to the rest of the vegetables tested) it would be prudent to be as precise as possible, or to perhaps error on the side of caution and assume 100% inorganic As. Perhaps estimates for other leafy vegetable species such as spinach, kale are available?	The assessment has been revised to assume that 100% of the total arsenic measured in lettuce is in the inorganic form.
4		4.1.1.6 (and other Sections <i>e.g.</i> , 5.2.5.2)	The proportion of the total Hg in fish in the form of methyl Hg was estimated at 79%. This was based on the relationship between methyl Hg and total Hg in nine fish samples. This seems like a fairly small sample size to base such a relationship, particularly since there was a fairly wide variation in the methyl:total Hg ratio in the samples tested. Is there any information in the literature to support using the 0.79 fraction?	The report has been revised to use the 95% UCLM fraction of methyl mercury content in local fish (96%) as opposed to the average (79%) originally used. Given that Health Canada indicates that methyl mercury content is commonly within the range of 60 to 95% of total mercury, use of 96% is considered sufficiently conservative.

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4		4.1.1.7	Table 4-13 suggests that the IEUBK model underestimated the Pb concentrations in house dust in East Flin Flon and Creighton communities, yet the IEUBK derived concentrations were put forth as the EPC for Pb in house dust for these communities. Please comment on this decision.	The HHRA has been revised to use the measured indoor dust lead concentrations in the exposure and risk assessment rather than the IEUBK-derived concentrations.
4		4.1.1.8	The footnote for Table 4-15 indicates that an adjustment factor (0.01) was used to calculate the inorganic fraction of total As in wild game. A reference in the literature should be provided to support the adjustment factor that was used	The adjustment factor of 0.01 is recommended for both chicken and beef by Schoof <i>et al.</i> (1999). This reference has been added to the footnote for Table 4-15.
4		4.1.1.8	Is there a reference or some other information available to support the assumption that 75% of the annual consumption of wild game was wild mammals and that 25% was wild birds.	These apportionments are similar to the central tendency estimates (CTE) derived for hunting populations in the Sudbury area in which survey respondents indicated that 26% of the annual wild game meals were wild birds with the remaining 74% large game. This discussion has been included in the revised report.
4		4.1.1.8	MC has some data on metal content in natural vegetation in the Flin Flon area (includes Labrador tea, alder, black spruce, jack pine, and wild sarsaparilla). This could be made available to the HHRA.	This data has been incorporated into the derivation of wild game meat concentrations,
4		4.1.1.9	The footnote for Table 4-17 indicates that an adjustment factor for grapes (0.35) was used to calculate the inorganic fraction of total As in blue berries. A reference in the literature should be provided to support the adjustment factor that was used. As well, given that grapes and blue berries are not related species, is the fraction used for grapes applicable for use with blue berries?	An inorganic arsenic content for blueberries was not identified in the literature. Within the Sudbury HHRA, an adjustment factor of 0.33 was used based on the average of all values for fruits reported within Schoof <i>et al.</i> (1999). Given this, and the similarities in skin texture between grapes and blueberries, a factor of 0.35 is considered to be appropriate for use in the current assessment.

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4		4.1.2.1	The reference used for the “Background Soil Concentrations” is incorrect. It should be Manitoba Conservation 2007 (not 2006). As indicated in an earlier comment, the 2006 report is the report on the garden study that was conducted in 2002, while the 2007 report is the report on the urban soil survey that was conducted in 2006. Also, the only “background” samples during the 2006 survey were from Cranberry Portage; no samples were collected in The Pas during the 2006 survey.	Revision has been made as suggested.
4		4.1.2.1	Drinking water data from Ontario were used in the calculation of background levels. Why were data from Saskatchewan and Manitoba communities not used in the determination of background exposure?	Despite efforts by members of the TAC, background drinking water concentrations could not be obtained for Manitoba or Saskatchewan. The report could not be revised, however, it is not anticipated that concentrations of COC in municipally supplied drinking water are significantly different from province to province.
4		4.1.2.1	There appears to be an error in the Min and Max values for Cu in Table 4-21.	The min, max and average columns from this table have been removed. They will be added to the final report once the correct values can be confirmed.
4		4.1.3	The footnote to Table 4-23 indicates that 3 samples were excluded from the vegetable category and 1 sample was excluded from the fruit category because the inorganic As content of these particular samples was reported as greater than the corresponding total As content. Why not just assume a fraction factor of 1.0 for these samples (as was done for methyl mercury when it exceeded total mercury in fish samples)?	Revision has been made as suggested.

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4		4.1.3	Concentrations in food in Tables 4-23 and 4-24 are expressed in ng/g, while in other tables showing concentration in food the units used are µg /g (e.g., Tables 4-7, 4-8, 4-25, and others). Using a consistent unit (i.e., µg/g) to express concentration should be considered.	Concentrations in Tables 4-23 and 4-24 have been converted to units of µg/g to be consistent with the remainder of the report.
4		4.1.3	Effort should be made to format Table 4-25 so that it appears complete on a single page.	Table 4-25 cannot be formatted to fit within a single page while still using a reasonable size font.
4	p. 4-69	4.2.3.1 Arsenic Toxicological References Doses	The Ontario Ministry of the Environment (OMOE) 24-hour average air quality criterion for arsenic of 0.3 µg/m <sup>3</sup> was used for both the 24-hour and chronic inhalation exposure limit. While the limit was developed for and is appropriate for the 24-hour averaging period, we are advised that it should not be considered for the longer-term chronic exposures. In the original draft toxicological profile for arsenic (provided in August 2008 by Intrinsic), a value for the long-term inhalation exposure developed by the California EPA had been selected, namely 0.03 µg/m <sup>3</sup> . Why was the value used for the chronic arsenic TRV changed?	Revision has been made as suggested.

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4	p. 4-72	4.2.3.2 Cadmium Toxicological References Doses	<p>For this assessment, the Ontario Ministry of the Environment (OMOE) Upper Risk Threshold (URT) criterion of 0.25 µg/m<sup>3</sup> was selected for the 24-hour average toxicological reference value (TRV) for cadmium. For this averaging period, we believe that a more appropriate value would have been the OMOE air quality criterion of 0.025 µg/m<sup>3</sup> as a 24-hour average.</p> <p>The argument was made that the 0.025 µg/m<sup>3</sup> air quality standard is not being phased in until February 2013 and so is presumably not relevant for this current assessment. It is our understanding that the phase-in period reflects the fact that the new standard is more stringent and some companies may have difficulty meeting it. The decision to have a phase-in period in Ontario is a management rather than a science-based decision; the lower 0.025 µg/m<sup>3</sup> limit is more appropriate since it is strictly science-based with no consideration of technological or economic factors. By using the URT for cadmium in the assessment, a standard is being used that, if exceeded in Ontario, would typically require abatement action by a company. In this sense, the URT is comparable to Manitoba's Maximum Tolerable Concentration. Under OMOE Regulation 419 – Local Air Quality, an exceedance of a URT would require a facility to immediately notify the OMOE in writing of the exceedance, and the company would then have three months to assess the emissions and submit an Emission Summary and Dispersion Modelling report to the OMOE. Continuing exceedances would necessitate abatement action.</p> <p>As stated, the most appropriate, health-based TRV for cadmium would be the value that is being phased in, namely 0.025 µg/m<sup>3</sup>. In the original draft cadmium toxicological profile of August 2008 provided by Intrinsic, Intrinsic had indicated that they would be</p>	<p>We disagree that it would be appropriate to use the OMOE air quality criterion of 0.025 µg/m<sup>3</sup> as a 24-hour average. All TRV selected for this assessment are valued developed by reputable regulatory agencies. While OMOE has developed the 24-hour air quality criterion of 0.025 µg/m<sup>3</sup>, this value is not being phased in until 2013 and as such cannot be considered a regulatory value at this time. It is our opinion that the OMOE URT criterion of 0.25 µg/m<sup>3</sup> is more appropriate for use at this time. A discussion and comparison of these criterion have been added to Chapter 5 of the revised report.</p> <p>For the chronic exposure (annual average) evaluation, the OMOE value of 0.005 µg/m<sup>3</sup> has not been utilized since it is based on an endpoint of cancer and cancer risks are evaluated utilizing the Health Canada Inhalation Unit Risk and as such use of the 0.005 µg/m<sup>3</sup> criterion is not considered necessary.</p>

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			<p>using 0.025 µg/m<sup>3</sup> as the short-term, acute, inhalation TRV for cadmium. Using the 0.025 µg/m<sup>3</sup> air quality standard will have implications for the results in this Human Health Risk Assessment.</p> <p>The value used for the annual average inhalation TRV for cadmium is not stated in the text, although Table 4-38 (p. 4-61) shows a value of 0.25 µg/m<sup>3</sup> for the chronic inhalation TRV. This use of a 24-hour average URT is inappropriate for a chronic inhalation limit. For the long-term, chronic inhalation TRV, Intrinsic had originally proposed using 0.005 µg/m<sup>3</sup>.</p>	

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4	p. 4-75	4.2.3.2 Copper Toxicological References Doses	The Ontario Ministry of the Environment (OMOE) 24-hour average air quality criterion for copper of 50 µg/m <sup>3</sup> was used for both the 24-hour and chronic exposure limit. While the limit was developed for and is appropriate for the 24-hour averaging period, it probably should not be considered for the longer-term chronic exposures. In the original draft toxicological profile for copper (provided in August 2008 by Intrinsic), the long-term inhalation exposure developed by RIVM had been selected, namely 1.0 µg/m <sup>3</sup> . Why was the value selected for the chronic exposure TRV changed?	Revision has been made as suggested.
4	p. 4-81	4.2.3.4 Lead Toxicological References Doses	Table 4-39 Summary of the Carcinogenic Toxicological Criteria chosen for the Human Health Risk Assessment lists two limits for lead but these limits were not described in the text nor were they used in the assessment. If these limits were not used in this assessment, why are they included in this table?	These limits have been removed from the table. Cal EPA is the only known regulatory agency to have derived exposure limits for lead based on carcinogenic effects and the U.S. EPA has determined that an estimate of carcinogenic risk from oral exposure (such as slope factor) using standard methods would not adequately describe the potential risk for lead compounds. As a result lead was not evaluated in this manner.
4	pp. 4-86 to 4-87	4.2.3.5 Mercury Toxicological References Doses	<ul style="list-style-type: none"> <li>Both of the values described (<i>i.e.</i>, 0.3 µg/m<sup>3</sup> and 0.06 µg/m<sup>3</sup>) are presented in the text as being the chronic inhalation exposure limit chosen for elemental mercury. In Table 4-38, the TRV listed for elemental mercury was 0.06 µg/m<sup>3</sup>.</li> <li>For inorganic mercury, the acute oral limit of 7.0 µg/kg/day, which is listed in Table 4-38, is not described in the text.</li> </ul>	The correct chronic TRV for elemental mercury is 0.06 µg/m <sup>3</sup> . Discussion regarding the acute MRL for inorganic mercury has been added to the text.
4	p. 4-94	4.3 Risk Characterization	The statement is made that, in the Provinces of Manitoba and Saskatchewan, the acceptable risk level for cancer risk is 1-in-100,000. For completeness, some type of reference or citation to an official statement of government policy from either province would help to support this assertion.	Additional discussion has been added to this section along with a reference to Health Canada (2006).

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5	p. 5-3	5.0 Results and Discussion	Biomonitoring is suggested as a means to further evaluate risk levels and to establish preliminary remediation goals (PRGs) for the COC. Before any biomonitoring programs are undertaken, however, the consultant will need to provide information to the TAC indicating their proposed methodology for the surveys and how they propose to use the results of these surveys (e.g., to “ground truth” the HHRA, to directly assess human health impacts, to set PRGs). Sufficient details on the methodology proposed, especially for the setting of PRGs, needs to be provided for review and approval by the TAC. This information also needs to be integrated into the draft report so that it can be part of the peer panel review.	Following the discussion of this topic during the TAC meeting in Winnipeg on February 23, 2009, it has been decided that no specific methodology for the integration of the biomonitoring results into the HHRA can be established prior to the completion of the biomonitoring program and analysis of the results.
5	p. 5-3	5.0 Results and Discussion	Which source of information will take precedence, the HHRA or the biomonitoring surveys? What approach will be taken if the biomonitoring results show low exposures relative to the HHRA? What about high exposures, especially if the biomonitoring data identifies levels associated with known health effects?	This issue will be addressed at a later date when the TAC has reviewed the study outcome and the confidence in the biomonitoring results can be determined.
5	p. 5-3	Table 5-1 Acute Inhalation Risk Estimates	Using the more appropriate inhalation TRV for cadmium of 0.025 µg/m <sup>3</sup> increases the number of times the TRV is exceeded from 1.9 to 19 in West Flin Flon, from 0.48 to 4.8 in East Flin Flon and Channing. Only Creighton would not be expected to have any exceedances.	The number of exceedances does not increased in a linear manner. The number of exceedances for the various TRVs are provided in Table 5-3.

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5	p. 5-6	Table 5-2 Frequency of 24-hour CRacute Exceedances,	<ul style="list-style-type: none"> <li>Using the more appropriate cadmium inhalation TRV could significantly increase the frequency of exceedances from the "2 of 210" shown in the table.</li> <li>In the text preceding this table, the statement is made that "occurrences are rare" of the exceedances. For lead, however, the frequency is "21 of 210" (<i>i.e.</i>, 10% of the samples), a frequency which is not insignificant.</li> <li>It would be beneficial to add the time period over which the samples were taken as a footnote to the table. The information would give context for the exceedances (<i>i.e.</i>, Did the 21 exceedances of the lead TRV occur in 1 year?).</li> </ul>	<p>Agreed, the impact of using alternative TRVs for cadmium has been presented in Table 5-3.</p> <p>The statement has been qualified to indicate that exceedances are somewhat rare.</p> <p>A footnote has been added to Table 5-2 to indicate the time period during which exceedances occurred.</p>
5	p. 5-11	5.2.1 Arsenic, Table 5-7 Concentration Ratios for Exposure to Arsenic in Ambient Air	<p>Using a more appropriate long-term inhalation TRV for arsenic of 0.03 µg/m<sup>3</sup> would increase the concentration ratio from 0.14 to 1.4 in East Flin Flon, from 0.28 to 2.8 in West Flin Flon and from 0.14 to 1.4 in Creighton. Only Channing would not have a concentration ratio greater than 1.0. The conclusion that "arsenic in ambient air in each of the COI are below the air standard protective of adverse effects to respiratory tissues" would no longer be valid for three of the four areas.</p>	<p>Agreed, the report has been modified to reflect the more conservative arsenic TRV.</p>
5	p. 5-18	Weight-of-Evidence Discussion for Arsenic	<p>The second bullet states that "Market basket foods were the main contributor to arsenic related risks for both the typical resident and the typical Flin Flon resident". This statement contradicts Figure 5-6 which compares cancer risk levels associated with site-related environmental exposure and the exposure from the consumption of market basket foods. For all cases, except the background, the risk from the site-related exposures exceeded the risk from the market basket foods.</p>	<p>This bullet has been revised to indicate that market basket foods was the main contributor to non-cancer risks (as illustrated in Figure 5-4).</p>
5	p. 5-19	Weight-of-Evidence Discussion for Arsenic	<p>The recommendation that a urinary arsenic study be conducted to assess the exposures from both the soil and air pathways is supported.</p>	<p>No response or revisions required.</p>
5	p. 5-19	Derivation of the STC for Arsenic	<ul style="list-style-type: none"> <li>Given that the most significant contributor to arsenic risk in East and West Flin Flon and Channing is</li> </ul>	<p>The STC is based on an incremental lifetime cancer risk estimate and the characteristics of a receptor over their</p>

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			through air inhalation (see Figure 5-5), why is no consideration being given to the reduction of air exposures to arsenic? <ul style="list-style-type: none"> <li>• Why is the STC based on an adult and not a toddler since a toddler is the more sensitive receptor? The toddler was used as the basis for criteria for other contaminants (e.g., cadmium, lead).</li> </ul>	entire lifetime are more accurately reflected by the characteristics of the adult. The derivation of the PRGs/STCs for carcinogens are consistent with the approaches recommended by agencies such as Health Canada, CCME, and the U.S. EPA. Health Canada acknowledged their support of this approach at the TAC meeting in Winnipeg on February 23, 2009.
5		5.2.2 Cadmium, Figure 5-9 Pathway Specific Hazard Quotients for a Toddler in each COI as a Result of Exposure to Cadmium	Using a health-based, chronic TRV for cadmium instead of the short-term Upper Risk Threshold would undoubtedly result in a higher air pathway component to the hazard quotient.	This statement is correct, As indicated previously, we disagree with the use of 0.025 µg/m <sup>3</sup> as an ambient air standard for Cadmium at this time.
5	p. 5-24	ILCR Estimates for Cadmium	As noted earlier, the use of the Upper Risk Threshold of 0.25 µg/m <sup>3</sup> is not appropriate since the use of this interim level is a management rather than a science-based decision. From Table 5-13, the use of the more appropriate level of 0.025 µg/m <sup>3</sup> results in 64 exceedances at the Provincial Building. In other words, 30% of the samples exceeded the lower criterion.	This statement is correct, As indicated previously, we disagree with the use of 0.025 µg/m <sup>3</sup> as an ambient air standard for Cadmium at this time.
5	p. 5-25	Derivation of a Residential Cadmium Soil PRG	Given that the lifetime cancer risk from the air pathway is significant (e.g., inhalation cancer risk ranges from 4.5(10 <sup>-5</sup> ) to 4.7(10 <sup>-4</sup> )), why was no consideration given to lowering inhalation exposures?	The results clearly indicated that consideration should be given to the reduction of exposures from this pathway. The report has been modified to reflect this concern.

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5	p. 5-30	5.2.3 Copper, Table 5-18 Concentration Ratios for Exposure to Copper in Ambient Air	The use of a more appropriate, lower long-term inhalation TRV for copper of 1.0 µg/m <sup>3</sup> does not change the conclusion that “concentrations of copper in ambient air in each of the COI are well below the RfC protective of adverse respiratory effects”. While the values of the concentration ratios do substantially increase, from 0.0045 to 0.223 in East Flin Flon, from 0.015 to 0.731 in West Flin Flon, from 0.0012 to 0.0581 in Creighton and from 0.0045 to 0.223 in Channing, the concentration ratios do all remain below 1.0.	No response or revisions required.
5	p. 5-50	5.2.4 Lead	The recommendation that a blood lead survey be conducted in Flin Flon and Creighton is supported.	No response or revisions required.
5	p. 5-51	5.2.5 Mercury	Is the assumption that 20% of the mercury in the air is methyl mercury still valid given that there is a local industrial source of mercury?	Anthropogenic sources of mercury generally do not produce methyl mercury. Methyl mercury in the environment is produced by biological organisms in soil, lakes, rivers, etc. Methyl mercury is then transported into air through volatilization or activities such as forest fires. The CCME recommends an assumption of 20% of total mercury present in ambient air as methyl mercury (CCME, 1996). Although this assumption is considered to be conservative, it was utilized in the current assessment and is not anticipated to underestimate the methyl mercury component of smelter emissions.
5	pp. 5-56 to 5-57	5.2.5.1 Inorganic Mercury	The recommendation that a biomonitoring program for inorganic mercury be conducted is supported.	No response or revisions required.
5		5.2.5.2	States in the last paragraph on the discussion of methyl Hg exposure that “17 of the 166 (fish) samples contained concentrations of total mercury above the human consumption guideline (0.5 µg/g d.w. total mercury) recommended by Health Canada”. Is this a correct interpretation and use of the guideline?	This discussion has been revised and additional references for consumption guidelines have been incorporated.

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6	pp. 6-9 to 6-22	6.4 Chemical Mixtures	Given that the concept of chemical mixtures is not incorporated into this HHRA, is there a need to discuss this concept in such detail? This material would better fit in an Appendix than in the main document with just a brief summary remaining in the main text.	The concept of chemical mixtures and the associated uncertainties are an important issue to consider when interpreting the results of an HHRA. It is felt that this discussion should remain within the main document rather than moving it to an appendix where it may not receive an appropriate level of attention.
6	pp. 6-26 to 6-29	6.6 Community Health Status Assessment: Flin Flon and Creighton	Given the small populations of Flin Flon and Creighton, can definitive conclusions be made for the specific health measures ( <i>i.e.</i> , mortality, cancer)? MB/SK Health needs to confirm this and ensure that it is an accurate use of their study findings.	The discussion provided in Section 6.6 summarizes the findings of the Community Health Assessment. Revisions can be made to this discussion if public health officials from Manitoba Health and Healthy Living and the Saskatchewan Ministry of Health indicate that they are warranted.
6		6.8 COC Lifetime Body Burden	The statement is made that the “COC in question do not bioaccumulate, resulting in very little body burden over time.” This statement is somewhat misleading, since lead (one of the COCs) does accumulate in the body where it can be stored in the bone. (This storage is described on p. 6-34 – 90% of the body burden of lead is stored in the bone while infants retain 31.7% of the total amount of lead absorbed).	Agreed, the text has been modified to reflect this concern.
7	p. 7-3		“Projected chemical concentrations in media used in the exposure modeling were assumed to remain unchanged over time”: The statement is made that “due to the continuing rate of decrease in smelter emissions...., media concentrations of the COC ... are expected to progressively decrease over time.” While this may be true of air and water concentrations, why would metal concentrations in the soil decline over time since metals do not degrade in the environment? While a decrease in smelter emissions would lead to a decreased rate of metal deposition over time, wouldn't the end result be a slower rate of accumulation rather than a decrease in concentrations?	This discussion has been revised to indicate that the rate of accumulation in environmental media is anticipated to decrease in the future. It has also been indicated that the assumption that current concentrations will occur 80 years into the future may underestimate risks <i>via</i> some pathways ( <i>e.g.</i> , outdoor soil ingestion) and overestimate risks <i>via</i> others ( <i>e.g.</i> , inhalation of outdoor air).

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8	p. 8-1	Table 8-1 Acute Inhalation Risk Estimates	<ul style="list-style-type: none"> <li>As noted previously, using the lower, short-term inhalation TRV for cadmium of 0.025 µg/m<sup>3</sup> would result in exceedances of the cadmium TRV in West Flin Flon (CRacute=19), East Flin Flon (CRacute=4.8) and Channing (CRacute=4.8). Only Creighton would experience no exceedances (CRacute=0.72). The magnitude of the exceedances is no longer “small”, especially in West Flin Flon.</li> <li>The magnitude of CRacute is 3.4 for arsenic and 4.2 for lead at West Flin Flon. While the consultant states that the “magnitude of exceedances are small”, 3.4 and 4.2 times the TRV for arsenic and lead, respectively, are not insignificant.</li> </ul>	<p>This statement is correct, As indicated previously, we disagree with the use of 0.025 µg/m<sup>3</sup> as an ambient air standard for Cadmium at this time. The statement has been qualified.</p>
8	p. 8-2	Table 8-2 Frequency of 24-hour CRacute Exceedances	<ul style="list-style-type: none"> <li>With a lower inhalation TRV for cadmium, one would expect the frequency of exceedances to be much higher than “2 of 210” in West Flin Flon and greater than 0 in both East Flin Flon and Channing.</li> <li>The occurrences of exceedances are described as “rare”. For lead, however, “21 of 210” is equivalent to a percentage exceedance of 10%. This 10% would be equivalent to exceedances 3 days/month, which could not be described as “rare”.</li> </ul>	<p>This statement is correct, As indicated previously, we disagree with the use of 0.025 µg/m<sup>3</sup> as an ambient air standard for Cadmium at this time. The statement has been qualified to indicate that exceedances are somewhat rare.</p>
8	p. 8-5	Cadmium	<p>The statement is made that the Community Health Assessment “presents a more accurate measure of the occurrence of lung cancer.” than that suggested by the application of Health Canada cancer risk values. MB/SK Health needs to confirm this and ensure that it is an accurate use of their study findings.</p>	<p>Revisions can be made to this discussion if public health officials from Manitoba Health and Healthy Living and the Saskatchewan Ministry of Health indicate that they are warranted.</p>

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Appendix A		Toxicological Profiles	Appendix A1-Arsenic: Table A1-1 Summary Table of Toxicity Reference Values Selected for the HHRA: The discussion states that the US EPA inhalation unit risk factor of 4.3(10 <sup>-3</sup> ) (µg/m <sup>3</sup> )-1 should not be used if the air concentration exceeds 2 µg/m <sup>3</sup> . A footnote to that effect should be included in Table A1-1.	Revision has been made as suggested.
Appendix A		Toxicological Profiles	Appendix A2-Selenium: In the response to previous comments (see Appendix F – Response to Comments – tox profiles), the comment was made that an appropriate short term oral TRV would be selected for selenium. In the final draft, however, no oral TRV was included so the response in Appendix F should be revised.	Revision has been made as suggested.
Appendix A		Toxicological Profiles	Appendix A4-Cadmium: No discussion is provided in the text justifying the use of the cadmium upper risk threshold (URT) as the chronic inhalation TRV. As noted previously, the original draft of the cadmium toxicological profile had chosen 0.005 µg/m <sup>3</sup> as the chronic inhalation TRV.	This issue is discussed in A4-3 – non-carcinogenic TRV section.
Appendix A		Toxicological Profiles	Appendix A5-Lead: In the response to previous comments (see Appendix F – Response to Comments – tox profiles), the comment was made that an appropriate short term oral TRV would be selected for lead. In the final draft, however, no oral TRV was included so the response in Appendix F should be revised.	Revision has been made as suggested.
Appendix A		Toxicological Profiles	Appendix A6-Mercury: Explanations were provided in Appendix F – Response to Comments – tox profiles for why only the inhalation exposure pathway for elemental mercury and the oral exposure pathway for methyl mercury were considered. It would be helpful if these explanations were included in the text since they do justify the decisions that were made regarding the choice of pathways.	Revision has been made as suggested.
Appendix C		Supplemental Data	C-1.2 Air Data, p. c-8: The title “Flin Flon Area Quarterly Airy Quality Reports” should read “Flin Flon Area Quarterly Air Quality Reports”.	Revision has been made as suggested.

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<b>Additional Manitoba Conservation Comments</b>				
4		Section 4.1.7.3	The 1995 Health Canada document A Handbook for Exposure Calculations suggests that consumption of home-grown vegetables make up approximately 7% of the total intake of vegetables. Is there a reason why the EPA (1997b) values of 6.2% for aboveground vegetables and 1.8% for below ground vegetables were used instead of the 7% recommended by Health Canada. Is the Health Canada recommendation considered out-dated? Furthermore, why were the EPA consumption amounts from the Northeast region used over those from the West or Midwest Regions?	The information provided in U.S. EPA (1997b) was used in the current assessment because it provided the most robust data set and was seasonally adjusted to account for the fact that lower consumption rates will occur during winter months. The Northeast region was selected over other regions because it is the most northern and was considered to be the closest representation of the growing season of Northern Canada. The Health Canada (1995) document is not available through the Health Canada website and has not been obtained for review. Additional text has been added to the discussion in Section 4.1.7.3 to provide additional justification for the use of the selected values.
4		Section 4.1.9.3	The HHRA document states on p. 4-53 that “the calculated 95% UCLM IVBA for all samples was 69%”. Assuming the HHRA used the numbers in Table 2 of the Bioaccessibility Report, then the mean is actually 69%, and the UCLM would be around 76% (according to UCLM calculations using Excel).	A supplemental report has been included in Appendix G showing the results of the re-analysis of two samples that were originally reported to have lead bioaccessibilities of 122 and 177%. The updated values for these samples were reported to be 56 and 54%, respectively. These updated values were used in the derivation of the 95% UCLM values for use in the HHRA.
4		Section 4.1.9.3	The HHRA on p. 4-53 references USEPA (2007) ...please confirm that this is the correct reference.	The correct reference description for U.S. EPA (2007) has been included in the references section of this chapter.
4		Section 4.2.2	In Table 4-38, under the Chemical column, should “Arsenic” be changed to read “Arsenic - Inorganic”.	Revision has been made as suggested.
4		Section 4.2.3.3	Under the subsection “Copper Health Effects” there is little mention of possible health effects from ingestion of Cu. All effects discussed seem to be related to inhalation exposure. Perhaps a summary of potential health effects from ingestion could be included, particularly since oral exposures are presented in the next subsection on TRVs	Revision has been made as suggested.
5		Section 5.1.1	According to the HHRA Table 5-2, the frequency of 24-hour CRacute exceedences for Pb was 10%. While this is infrequent, it should not considered “rare” as indicated in the text.	The statement has been qualified to indicate that exceedences are somewhat rare.

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5		Section 5.1.2	The HQ for acute soil exposure to toddlers for Cu was above 1.0 in all COIs and was particularly high in West Flin Flon (Table 5-3). With this in mind, it may be prudent to offer a more detailed explanation as to why the elevated levels of Cu in soil do not present a significant health risk even though the HQ values seem to indicate there may be a concern.	Revision has been made as suggested.
5		Section 5.2.1	The y-axis on Figure 5-5 appears to have an error ( <i>i.e.</i> , "5.1E-05"). Please clarify.	This is not an error. Microsoft Excel selected the scale to allow for the minimum and maximum results to be illustrated on a single figure.
5		Section 5.2.4.2	Add BLL (blood lead level) to the list of Abbreviations and Acronyms.	Revision has been made as suggested.
5		Section 5.2.4.2	As indicated in the foot note to Table 5-26, the OMOE SRML for lead was recently changed (OMOE 2008). As such, using the old values (from the OMOE 2001 Fact Sheet: Lead) in Table 5-26 may no longer be relevant.	The OMOE (2001) SRMLs have been removed from Table 5-26.
5		Section 5.2.2	Add RTDI (residual tolerable daily intake) to the list of Abbreviations and Acronyms.	Revision has been made as suggested.
5		Section 5.2.5.1	Add the word "Inorganic" to the title on Figure 5-22.	Revision has been made as suggested.
5		Section 5.2.5.1	Why were dental amalgams not mentioned as a possible source of Hg in the exposure calculations in Chapter 4?	

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Chapter	Page	Section	Comment	Response/Resolution
5		Section 5.2.5.1	<p>The text on the top half of page 5-56 needs clarification. Particularly in reference to the following statements:</p> <p>“Assuming that exposure to inorganic mercury remains constant from all sources other than soil/dust, the RTDI to be allocated to soil/dust <i>via</i> ingestion and dermal exposure is 0.078 µg/kg/day for the toddler. Conservatively assuming 100% bioavailability of inorganic mercury in soil, a soil concentration of 46 µg/g (and an associated indoor dust concentration of 3.0 µg/g) produces an exposure of 0.11 µg/kg/day and results in a total HQ of 1.0 (Table 5-32)”.</p> <p>If the RTDI to be allocated to soil/dust is 0.078 µg/kg/day, then where did the figure of 0.11 µg/kg/day used in the next sentence come from?</p>	This discussion has been revised for clarity.
5		Section 5.2.5.1	<p>The footnote at the bottom of Table 5-32 needs clarification to agree with the previous text and content of the Table</p>	Revision has been made as suggested.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
<b>Lawrence Elliott (MOH, Nor-Man/MB Health)</b>				
			<p><b>General Comments:</b> • Overall and in most sections, the layout and writing are clear and logical; however, there are parts that are uneven in this regard. There are sections where data seems to be presented out of place, for example, in Chapter 2 – Literature Review and Gap Analysis, the Results of the snow sampling conducted for the HHRA are inserted on page 2-19; this seems more logically placed in Chapter 4 and/or 5. The same comment applies to the bioaccessibility data collected specifically for this study, currently presented in Chapter 2. Similarly, new data tables are presented in Ch. 8, Conclusions and Recommendations; I feel that this Chapter should be reserved for narrative summation, discussion, conclusions and recommendations, if necessary referring back to specific tables in previous chapters.</p> <p>(cont.)This final chapter especially needs to be written at a less technical level to the extent possible, as it is where the broader audience will focus. The Appendices should be logically ordered. The tense used should be consistent throughout – there are several places in the introductory chapters where the future tense is used in referral to what will or may be done in the study (pages 1 -13 “may be predicted...” and 2-18 “it is anticipated that this will serve as...”</p> <p>There are other areas where the writing is overly reliant on shorthand symbols, e.g., Ch. 1, page 1-6: “...from the smelter such that Zn&gt;Pb&gt;As&gt;Cu...” Finally, the Report should be consistent in the use of either “COC” or “COPC”.</p>	<p>The report has been revised to consistently use past tense. The use of shorthand symbols has been reduced where appropriate.</p> <p>The report has been revised to consistently use COC.</p>

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
			<ul style="list-style-type: none"> <li>The Report should be clear and consistent on whether Risk Management is part of the scope of the current study or not; in Chapter 1 there are inconsistencies in this regard.</li> </ul>	Chapter one has been revised to consistently indicate that risk management recommendations are a part of the scope of the current study.
			<ul style="list-style-type: none"> <li>Similar to the previous comment, the Report should be clear on the role of potential biomonitoring and its relationship to the scope of the current report; hopefully, our TAC discussions will help here.</li> </ul>	A discussion of the potential role of biomonitoring has been added to Chapter 8.
1 and 2			<ul style="list-style-type: none"> <li>In Chapters 1&amp;2, it was stated that current data for surface water concentrations of COC's was not an important data gap; however, subsequent Chapters refer to the incidental single-sample surface water measurements made in the course of the Fish &amp; Berry Study being used as EPC's for recreational water exposure. Yet these surface water EPC's are not included in Table 4-25 "Summary of Exposure Point Concentrations Used in the HHRA". Please clarify.</li> </ul>	Chapters 1 and 2 have been revised to indicate that additional surface water samples were collected to fill this data gap. Surface water concentrations have been added to the Summary of EPCs Table in Chapter 4.
			<ul style="list-style-type: none"> <li>In Results, the predicted elevated HQ's and ILCR's for arsenic are in contrast to the findings and conclusions of the Sudbury Soil Study for the Falconbridge site, which had similar EPC's in most cases, with a higher soil concentration of arsenic. The current report mentions this discrepancy in Ch. 5 page 5-18, but does not explore why this discrepancy occurred. This should be further explored, perhaps with sensitivity analyses demonstrating how different parameters could have led to this discrepancy in this case. The same goes for other COC's where different conclusions were reached.</li> </ul>	Comparing arsenic concentrations in environmental media in Falconbridge and West Flin Flon, the EPCs for soil (79 µg/g in Falconbridge and 77 µg/g in West Flin Flon) and drinking water (2.6 µg/L in Falconbridge and 3 µg/L in West Flin Flon) are similar. The primary difference between these two communities is the concentration in ambient air. The concentration of arsenic in ambient air in West Flin Flon (0.084 µg/m <sup>3</sup> ) is 35 times higher than the concentration in Falconbridge (0.0024 µg/m <sup>3</sup> ). Given that the risk associated with the inhalation of ambient air accounted for approx. 54% of the total ILCR in West Flin Flon, this has a large influence on the overall findings. In addition, the results of the Sudbury report had the benefit of having the results of the urinary arsenic study to provide an additional line of evidence in the overall assessment of risk. The current Flin Flon HHRA is indicating that there is the potential for unacceptable risks but these conclusions may be significantly revised following the completion of the urinary arsenic study.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
			<ul style="list-style-type: none"> <li>The two references to the MB/SK Community Health Status Assessment on page 8-5 in the context of specific discussions and recommendations regarding arsenic and cadmium, respectively, both seem out of context. For example, current lung cancer incidence is more reflective of exposures at least 20 years ago rather than current risk. Reference to the Community Health Status Report, if made, might best be kept to a general discussion of how that study provided some reassuring evidence about the overall mortality and cancer incidence of the studied communities, in comparison to provincial averages.</li> </ul>	<p>Additional discussion has been added to recognize the limitations of the community health status assessment.</p>
			<ul style="list-style-type: none"> <li>I feel that the analyses presented in the Sensitivity Analysis section are very helpful, and that this section should be expanded significantly and should explore the impact of varying parameters in both directions, where relevant. For example, it could explore the impact of a range of higher and lower relative bioaccessibilities, as well as the impacts of different assumptions in estimating EPC's. In some cases, it seemed like a series of very conservative assumptions made in sequence may have made a significant over-estimate of risk probable.</li> </ul>	<p>Additional discussion has been added to the sensitivity analysis illustrating assumptions that may have resulted in both an over- and under-estimation of risks.</p>
			<ul style="list-style-type: none"> <li>The discussion of deterministic vs. probabilistic analyses which was presented in Ch 4.1.6 should be reiterated and expanded in Ch 7 Limitations and Uncertainties, and referred to in the Conclusions. Some worked examples of applying a probabilistic approach to scenarios where elevated risks were estimated using the deterministic approach would be very helpful in further demonstrating and interpreting the inherent uncertainties.</li> </ul>	<p>Although it is agreed that the application of a probabilistic assessment may help to illustrate the effect of variability on the overall risk estimates, this approach often results in greater confusion and mis-interpretation of results. The upper and lower limits of a probabilistic assessment represent such extreme conditions that they tend to create a greater degree of uncertainty in the risk estimates. In addition, given that many of the parameters used in the assessment are point estimate values recommended by agencies such as Health Canada, there are limited parameters for which distributions can be used within a probabilistic assessment. Overall, use of conservative yet realistic point estimate values has been found to provide appropriate risk estimates that can be understood by an</p>

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
				audience with a varying level of risk assessment experience.
1	Page 1-1		“(TAC) was been...”	Correction has been made.
1	Page 1-8		“...is be...”	Correction has been made.
1	Page 1-9		“...wasd be...” in both 4th and 5th paragraphs.	Correction has been made.
1	Page 1-10		“...the Manitoba Conservation (2006) ___ is anticipated to...”	Correction has been made.
1	Page 1-11		“...histroically...”	Correction has been made.
1	Page 1-15		“...Health and Safety Committee that are...”	Correction has been made.
3 and 4	Page 3-2 and 4-3:		“predominate” is a verb; “predominant” is the spelling of the adjective.	Correction has been made.
3	Page 3-14		“...identified as being of COC.”	Correction has been made.
4	Page 4 -67		should be “papular erythematous rash” not “popular” (Spellcheck caused this one!)	Correction has been made.
4	Page 4-76		“...residential uses. Particularly...” (should be comma, not period, between these.	Correction has been made.
<b>Saskatchewan Ministry of Environment - submitted March 3/2009</b>				
1		Chapter 1/ Page1-1/1.0	Second paragraph mentions “eleven metals were elevated” but on Page 1-7 second paragraph, it mentions “twelve chemicals”, first example is missing sulfur	The discussion on page 1-1 has been revised to indicate that “...concentrations of sulphur and the following eleven metals were elevated...” to be consistent with the discussion on page 1-7.
1		Chapter 1/1.5/Page 1-9	Typo – Fourth paragraph, “wasd”	This error has been corrected.
1		Chapter 1/1.5/Page 1.11	Typo – Third paragraph, “Histroically”	This error has been corrected.
2		Chapter 1/2.2.1/Page 2-6	Why might using a smaller filter sample size result in increased concentrations?	Smaller samples may result in greater uncertainty during laboratory analysis and the use of elevated detection limits. This will not affect chemicals for which there are sufficiently high concentrations, but for those that are found at low concentrations, the analysis may not be able to reach the preferred level of uncertainty or minimum detection limit.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
4		Chapter 4/4.1.1.5/Page 4-10	The garden soil data, how wide spread were the gardens? How representative are they of the different parts of Flin Flon in the HHRA?	A Figure has been added to this section displaying the locations of the home garden sampling plots included within the Manitoba Conservation study. Given that the maximum concentrations of COC in residential soil were generally located within the community of West Flin Flon, and significantly lower concentrations were found in East Flin Flon, this data set was considered to provide an accurate representation of the range of concentrations within the study area.
4		Chapter 4/4.1.17/Page 4-13	Only one indoor dust sample was taken in Channing, does this pose a problem in doing calculations now?	While it would be preferred to have multiple sample locations in Channing, arrangements could only be made to sample one location. Data from each of the four COI were pooled to derive regression equations relating outdoor soil to indoor dust to be used for each community. Given that concentrations of COC in Channing soil were significantly lower than in other COI, it is not anticipated that this deficiency will result in the under-estimation of exposure and risk to residents of Channing.
4		Chapter 4/4.1.1.8/Page 4-16	How did you come up with the cadmium figures in grouse and mallard, 0.30 and 0.32 respectively? (Table 4-15) Why so much higher than deer and moose?	Concentrations of COC in wild game are based on measured concentrations in forest soils, surface water, and sediments as well as literature-based bioconcentration factors and regression equations to predict uptake into food items. Literature-based information indicates that benthic and soil invertebrates can accumulate significant concentrations of cadmium. Since a large portion of the diet of the mallard and grouse is invertebrates, exposure to cadmium is much greater for these animals than for wildlife that do not consume benthic or soil invertebrates.
4		Chapter 4/4.1.1.10/Page 4-19	How do recent water samples from Manitoba effect calculations, as they are lower?	Given that exposures to COC via incidental ingestion and direct contact with surface water while swimming are several orders of magnitude lower than many other exposure pathways, the use of the lower surface water concentrations collected by Manitoba Conservation in the HHRA will not result in noticeable changes to the overall exposure and risk calculations.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
4		Chapter 4/4.1.2.1/Page 4-22	Table 4-21 Your copper figures have a 95% UCLM which is higher than the Max., 40.8 <i>versus</i> 0.00126. There is a mistake somewhere.	The min, max and average columns from this table have been removed. They will be added to the final report once the correct values can be confirmed.
4		Chapter 4/4.1.2.1/Page 4-26	Cadmium, you mention raw data was not “obtained/not available.” Please clarify, it was not available for some reason or you just didn’t get it as it wasn’t important?	The raw data for cadmium was not available, and given the limited amount of information and the time restrictions of the current project, it was determined to be more appropriate to utilize the recommended TDI from CCME rather than pursuing efforts to try to obtain the original data. This discussion has been modified in the current report.
4		Chapter 4/4.1.2.1/Page 4-27	Copper, second paragraph, explain what is meant by “because they fulfilled all of the selection criteria and were most appropriate for copper.” Why was Port Colboure data which had some higher numbers (21,935 and 685) not used and why did you say the numbers were lower at the beginning of the paragraph?	Within section 4.1.3, a set of criteria are listed for the selection of an appropriate database for the derivation of an EDI. The CTDS data met each of these six criteria. While a number of the food categories had lower average concentrations in the Port Colborne study, there were categories that had higher concentrations than found in other studies. The discussion has been revised to reflect this. Port Colborne data was not included in the derivation of the EDI since there was sufficient data for copper provided in the CTDS study and only average concentrations per food category were provided for the Port Colborne data as opposed to all of the raw data.
4		Chapter 4/4.1.6/Page 4-31	It discusses using deterministic analyses <i>versus</i> probabilistic analysis and using the latter if elevated risks are found under a reasonable maximum exposure scenario, please explain this.	This discussion is meant to indicate that the current assessment uses exposure parameters and assumptions to represent a reasonable estimate of a maximum exposure scenario without being unrealistically conservative. If this approach results in the prediction of unacceptable risks, then a probabilistic assessment may be considered to help indicate the probability or likelihood of the occurrence of unacceptable risks.
4		Chapter 4/4.1.7.2/Page 4-38	Do we always assume a 10-hour workday?	It is common to assume a typical workday will last between 8 and 10 hours. Given that the commercial worker scenario evaluated in the current assessment was intended to represent an outdoor laborer, it was considered to be conservative yet realistic to assume that an adult would spend 10 hours per day working outdoors.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
5		Chapter 5/5.1.1/Page 5-6	Table 5-2, what is "7 of 210", explain where this comes from?	This indicates that 7 of the 210 available air measurements are above the 24-hour acute TRV. Clarification has been added to this section.
5		Chapter 5/5.1.1/Page 5-7	Table 5-3, relating to the table on discussion below it, the report uses words like "marginal exceedance", "magnitude of exceedances are small", what would be the number that would raise concern?	Generally, Hazard Quotients (HQs) or Concentration Ratios (CRs) that are less than 1.0 are considered to be indicative that no risks are anticipated, and values between 1.0 and 10.0 are considered to be minor or marginal exceedances. Values greater than 10 are considered to be notable and generally require further consideration.
5		Chapter 5/5.2.1/Page 5.8	Need to reassess with new Creighton water limits.	The assessment of exposure to arsenic for residents of Creighton have been revised to reflect the corrected drinking water concentration.
5		Chapter 5/5.2.1/Page 5-12	Figure 5-4, does this table mean, for a toddler in the COI, the arsenic in drinking water, not counting Creighton's and the market basket, give a HQ above 1?	Yes, the HQs associated with the consumption of market basket foods is approximately 0.82 and with the consumption of drinking water in Flin Flon is 0.36 to produce a combined HQ of 1.2.
5		Chapter 5/5.2.2/Page 5-14	Table 5-14, explain this table, not sure how number arrived at.	The exposure and risk estimates in Table 5-14 represent the values predicted within the HHRA for a toddler living in West Flin Flon. Since the tolerable daily intake (TDI) for cadmium is 1 µg/kg/day, the predicted daily exposures from market basket foods, local foods, drinking water, and air are subtracted from the TDI to produce the residual TDI (RTDI) that can be allocated to exposure from soil/dust while still resulting in a total exposure below the TDI. Additional text has been added to this discussion to provide clarification.
5		Chapter 5/5.2.4.2/Page 5-49	Possible typo, third paragraph, third line says "375 µg/dL", should it be 375 µg/g?	This error has been corrected.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
5		Chapter 5/5.2.5.1/Page 5-51	Local wild game is said to contribute 57% to 59% of inorganic mercury derived from calculations. This figure seems high, what is driving it?	Since concentrations of inorganic mercury are low in COI other than West Flin Flon, the overall exposure is relatively low. Since the concentrations in wild game were predicted based on a number of conservative assumptions regarding the bioconcentration of mercury and the area in which wild game will live, exposure <i>via</i> consumption of local wild game is anticipated to be overpredicted. This, combined with the fact that other sources of mercury exposure are relatively minor, results in a large portion of the total exposure for residents of East Flin Flon, Creighton, and Channing to be attributed to wild game consumption ( <i>i.e.</i> , 57 to 59%).
8		Chapter 8/8.1.2/Page 8-3	Below table 8-5 it says "The chronic residential results are discussed further in Section 8.1.2". This is the section, what is this referring to?	This has been corrected to indicate that the chronic residential results are discussed further in Section <u>8.2</u> .
appendix		Wild game	What geographic area are we talking about to get the calculation?	Wild game were assumed to live within a 15 km radius of the HBMS complex.
		Add Acronyms	ECF	Revision has been made as suggested.
			GSD	Revision has been made as suggested.
			CTF	The acronym is not found in the document.
			IOM	Revision has been made as suggested.
			CRL	Revision has been made as suggested.
			EU	Revision has been made as suggested.
			PbB	Revision has been made as suggested.
			LADD	Revision has been made as suggested.
			CFLP	This was not added to the list, as it is not an acronym but a type of mouse used in toxicity testing.
			PTWT	The acronym was not found in the document.
			ADAF	Revision has been made as suggested.
			MSA	Revision has been made as suggested.
			PRA	Revision has been made as suggested.
			RBA	Revision has been made as suggested.
			ABA	Revision has been made as suggested.
			IUBA	The acronym was not found in the document.
			NOVEL	corrected, not an acronym
			PAH's	Revision has been made as suggested.
			HI	Revision has been made as suggested.

Human Health Risk Assessment of Flin Flon, Manitoba, and Creighton, Saskatchewan: Draft Report (January 2009), Comments and Responses				
Chapter	Page	Section	Comment	Response/Resolution
			WOE	Revision has been made as suggested.
			BINWOE	Revision has been made as suggested.
			TTD	Revision has been made as suggested.
			TCDD	Revision has been made as suggested.
			CI	Revision has been made as suggested.
			AWRA's	Revision has been made as suggested.
			SSRA's	Revision has been made as suggested.

## Preliminary General Comments: HHRA of Flin Flon and Creighton From Population Health Unit (MCRHR); February 23, 2009

This is a general summary of the comments from our review of the draft report. It includes comments on content, as well as editorial (for clarity) and identification of some typographical errors. Generally, this is a well written, comprehensive document but would greatly benefit from an executive summary (even if this is following the peer review but would need to be seen by TAC prior to final report). Additional information is required on the objectives of the biomonitoring survey and how this fits generally into the overall HHRA and risk management. Some alteration is required in the document to have similar tense throughout (is, was, will, etc.)

There were two areas in which we have questioned the results of some of the monitoring data. One was mentioned in the email already and was discussed at the last TAC meeting (*i.e.*, Creighton-2 arsenic water sample value). The other area that should be double checked is the Creighton annual average metal concentrations associated with PM10 in ambient area for lead (Table 2-2). This is much greater than previous reports. There are implications for these two issues in various parts of the document.

<b>Section</b>	<b>Comment</b>	<b>Response</b>
<b>Table of Contents</b>		
Page 5: Abbreviations	Add: CRL; RTDI; UCLM; IPCS; ISS	Revision has been made as suggested, with the exception of ISS.
General comment on all the Tables of Contents – to include more headings to make it easier for reviewers		
<b>CHAPTER ONE</b>		
1-3: P2 – sentence ‘...available governing area-wide of risk assessment in Canada.’	Check wording of this sentence - Remove ‘of’ in the sentence?	Revision has been made as suggested.
1-7: Fig 1-3 is blurry.	Increase size of figure to improve readability?	Unfortunately due to the quality of the Figure originally provided in the Manitoba Conservation (2007) report, enlarging this Figure decreases its clarity. The quality of the Figure cannot be improved by Intrinsic.
1-8: Title “Soil Sampling & Analysis”	Should be changed to “Soil & Indoor Dust Sampling & Analysis”	Revision has been made as suggested.
1-9: under bullet – Outdoor Soil:	typo “wasd” needs to be addressed	This error has been corrected.
1-11: under bullet – Local Fish:	typo “Histroically” needs to be addressed	This error has been corrected.
1-11: P3 – sentence ‘...address uncertainties, the local food...in early 2008, gathered information...’	Remove commas after ‘uncertainties’ and ‘2008’.	This sentence has been revised.
1-12: under bullet – Drinking Water: ‘...of a Manitoba Conservation sampling program...’	Clarify that the water samples from Manitoba Conservation are for Flin Flon drinking water.	Revision has been made as suggested.
1-12: under second bullet: ‘...arrangements were made to sample water from a residential location...’	Clarify how long the water was sampled from these locations.	This discussion has been revised to provide more details of the drinking water sampling programs completed by HBMS and JW.

<b>Section</b>	<b>Comment</b>	<b>Response</b>
1-13: under second bullet: 'use of alternate values... outdoor air may be considered...'	Should state what was done, not what may be done as it already has been done.	Revision has been made as suggested.
1-13: under second bullet: 'concentrations in indoor air may be predicted...'	Should state what was done, not what may be done as it already has been done.	Revision has been made as suggested.
1-13: under third bullet: '...reported in three main studies...'	Please reference these studies after this sentence.	This discussion has been revised to provide a better description of the data used to assess exposure from direct contact with surface water.
1-13: last sentence	Reword for clarity	Revision has been made as suggested.
1-14: first bullet: '...of the COPCs will be compiled and incorporated...'	Should read '...were compiled and incorporated...'	Revision has been made as suggested.
1-14: under <i>Additional Studies</i> 'Once the HHRA is complete..'	Consider that the HHRA is complete upon completion of this document? Reword?	The additional studies section has been removed from the revised report. This discussion was originally included prior to the completion of the HHRA. Recommendations for additional studies (i.e., biomonitoring) are included in Chapters 5 and 8.
1-16: Typo in last sentence of the second last paragraph	"a residential locations"	This error has been corrected.
1-17: in Table 1-1 under Data Gaps for Surface Water typo: '...for recreational activities...'	Reword to '...for recreational activities...'	This error has been corrected.
1-18: P4 – refers to Stakeholder Consultation	Clarify that stakeholder consultation did occur for this assessment or refer reader to section about stakeholder consultation.	Revision has been made as suggested.
<b>CHAPTER TWO</b>		
2-4: Title 2.1.3 Supplemental Soil Sampling Program	Could read Supplemental Soil and Indoor Dust Sampling Program	Revision has been made as suggested.
2-4: P5	No mention of frequency of vacuuming or dusting in home prior to testing (as background info from household survey)	Participants were requested not to vacuum two weeks prior to the sampling program. This has been added to the revised report.
2-6: P1 – '... will likely provide the most useful data.'	Should review and state what provided the most useful data as this should have already been done.	This comment was removed from the discussion since it is not necessary to include as part of a data review.
2-6: P2 – 'According to HBMS...10 cm filter'	Reword for clarity.	Revision has been made as suggested.

Section	Comment	Response
2-6: Lead in Table 2-2	Why is Pb so much higher in Creighton than Ruth Betts whereas other COC are generally lower? This should be double check as the recorded values from previous data files received from Manitoba Conservation for Creighton Pb levels which showed much lower levels.	These results are confirmed to be correct based on data provided by HBMS. As provided in the discussion below Table 2-2, during the sampling period from 2003 to 2006, a dichotomous air sampler was used for the Creighton School location. Personal communications with HBMS indicated that reported concentrations for metals associated with the PM10 component significantly increased when this sampler was used relative to periods in which it was not. It should be noted that for the 2007-2008 sampling period, the dichotomous air sampler was not used at the Creighton School and results were more consistent with previous data. The data collected during 2007-2008 was used in the HHRA.
2.6: P2 – “Annual average concentrations of lead , copper, and zinc from the Creighton school are notably higher”	From Table 2-2 only Pb is notably higher (Cu and Zn are less than Ruth Betts) Cd slightly higher.	This discussion has been revised to indicate that only concentrations of lead were higher from the Creighton School location relative to Ruth Betts.
2-6: Under Data Gap Analysis	States that HBMS increased measurements for air quality to include certain parameters that weren't measured in the past: a reader may not understand why Manitoba Conservation was not approached to measure PM10 & 11 metals. Explain why PM10 concentrations weren't added to the MB Conservation monitoring.	The limitations of the data collected by Manitoba Conservation were highlighted in a number of reports prepared during the initial stages of the HHRA which were reviewed by the TAC. Manitoba Conservation made no indications that their current sampling program could be revised to include the collection of PM10 samples and the analysis of additional chemicals.
2-9: 2.3.2 Data Gap Analysis	Clarify that no additional produce data was collected for this HHRA. Also last sentence needs rewording.	Revision has been made as suggested.
2-9: P4 beginning with “The most: ‘...available, likely collected as...’	Should be reviewed & reworded as a fact not as speculation	Revision has been made as suggested.
2-9: P4 General comment:	With the present document (in retrospect rather than prospective), need to say this data wasn't used but more sampling was done.	Revision has been made as suggested.
2-10: P2- half way through “ two locations in Creighton”	With the misinterpretation of the HBMS sampling (See comment on Page 4-8 Table 4-6), there was only one location in Creighton sampled.	Revision has been made as suggested.
2-10: P3 -	To help with flow of the document, perhaps useful to refer reader to where results are later found in document (Ch 4) and methodology (Appendix). Could clarify that reference JW 2008 is also in Appendix.	Please refer to section 2.4.3. Additional references to the location of data were included where appropriate throughout the revised report.
2-11: Table 2-6	Under “Group” in Table 2-6 should state either “Flin Flon Area” or “Flin Flon / Creighton” as some of those lakes are in proximity to Flin Flon but on SK side.	Revision has been made as suggested.

<b>Section</b>	<b>Comment</b>	<b>Response</b>
2-18: P1 '...blueberries, it is anticipated...'	Avoid use of 'it is anticipated' and state what happened. Should this be "was" anticipated?	Revision has been made as suggested.
2-18: P3 – '... wild blue berries...'	Should be one word – '... wild blueberries...' Typo	Revision has been made as suggested.
2-18: P3 – 'Details of this study, and its results...'	Comma should be removed – 'Details of this study and its results...'	Revision has been made as suggested.
2-19: P1 – Snow data	Indicate why one snow sample only was collected at each site <i>versus</i> replicates.	The methodology for the snow sampling study was prepared by JW and HBMS with input from Manitoba Conservation. Given that snow ingestion was not considered to be a significant exposure pathway, Intrinsic does not have concerns regarding the absence of replicate samples.
2-19: P3 – Bioavailability/Bioaccessibility	Could bioavailability and bioaccessibility be defined here to help clarify for the reader what each term means as this is the first time that these two terms are used in the document?	The definition of bioavailability has been added to Section 2.9. Bioaccessibility is defined within the first sentence of Section 2.9.1.
2-25: P2 – '...the bioaccessibility of lead, and arsenic...'	Remove comma – '...the bioaccessibility of lead and arsenic...'	Revision has been made as suggested.
2-32: Under 2.10.1 Fish – 'Information concerning the quantity of fish consumed by the survey respondents is not available.'	Under 2.10 Flin Flon and Creighton Food Survey Summary it states that 'the purpose of the survey was to gather information... the type, and the quantity of the food item consumed.' However, looking at the survey questionnaire (Appendix P), it does not ask the respondent about serving sizes. This should be clarified in the report, why was this not part of the food survey when the report said it was part of the information collected. Rather than use "quantity" of fish should use "frequency of consumption" as the questionnaire had no question on serving size.	This discussion has been revised to indicate that residents were asked to indicate the <u>frequency</u> in which local food items were consumed. The questionnaire did not ask residents to indicate the serving sizes of local foods as it is often very difficult to estimate the mass of food items such as fish fillets, wild berries, or wild game servings. There is significant room for error in this estimation and data can often be highly skewed. As a result, a generic meat serving of 8 oz. is recommended by a number of sources for predicting exposure from the consumption of impacted meats.
<b>CHAPTER THREE</b>		
3-2: P4: '...separate communities Of interest...'	Change Of to of: '...separate communities of interest...'	Revision has been made as suggested.
3-3: Figure 3-1	Fig. 3-1 is blurry, can this be refined?	This figure has been replaced with one with greater resolution.
3-10 and 3-11 Discrepancy with Table 3-4 and text following	In table, thallium is 0.46% (3 of 652), zinc is 0.31% (2 of 652), chromium is 0.31% (2 of 652) but in paragraph following table, states thallium have four samples for 0.6% and zinc and chromium had three samples for 0.46%.	The information provided in Table 3-4 is correct. The discussion following this table has been revised to be consistent.
3-24 Graphic	Could mention in text that Surface Water pathway also included Dermal Contact and Snow was considered as "Ingestion" (with air deposition to snow)	Discussion has been added to the "Additional Recreational Pathways" section.

Section	Comment	Response
<b>CHAPTER FOUR</b>		
Chapter 4 – Table of Contents	Include more bullets ( <i>i.e.</i> , 4.1.1.1) to help reader find sections.	
Chapter 4	No section exists on snow data in this chapter. It may be more appropriate to remove the 95% UCLM data in Chapter 2 (Page 2-19 Table 2-15) and put in Chapter 4 where 95% UCLM is defined and discussed.	Revision has been made as suggested.
For Chapter 4 it would be good to have a better explanation for EPC, STC, PRG early on. It is fully explained in various parts of Chapter 4 and 5 but would be better to have an explanation upfront.		EPC is defined in Section 4.1.1. A brief description of PRGs and STCs have been added to Section 4.0.
4-3: P2 – ‘...chemical-specific regression equations based on historical paired TSP and PM <sub>10</sub> data were used to predict concentrations of COC associated with the respirable PM <sub>10</sub> fraction.’	Suggest adding where the historical data was from: <i>e.g.</i> , chemical-specific regression equations based on historical paired TSP and PM <sub>10</sub> from the <u>Provincial Building</u> were used to predict Ar, Cd, Cu and Pb and <u>Ruth Betts and Creighton</u> for Hg and Se.	Revision has been made as suggested.
4-3: P3 – typo ‘The community of east Flin Flin...’	Change ‘Flin Flin’ to ‘Flin Flon’ TYPO	This error has been corrected.
4-4: P1 – move whole paragraph to 4-3: P2	More appropriate – seems it would improve readability to introduce current air monitoring measurements being done at beginning of section. Also the paragraphs preceding 4-4: P1 talks about current air monitoring measurements so air monitoring measurements should be introduced first. Should this be added in Chapter 7 as an uncertainty factor?	This section has been revised to improve clarity.
4-4: P3	This whole section should be reviewed; it appears to be fragmented and is hard to follow. Perhaps a new section should be created regarding the regression equations ( <i>i.e.</i> , 4-4: P3 – new section 4.1.1.2a – Development of Regression Equations)	This section has been revised to improve clarity.
<u>WATER DATA</u> Page 4-6 Drinking water re: the 2 sampling points for Creighton by HBMS	Readjust to state only one sampling location in Creighton.	Revision has been made as suggested.
4-6:	Move 4.1.1.7 Indoor Dust Concentrations to after 4.1.1.1 Surface Soil Concentrations. For clarity and flow of reading.	This revision will be considered depending on time constraints for the completion of the revised report.
4-6: P4 – drinking water was collected for three locations in Flin Flon and two locations in Creighton.	Where exactly was the water samples collected – at residential homes? <u>Also this is now one site in Creighton.</u>	Descriptions of the sampling locations have been included in this discussion.
4-7: P2 – ‘JW’ should be ‘Jaques Whitford’ at the beginning of the paragraph.	Acronyms shouldn’t be used at the beginning of a paragraph. Clarity and flow of document.	Revision has been made as suggested.

Section	Comment	Response
<p><b>Page 4-8: Table 4-6</b>  <b><u>(THIS IS FROM THE EMAIL SENT PREVIOUSLY)</u></b>                      (1) In regards to the sampling of the arsenic levels in the Creighton drinking water I have a query regarding the sample site for the Creighton-2 results.</p> <p>The arsenic levels taken from Creighton-2 sampling site had a mean of 9.8 and a max of 12 ug/L with a 95 UCLM of 10 ug/L. (thus the mean is nearly at the MAC or maximum acceptable concentration level).</p> <p>The interesting fact is all the other sample sites have significantly lower levels (all the same drinking water supply):                      So:</p> <ol style="list-style-type: none"> <li>1) Creighton-1 sample mean is 1.9 (max is 2.4);</li> <li>2) JW Sampling at 11 other sites: mean was 2.0 with max of 2.7; and</li> <li>3) The levels taken by the community (as required by Sk Ministry of Environ) on Table 2-5 were 2.2 for 2005 and 1.4 for 2006.</li> </ol>	<p>Thus one site has a mean arsenic level 4 to 5 times higher than all the other sites. Is there something about this site that we should be aware of? Is this site specific or something to do with the water supply? This makes quite a difference in terms of public health advise re: water supply but also to the HHRA calculation and areas for biomonitoring</p> <p>Alan and Elliot – can you let George and I know where this site is and what building it is? I'm not aware of anything within a specific site that would elevate that site's arsenic level (as you could have with plumbing for lead or copper) but I think we should check this out (? Is it a HBMS building on site, confirm that it is on CR municipal water system, is there a cistern?).</p> <p><b>According to HBMS the site that was giving the high Arsenic levels was actually a raw water source, it was a sampling error. This information will be forwarded to Intrinsic and the new calculations will reflect this.</b></p> <p>This has ramifications in various parts of the document.</p>	<p>The report has been revised to reflect the exclusion of the untreated water samples.</p>
<p>4-10: P3 – ‘...the derivation of EPCs were based on data for fish collected from all lakes included in the sampling program to be representative of an overall community-based estimate.’</p>	<p>The results from the food survey were not considered? <i>i.e.,/</i> types of fish that family consumes (<i>i.e.,/</i> 2-33 – pickerel caught more than pike, trout or perch). Adjust data based on fish consumption? <i>i.e.,/</i> pickerel – 3 samples @ 5, 10, 15 µg/g jack – 10 samples @ 20, 25, 30 µg/g Jack is disproportionate in the community-based estimate since more pickerel is consumed (based on survey)</p> <p>Clarity is required for how the 95% UCLM for the EPC for fish was determined (<i>e.g.,</i> now for Pb the Stantec document uses wet weight with many values below method detection limit, for EPC uses wet weight, and ProUCL uses dry weight) – this makes it difficult to work through analysis. For example hard to compare Pb EPC of 0.0067 µg/g ww (Table 4-8) with the values in Table 3-7 (Appendix E) and the calculations on Page N-89 in Appendix N.</p>	<p>The EPC calculations have been revised to reflect that the fish tissue concentrations reported by Stantec are in units of wet weight.</p> <p>Additional discussion has been added to Chapter 4 in which concentrations of COC in each individual fish species are presented for comparison to the concentrations for all species grouped together. Since the local food survey indicated that walleye is the most commonly consumed species, the EPC fish concentration is the higher of the 95% UCLMs calculated for all fish species or walleye.</p>

Section	Comment	Response
4-10: P3	Insert a reference to refer the reader to Appendix E for data on fish tissue concentrations.	Revision has been made as suggested.
p.4-11/4-12 "For samples where the Concentration of MeHg exceeded the total Hg the fraction was assumed to be equalled to 1.0" (i.e., from bottom of Table 4-10 the sample was assumed to be 100% MeHg" p. 4-12)	Check with MB C and Sk M of E for this	This is considered to be a conservative assumption. No revisions are required.
4-11: Table 4-8 – Exposure Point Concentrations for COC in Fish	<p>Total mercury 95% UCLM = 0.074 µg/g whereas Appendix N (N-10) mercury (all fish) UCL = 0.296. Shouldn't they be the same? More explanation required as to how the data was used. Was all the survey info on types of fish and which lakes just used as background info with the overall combined fish and lake UCLM used in the calculation?</p> <p>Explanation required re: use of survey data (most common lakes fished, and most common fish consumed), the specifics for fish testing and how incorporated in the model.</p>	Additional discussion has been added to this section.
4-11: Table 4-9	Discrepancy? Denare Beach range of mercury in fish 0.23-0.91 µg/g from App. E – 3.17 but the 95% UCLM = 0.15 µg/g (Table 4-9). Shouldn't the UCLM be within the range?	This table has been revised to reflect the understanding that fish tissue concentrations were provided on a wet weight basis (it was originally reported that data was for dry weight which involved converting the data to wet weight).
4-11: P1	Make a reference to Appendix N (ProUCL Results)	Reference to Appendix N was not added to this paragraph. In the second paragraph of Section 4.1.1 (page 4-2) there is a universal statement referring readers to Appendix N for all ProUCL outputs (i.e., "ProUCL output summaries for each COC in all environmental media are provided in Appendix N").
4-11: P2 – 'For samples where the concentration of methyl mercury exceeded the total mercury concentration, the fraction was assumed to be equal to 1.0'	<p>Could it be explained here how methylmercury could exceed total mercury, and why the fraction was assumed to be 1.0? References?</p> <p>Refer to SK and MB environment staff</p>	Additional discussion has been added to this section indicating that this occurrence is assumed to be the result of standard laboratory error and variability in analysis.
4-11: P2 – '...in the form of methyl mercury (0.79) was applied...'	Reword sentence to read '... in the form of methyl mercury was calculated to be 0.79 and was applied...'	Revision has been made as suggested.
4-12: Table 4-10 – "The methyl mercury concentration exceeded the total mercury concentration..."	Again, please explain somewhere how methyl mercury concentrations could be greater than total mercury concentrations.	An explanation has been added to this discussion.
4-14: P2 – 'IEUBK'	This is first part of document that IEUBK used. Either define / explain 'IEUBK' model or refer reader to Chapter 5. Typo	A definition of IEUBK has been added.
4-14: P2 – '...was used to predict concentrations of lead	Add '...was used to predict concentrations of lead in indoor dust <b>based on outdoor</b>	Revision has been made as suggested.

Section	Comment	Response
in indoor dust.'	<b>soil and outdoor air lead concentrations.'</b> To help clarify to the reader how this works.	
4-14: P2 – 'It should be noted that the indoor dust concentration predicted using the MSA module and the EPCs for outdoor soil and outdoor air is similar to the measured EPC for indoor dust.' It then refers to West Flin Flon – MSA approach predicted 289 ug/m <sup>3</sup> and measured 265 ug/m <sup>3</sup> .	Difference between: East Flin Flon: Measured = 320 µg/m <sup>3</sup> MSA Approach = 122 µg/m <sup>3</sup> Creighton: Measured = 264 µg/m <sup>3</sup> MSA Approach = 178 µg/m <sup>3</sup>  Conclusion that the measured and predicted approaches are similar does not appear to be correct based on above values.	The report has been revised to use the 95% UCLM measured concentrations of lead indoor dust as the EPCs. However, given that the MSA module accurately predicted indoor dust concentrations for West Flin Flon, this relationship was still used to derive the STC which is based on the exposure and risk calculation for West Flin Flon.
p. 4-14 and 4-15:	For clarity and to help follow the reasoning <i>etc.</i> of the discussion on page 4-14 can Table 4-12 have something added to it like "Predicted Concentrations of COC...Equations <i>using the IEUBK MSA Module</i> ".	Revision has been made as suggested.
<u>Wild Game Tissue Concentration</u> p. 4-16	For clarity, up front say that it is predicted tissue concentration rather than measured.	This is indicated in the second sentence of this section.
4-16: P1 – 'Based on the comparison provided in Table 4-13, the measured and predicted indoor dust EPCs are generally similar.'	See above comment – is not true for two lead estimations. Reword sentence to read: 'Based on the comparison provided in Table 4-13, the measured and predicted indoor dust EPCs are generally similar for arsenic, cadmium, copper, mercury and selenium.' & "somewhat similar" for lead?	The HHRA has been revised to use the measured indoor dust lead concentrations instead of the concentrations predicted using the MSA module. This discussion has been revised.
4-16: P2	Add sentence to end of paragraph: 'Appendix O outlines the methods used to estimate wild game tissue concentrations.' Placing this sentence here rather than at the end of the section seems more appropriate so the reader can reference Appendix O instead of wondering how the concentrations were determined.	Revision has been made as suggested.
4-17: P1	Remove final sentence 'A detailed description of the... Appendix O.' See above comment.	Revision has been made as suggested.
4-18: Figure 4-2	This figure is slightly blurry, could this be improved?	Effort has been made to improve the clarity of this figure.
4-21: P2 – selection of Cranberry Portage and The Pas for background soil concentrations.	What about naturally occurring metals in soils around Flin Flon/Creighton? A comment that it is not possible to know the 'background' levels for FF/Cr <i>versus</i> the background plus contamination.	Revision has been made as suggested.
4-21: P3 – 'Background drinking water for Manitoba or Saskatchewan was not identified.'	SaskH2O is a source of data for drinking water in Saskatchewan and Manitoba would likely have some data. It would be good to comment on why Ontario data used and a comment on what potential difference there would be from Ont and MB/SK.	Despite efforts by members of the TAC, background drinking water concentrations could not be obtained for Manitoba or Saskatchewan. The report could not be revised, however, it is not anticipated that concentrations of COC in municipally supplied drinking water are significantly different from province to province.

Section	Comment	Response
Market Basket Estimated Daily Intakes p. 4-23 lays out the criteria for determining the most appropriate data to use in the FF HHRA	Question – curious was there any foods that were found to be more “popular” in the local area – for example out east salmon, or lobster might be purchased more often than other store meat sources? Could be included as an uncertainty	The local food survey did not include questions regarding the consumption of market basket food items. It was assumed that the diet of Flin Flon-area residents would be similar to the Canadian averages.
4-26: Title typo ‘Cadmuim’	Should read ‘ Cadmium’	This error has been corrected.
Page 4-29: Table 4-25: Creighton Drinking Water µg/L EPC needs to be changed from 10 to corrected value.		Revision has been made as suggested.
p.4-41 serving size of fish used was 227 g (8 ounces) as recommended by the Great Lakes Sport Fish Consumption Advisory Task Force	Can we reference a local consumption rate; have a MB or SK reference. For example Table 1, page 4 of the Saskatchewan Document “Mercury in Saskatchewan Fish: Guidelines for Consumption Updated to 2004” also uses 8 ounces...and it appears maybe to the locals as more applicable. Also Health Canada’s Food Guide recommends that an adult male serving size to be 75 g and a male could eat 3 servings per day for a total of 225 g. Good to use more regional based reference or national based unless the Ontario source of info had the original determination of the size of serving (the Great Lakes document mainly says they picked that number because it is the general number used).	Revision has been made as suggested.
Page 4-52: Table 4-33: Why such a large discrepancy in the model assumptions for ventilation rates (for some ages it is almost a 3 fold difference)?		This is largely due to the fact that the IEUBK model has derived specific ventilation rates for 1 year age classes, whereas the Health Canada values are for an infant (0 to 6 months), toddler (6 months to 4 years) and a child (5 to 11 years). Since the Health Canada values span a large age group, it is likely that they will over-predict ventilation rates for the younger portion of the age group. For example, the Health Canada ventilation rate for a toddler (9.3 m <sup>3</sup> /day) would be compared to the ventilation rates for IEUBK age groups for 1-2, 2-3, and 3-4 year age classes which is likely a significant overestimation for the 1-2 and 2-3 age groups. The U.S. EPA indicates that the ventilation rates are mid-range values based on EPA studies. The Health Canada rates are arithmetic means based on probability density functions for 24-hour breathing rates.
Page 4 – 67: 3 paragraph from the top: TYPO “ popular erythematous rash” should be “papular erythematous rash”.		This error has been corrected.
Page 4-76; last paragraph; 60.000 should likely be 60,000. TYPO?		This error has been corrected.

<b>Section</b>	<b>Comment</b>	<b>Response</b>
p. 4-93 Section 4.25 Speciation Discussed Pb and As somewhat but nothing on the other 4 COCs.	Any final conclusions seem a little weak, or there is no clear reason/focus to the paragraphs. Perhaps an explanation of what 'could' or 'can' be done and what was done with an explanation of why any difference.	This section has been clarified.
Page 4-94 Paragraph 4: Is there a reference for the standard acceptable risk for the provinces (also small typo: "provinces <u>g</u> of")	Health Canada has a risk as 'essentially negligible' where the estimated ILCR is < 1 in 100,000.	Reference to the acceptable risk levels in the provinces of Manitoba and Saskatchewan have been removed and replaced with discussion of the acceptable risk as defined by Health Canada.
p. 4-96 top sentence "an alternate acceptable risk level, such as one-in-one hundred thousand ( $1 \times 10^{-5}$ ) may be appropriate".	This would be "an alternate" from what? Given that MB does have the same criteria for risk, as stated in the bottom paragraph on page 4-95 and Health Canada already states 1 in 100,000?	This sentence has been removed.
<b>CHAPTER FIVE</b>		
Arsenic - p.5-5 Table 5-1; the 24-hr TRV for As is 0.3 $\mu\text{g}/\text{m}^3$ while the 1-hr TRV is 0.19 $\mu\text{g}/\text{m}^3$ .	Appendix A, Table A1-1 p. A1-1 shows there is no reference provided for the acute 24-hour but accepted as a regulatory by the OMOE while on p.A1 – 8, 2 <sup>nd</sup> paragraph says it was derived by OMOE – Can there be a reference stating the OMOE?	The table is correct in that it indicated that OMOE did not provide details of the primary toxicological study used to derive their 24-hour AAQC.
Mercury - p.5-5 Table 5-1; the 24-hr TRV for Hg is 2.0 $\mu\text{g}/\text{m}^3$ while the 1-hr is 1.8 $\mu\text{g}/\text{m}^3$ .	Appendix A, Table A6-1 p. A6-1 shows there is no reference for acute 24-hour for inorganic Hg, but it is accepted as a regulatory by the OMOE; also page A6-8 discusses inorganic Hg but does not mention the acute effects for inhalation and the reference values used – can you please clarify why there is no reference and why it is then used by the OMOE, did the OMOE derive the value?	The table is correct in that it indicated that OMOE did not provide details of the primary toxicological study used to derive their 24-hour AAQC.
Copper – p.5-5 Table 5-1; the 24-hr TRV is 50 $\mu\text{g}/\text{m}^3$ while the 1-hr TVRV is 100 $\mu\text{g}/\text{m}^3$ .	Appendix A, Table A3-1, p. A3-1 shows there is no reference for the 24-hour inhalation, but that it is accepted as a regulatory by the OMOE; page A3-3, just states that the values were based on studies done by Whitman and Gleason – can you please clarify why there is no reference and why it is then used by the OMOE, did the OMOE derive the value based on the Whitman Gleason study?	The table is correct in that it indicated that OMOE did not provide details of the primary toxicological study used to derive their 24-hour AAQC.

Section	Comment	Response
<p><b>ARSENIC - Long Term Residential Results p. 5-8 to 5-19</b>  <b><u>(THIS IS FROM THE EMAIL SENT PREVIOUSLY)</u></b>                      (1) In regards to the sampling of the arsenic levels in the Creighton drinking water I have a query regarding the sample site for the Creighton-2 results.</p> <p>The arsenic levels taken from Creighton-2 sampling site had a mean of 9.8 and a max of 12 µg/L with a 95 UCLM of 10 µg/L. (thus the mean is nearly at the MAC or maximum acceptable concentration level).</p> <p>The interesting fact is all the other sample sites have significantly lower levels (all the same drinking water supply):                      So:                      4) Creighton-1 sample mean is 1.9 (max is 2.4);                      5) JW Sampling at 11 other sites: mean was 2.0 with max of 2.7; and                      6) The levels taken by the community (as required by Sk Ministry of Environ) on Table 2-5 were 2.2 for 2005 and 1.4 for 2006.</p>	<p>Thus one site has a mean arsenic level 4 to 5 times higher than all the other sites. Is there something about this site that we should be aware of? Is this site specific or something to do with the water supply? This makes quite a difference in terms of public health advise re: water supply but also to the HHRA calculation and areas for biomonitoring</p> <p>Alan and Elliot – can you let George and I know where this site is and what building it is? I'm not aware of anything within a specific site that would elevate that site's arsenic level (as you could have with plumbing for lead or copper) but I think we should check this out (? Is it a HBMS building on site, confirm that it is on CR municipal water system, is there a cistern?).</p> <p><b>According to HBMS the site that was giving the high Arsenic levels was actually a raw water source, it was a sampling error. This information will be forwarded to Intrinsic and the new calculations will reflect this.</b></p> <p>Whole section of 5.2.1 Arsenic needs changing.</p>	<p>The exposure and results for arsenic have been revised based on the exclusion of the Creighton-2 drinking water concentrations.</p>
<p><b>ARSENIC - Long Term Residential Results p. 5-8 to 5-19</b></p> <p>Bottom page 5-14 “while new data, more relevant data area developed for use in North America HHRAs”</p>	<p>Can we get references for the new data, more relevant data?</p>	<p>Since this data has not been endorsed by any regulatory agencies and therefore was not utilized in the assessment, this text has been removed.</p>
<p><b>For Figures like Figure 5-4:</b></p>	<p>Can you also provide a table to give a more accurate review of the HQ?</p>	<p>Detailed exposure and risk tables are provided in Appendix M.</p>

Section	Comment	Response
<p><b>CADMIUM - Long Term Residential Results p. 5-20 to 5-26</b></p> <p>Table 5-13, p. 5-24 (for June 2007 – July 2008)</p> <p>It states that there were 2 exceedances for Creighton TSP derived Cd data based on 0.025ug/m<sup>3</sup>; however, I found 3 exceedances (June 07 to July 08).</p>	<p>Go through the Air Quality Results from FF/Creighton and see how many exceedances and which were related to forest fires and winter wood burning for 2008 year.</p> <p><b>See Document Cd Comparison Data in the Draft Document Folder.</b></p> <p><b>TSP – July 08 to June 07 = 3 exceedances</b>  <b>PM10 – July 08 to June 07 = 5 equivalent or exceedances</b></p> <p>Please advise re: OMOE guidelines being for TSP.</p> <p>There is a issue that some exceedances may be due to times of forest fires and climate situations with wood smoke use in Creighton.</p>	<p>It is not anticipated that the exceedances related to winter wood burning or forest fires can be confidently distinguished from smelter related exceedances.</p> <p>Based on the information provided to Intrinsic, between the dates of June 2007 and July 2008, there were two days in which the concentration of cadmium in TSP measured at the Creighton School exceeded a value of 0.025 µg/m<sup>3</sup> (i.e., 0.028 µg/m<sup>3</sup> on June 17, 2007 and 0.051 µg/m<sup>3</sup> on June 7, 2008).</p>
<p>p. 5-24, 3<sup>rd</sup> paragraph, it states that the HHRA uses PM10 data ...</p>	<p>It is not clear then why they did their comparison using TSP data rather than the PM10 data. Could Intrinsic clarify this please?</p>	<p>The comparison to TSP was completed in Table 5-13 to explain why the HHRA results differ from the results provided within the Manitoba Conservation air monitoring findings in which Manitoba Conservation uses TSP data. This discussion was included to illustrate why the HHRA is predicting risks when Manitoba Conservation is not.</p>
<p>p. 5-24 first paragraph, “since there is no air monitoring located in East Flin Flon or Channing...will result in an over or underestimation...”</p>	<p>Uncertainty factor to add to chapter seven</p>	<p>Based on comments provided by Dave Bezak from Manitoba Conservation and Stephen West from HBMS at the TAC meeting in Winnipeg on February 23, 2009, it has been confirmed that based on distance from the smelter and prevailing wind direction, use of the Ruth Betts air monitoring data for the community of East Flin Flon is considered to be a conservative assumption and will likely over predict risks. This has been included in the revised report.</p>

Section	Comment	Response
<p><b>CADMIUM - Long Term Residential Results p. 5-20 to 5-26</b></p> <p>Table 5-12- ILCR according to Table 5-12 lays out the ILCR for each COI. And it states that the ILCRs were above the acceptable risk level of <math>1.0 \times 10^{-5}</math> in EACH of the four COI.</p> <p>Furthermore, p.5-24 states “that the results of the CHA be considered as it represents a more accurate measure of the occurrence of lung cancer among residents in the FF / Creighton area”.</p>	<p>Can't be over confident that the CHA results not indicating an incremental risk or no risk because of the wide confidence limits 4 / 10,000 to 4/100,000 may not be detected in the data.</p>	<p>This statement is not intending to indicate that the CHA results show no incremental risk in the occurrence of lung cancer but rather that the results of this study may provide a more accurate measure given the conservatism associated with the HHRA assumptions.</p>
<p><b>Lead</b> p. 5-35 top paragraph “All HQs were below the acceptable HQ value of 1.0. These HQs were derived using the EPC for soil and dust selected to be representative of typical exposure to residents in each of the COI.”</p>	<p>For clarity, it would be good to reiterate that the IEUBK model also used the EPC for each COI.</p>	<p>As suggested, this has been added to the first sentence of Section 5.2.4.2.</p>
<p><b>Lead</b> p.5-39 mid of top paragraph “Secondly, the assumed ingestion rates set as default...”</p>	<p>Clarify ingestion rates for what – from the rest of the paragraph reader assumes ingestion rates for soil and dust.</p>	<p>Revision has been made as suggested.</p>
<p><b>Lead</b> p. 5-39 Table 5-24 “Percent Contribution of...Flin Flon Receptors”</p>	<p>Suggest for clarity to re-title saying “Percent contribution...Flin Flon Receptors as predicted by IEUBK”</p>	<p>Revision has been made as suggested.</p>
<p><b>Lead</b> p.5-39 Figure 5-19 is for West FF children aged 1 to 2 as predicted by the IEUBK model and p. 5-33 Figure 5-13 is for West FF toddler.</p>	<p>(1) For clarity and to ensure accurate comparison is toddler and children aged 1 to 2 in this context the same receptor? If so can the titles be re-tiled to reflect this?</p> <p>(2) Again for comparison sake, can Figure 5-13 have an addition to the title saying “Contributions...West Flin Flon as predicted by the HHRA Exposure Model”</p>	<p>Due to differences in the assessed age categories, a direct comparison cannot be made between the HHRA model results and the IEUBK results. However, the toddler represents the most sensitive receptor within the HHRA model and the child age 1-2 years represents the most sensitive receptor within the IEUBK model. Figures 5-13 and 5-19 have been revised to clearly indicate that they represent data for the toddler from the HHRA model and the child age 1-2 for the IEUBK model.</p>
<p><b>Lead</b> p.5-39 ....</p>	<p>To conclude the comparison of the HHRA model and the IEUBK model can there be some concluding statement added stating which model was finally accepted/used in the HHRA?</p>	<p>Under the “Selection of a residential lead STC” section, a statement has been added to indicate the STC is based on the results of the IEUBK model.</p>

Section	Comment	Response
<p><b>Lead</b> p.5-41 2<sup>nd</sup> paragraph "...the IEUBK model, a STC of 105 ug/g was derived to be protective of 5%...BLL of 10 ug/dL"</p>	<p>(1) Suspect this to be a TYPO in that it should be "a STC of 375 ug/g was derived to be protective of 5%...BLL of 10 ug/dL" (2) For way of comparison the STC derived using the HHRA model explains how the TDI of 3.6 is used and how the air, drinking water and dietary lead intake are remained constant with only soil/dust changes; is this the same of IEUBK and could a sample equation be added in this text to illustrate an example of the calculation? If not just more discussion as is found on p. 5-40 when the STC in the HHRA Model was derived.</p>	<p>This discussion has been revised for clarity.</p>
<p><b>LEAD: page 5-41:</b> Is there a typo in the second last paragraph `therefore, using the IEUBK model, an STC of 105 µg per g was derived to be protective of a 5% probability of exceeding a BLL of 10 (should that be 5 µg per dl) as in the statement just proceeding Figure 5-21</p>	<p>TYPO</p>	<p>Revision has been made as suggested.</p>
<p><b>Lead</b> p.5-47 Table 5-27</p>	<p>For completeness, can the slope factor of 0.1388 (as noted on top of page 5-44) be added in for the <i>Lewin et al., 1999</i> study (its in the text but not the table). Furthermore, for the <i>Mielke study</i> no slope factor was reported (p.5-54 bottom of second paragraph); and for the <i>Johnson and Bretsch</i> study no slope was derived (mid 3<sup>rd</sup> paragraph) can this be stated in the table as well <i>i.e.</i>, "no slope factor reported/derived".</p>	<p>Revision has been made as suggested.</p>
<p><b>Lead</b> p.5-50 "Recognising the limitations are associated with using the IEUBK model for a community based assessment."</p>	<p>For clarity are those the limitations that are outlined on the top of p. 5-39?</p>	<p>This statement has been revised for clarification.</p>
<p><b>MERCURY - Long Term Residential Results p. 5-51 to 5-57 Inorganic</b>  p. 5-51 mid 2<sup>nd</sup> paragraph; "concentration of Hg in tissues of wild game were predicted based on concentration of Hg measured in forest soils, surface water sediments and through conservative uptake and bio-transfer factors"</p>	<p>Could include as an "Uncertainty Factor" in Chapter 7</p>	<p>This has been added as an item in the uncertainties discussion in Chapter 7.</p>

<b>Section</b>	<b>Comment</b>	<b>Response</b>
<p><b>MERCURY - Long Term Residential Results p. 5-51 to 5-57 Inorganic</b></p> <p>p.5-52 Figure 5-22.</p>	<p>Re-title the Figure 5-22, p. 5-52, on include "inorganic" Mercury Exposure to a West Flin Flon Toddler. (Labelling of Fig 5-22)</p>	<p>Revision has been made as suggested.</p>
<p><b>MERCURY – p5-56</b></p> <p>The assumed bioavailability of 100%</p>	<p>Should the HHRA be stating what the bioaccessibility results were but because of the uncertainty of the science the most conservative approach was taken (ie 100% assumption). This may help to provide some ideas to the conservative nature of the HHRA HZ info.</p>	<p>This discussion is included within this section.</p>
<p><b>MERCURY - Long Term Residential Results p. 5-51 to 5-57 Organic</b></p> <p>p. 5-57 CCME recommends 100% of Hg total in fish be MeHg, however, HHRA samples fish had a MeHg of 79%</p>	<p>Which factor was used in the HHRA 100% or the 79%? – is it safe to use 79% when the results are in question (e.g., Why are methyl Hg levels higher than Total Hg)</p>	<p>The HHRA originally utilized the average methyl mercury content of 79%, The HHRA has has been revised to use the 95% UCLM value of 96% which is considered to be very conservative and more in line with the generic literature-based values.</p>
<p><b>MERCURY - Long Term Residential Results p. 5-51 to 5-57 Organic</b></p> <p>Appendix M-</p>	<p>Appendix M – No methyl Hg table for Creighton – explanation? (that is no equivalent of Table 5-34 for Creighton – following M27 is there a Methyl Mercury – Creighton table)</p>	<p>A methyl mercury exposure table for Creighton residents has been added to Appendix M.</p>

Section	Comment	Response
<p><b>MERCURY - Long Term Residential Results p. 5-51 to 5-57 Organic</b></p> <p>p. 5-60 "HQs for the infant are very low because it was assumed that they would not be consuming fish within the first 6 months of birth"</p>	<p>What about the transfer to breast milk, any reference to support no transfer to the fat of breast milk? The ATSDR states breast milk is an exposure route for breast milk, especially <u>inorganic</u> mercury, can Intrinsik clarify the statement on page 5-60.</p>	<p>While this may be a potential pathway of concern, a literature search has indicated that there are no relevant methodologies for evaluating this pathway for mercury. Those guidance documents that include an assessment of the transfer of chemicals <i>via</i> breast milk have indicated that it is not relevant for mercury. A guidance document prepared for Cal EPA recommends a breastmilk bio-transfer factor of zero for both inorganic and methylmercury (McKone, 1992). A number of U.S. EPA documents, including U.S. EPA (2005), provides guidance for transfer of chemicals such as dioxins (and dioxin-like PCBs) for exposure from ingestion of breastmilk, but either do not recommend biotransfer factors for mercury or recommend a value of zero.</p> <p>McKone, T.E. 1992. CalTOX, a Multimedia Total-exposure Model for Hazardous-wastes Sites. Part III: The Multiple-pathway Exposure Model. Office of the Science Advisor, Department of Toxic Substances Control, California Environmental Protection Agency, Sacramento, California.</p> <p>EPA. 2005. Human Health Risk Assessment Protocol for Hazardous Waste Combustion Facilities.</p>
<b>CHAPTER SIX</b>		
<p>Section 6.8 p. 6-32 "3 of the 6 COC are actually essential elements"</p>	<p>In the state that is found coming out of the smelter and Not sure if this is what the community needs to hear – not sure they want to get it from the smelter. Should reword.</p>	<p>This sentence has been removed from this section.</p>
<p>6.8.3 p.6-33 "overview of the update..."</p>	<p>Should "update" be "uptake"? TYPO</p>	<p>Revision has been made as suggested.</p>
<p>6.8.3 Cd – 1<sup>st</sup> paragraph talking about 5-10% of Cd found in human milk</p>	<p>What are the implications for the HHRA for infants?</p>	<p>While this may be a potential pathway of concern, a literature search has indicated that there are no relevant methodologies for evaluating this pathway for cadmium.</p>
<p>6.8.3 last sentence for Hg "as well it can be excreted through breast milk"</p>	<p>Note contradictory statement from Chapter 5 (P5-60), where it states HQ's for infants are very low as it was assumed that they would not be consuming fish within the first 6 months of birth. But for inorganic Hg, there can be exposure through breast milk.</p>	<p>Please refer to the response to the breast milk comment above.</p>

Section	Comment	Response
Section 6.3 last paragraph, p.6-9 “no association between COC concentrations and employment by HBMS was noted”	– this info should be shown in Appendix	It was not considered to be necessary to provide a detailed comparison of indoor dust concentrations for homes occupied by HBMS employees relative to non-HBMS employees. A general statement of the findings is provided.
Section 6.4.1 on the bottom of p.6-10	the web-link <a href="http://www.atsdr.cdc.gov/iphome.html">http://www.atsdr.cdc.gov/iphome.html</a> had been changed to <a href="http://www.atsdr.cdc.gov/interactionprofiles/">http://www.atsdr.cdc.gov/interactionprofiles/</a> Update link	Revision has been made as suggested.
Section 6.4.2 p.6-12 Table 6-2 the footnote on the bottom of the table	This table may be misleading with Arsenic coming under “Metals with Possible Beneficial Effects” (at least have more of a discussion in text regarding uncertain animal research and no human research on the potential beneficial role of ‘small doses’ – ‘larger’ doses are still the issue.	The table has been modified to improve clarity.
<b>CHAPTER SEVEN</b>		
p.7-2, Section 7.3.1 Under “...data generated from MB Conservation...and Jacques Whitford...” first paragraph	Title is for both MB and JW data and HHRA does mention 10% of residential soils were sampled by JW, so in the same token / comparison - how many properties (non-residential) were sampled by MB Conservation; also what was the overall total number of sites sampled or number of samples taken and it would be nice to have a paragraph stating what percentage was in Creighton vs. Flin Flon.	Additional details regarding the soil sampling programs have been added to this discussion.
p.7-3 middle of third paragraph “for chemicals such as arsenic where the inhalation pathway is a major contributor to the overall risk level..”	Suggestion to use Cadmium as the example based on Chapter 5 p. 5-20 to 5-26.	Cadmium has been included along with arsenic as one of the COC in which the inhalation pathway is a major contributor to the overall risk level.
p.7-3 top of 4 <sup>th</sup> paragraph “in most cases...a value equal to the detection limit was selected”	For clarity, please provide an example of where this was done (i.e., what value for what chemical for what environmental media)	Examples have been added to this section as suggested.
p.7-3 last sentence of 4 <sup>th</sup> paragraph “in situation when laboratories could not meet standard DL and elevated levels were reported .... a value of ½ the DL” was used	(1) For clarity please provide an example of a situation where this occurred, as with previous statement; (2) this whole statement is a little confusing in that the DL could not be met and elevated levels were reported so why assign a value of ½ DL?	This discussion has been revised to more accurately reflect the final methods employed in the HHRA.
p.7-4 Top is a section re: uncertainty of outdoor air concentration to represent indoor levels	<b>Before this section and following the MB JW soil sampling program</b> , Strongly suggest <b>adding a section</b> stating the uncertainty of how the COC measurements in the PM10 range were calculated using Regression Equations. e.g., chemical-specific regression equations were used, based on historical paired TSP and PM10 from the <u>Provincial Building</u> were used to predict As, Cd, Cu and Pb and <u>Ruth Betts and Creighton</u> for Hg and Se. Suggest add section such as “Use historical paired TSP and PM10”	A section describing the uncertainties associated with the derivation of ambient air concentrations has been added to Chapter 7 as suggested.

<b>Section</b>	<b>Comment</b>	<b>Response</b>
p. 7-4 second paragraph "However, since air sampling has not been completed..."	Replace "be" with "been" TYPO	Revision has been made as suggested.
p.7-5 last sentence top paragraph "...method selected to characterize exposure and risk associated with indoor dust provided an <b>accurate</b> assessment for the overall population"	Recommend replacing the word "accurate" with a more risk type word – maybe confident, approximate, estimate, reasonably accurate, etc.	This has been revised to say "reasonably accurate..." as suggested.
p.7-5 3 <sup>rd</sup> paragraph discusses that the frequency of meals was 1.5 / week and 1 / week for fish and wild game respectively;	However, an uncertainty factor not mention here is the serving size, it was assume the serving size would be 227g but it was not derived from the local population and a discussion of its implications are requested. Important to mention serving size is an assumption.	Additional discussion has been added to address this uncertainty.
p. 7-5 last paragraph, "Since concentrations of mercury were not measured in wild game..."	Recommend inserting a separate Uncertainty paragraph indicating that the given this fact there is some uncertainty related to the numbers generated for the concentrations of COCs in the wild game tissue. Also a discussion relating to the implications of this.	Additional discussion has been added to address this uncertainty.
p.7-5 5 <sup>th</sup> paragraph - The discussion of mercury in fish vs. mercury in wild game.	Is the discussion referring to total mercury EPCs? Can this be clarified? From p. 5-51/5-52 it appears that methyl Hg is greatest for fish while inorganic is greatest for wild game?	This discussion has been revised to reflect the revisions to the wild game modelling using the measured mercury concentrations in wild plants, and the confirmation that fish tissue concentrations were reported in units of wet weight rather than dry weight.
p. 7-6 2 <sup>nd</sup> paragraph; "STC for mercury allocated a portion of the RTDI to exposure anticipated from dental amalgam"	But as with the comment on lead being found in hair dye and cosmetics I would suggest that the HHRA also make clear or give examples of mercury exposure through consumer products could also occur through things such as <b>herbal remedies and skin lighting cream</b> – areas that the HHRA did not examine. (part of the uncertainty factor)	Revision has been made as suggested.
p. 7-7 3 <sup>rd</sup> paragraph "...the potential contribution from cigarette smoke to COC body burden could not be accurately quantified..."	Would request for it to be clear on what COCs the document is referring to, is it just As, Cu, Pb and Se or is it all 6 COCs?	The section of text discussing the level of COC in cigarettes has been updated to include discussion of all 6 COC.

Section	Comment	Response
<p>p. 7-7 re-suspended dust pathway; US EPA recommends studying it if site-specific condition indicate it to be a potential pathway...the HHRA did not specifically evaluate the re-suspended dust pathway</p>	<p>It appears that tailings dusting events would not be considered as re-suspended as it would be captured at the Creighton school monitoring site. What type of thing would be included as 'site-specific exposure setting characteristics indicating a potentially significant pathway' for resuspension?? Would street-cleaners (dry), vacuuming, etc. be included?</p>	<p>It is uncertain what Health Canada would consider to be applicable in this situation. In many RAs, ambient air monitoring data is not available. As a result, the inhalation pathway is assessed by multiplying the COC soil concentration by a generic atmospheric dust concentration to estimate the concentration in ambient air available for inhalation. Since ambient air measurements are available for the current HHRA, additional consideration of re-suspended dust was not considered to be necessary.</p> <p>Construction activities has been added as an example to this discussion as a situation where someone may receive elevated exposure to re-suspended dust.</p>
<p>p.7-7 following "the re-suspended dust pathway"</p>	<p>Is this a TYPO with "A site-specific bioaccessibility study was conducted as part of the HHRA" meant as a subtitle (as it is not linked to resuspended dust pathway)</p>	<p>Subtitle added as per suggestion.</p>
<p>p.7-11 explains that the different inputs variables were tested and is outlined in Tables 7-4 to 7-9</p>	<p>Could some discussion be put into the document regarding the findings, the implications, any significant issues, etc? Also a comment or tables for other COI sites. It would also be good to have an additional column or two with the two HQ values (from value used and with adjusted value)</p>	<p>A summary of the most significant findings of the sensitivity analysis has been added to Section 7.4. Reference to HQs has been included in this discussion for variables that had a significant impact on the predicted HQs.</p>
<p>P 7-11, 7-12 and 7-13: Implications of uncertainty due to water Hg testing</p>	<p>With the new information regarding the follow-up testing of lakes for Hg, this could be included as an uncertainty and a sensitivity analysis could be done for Hg (using test results from Stantec and those done through MB Conservation)</p>	<p>All exposure and risk calculations associated with mercury in surface water have been revised based on the supplemental results collected by Manitoba Conservation.</p>
<p>P 7-13: Sensitivity Analysis for Lead as Assessed Using the HHRA Model</p>	<p>Can comparative sensitivity analysis be done for the IEUBK model?</p>	<p>A sensitivity analysis for the IEUBK model was added to Chapter 7.</p>
<p><b>CHAPTER EIGHT</b></p>		
<p>P8-1 It might be worth commenting that some exceedances of air quality can be due to forest fire (summer) or, for Creighton, wood (winter) smoke.</p>		<p>This comment has been added as suggested.</p>
<p>P8-3: Table 8-4: Needs recalculating based on the corrected Creighton arsenic in water results</p>		<p>Results have been revised.</p>
<p>P 8-4 (bottom) and top p. 8-5 "risks associated with ... the consumption of drinking water were significant"</p>	<p>This will need to be changed based on water results from Creighton</p>	<p>Results have been revised.</p>

Section	Comment	Response
P8-5 2 <sup>nd</sup> paragraph:	Any age recommendations because of the ingestion rate (proportion) is higher for infants/toddlers / infants so study could be made stronger by oversampling young children?	While it is important to ensure that there is an adequate number of children included within a urinary arsenic study, receptors of all age categories will have unique opportunities for exposure to arsenic and offer valuable insight into the overall community health.
p. 8-5 4 <sup>th</sup> paragraph "...it is recommended that the results of the CHA be considered as it presents a more accurate measure of the occurrence of lung cancer..."	But with the challenges of small population numbers leading to some limits to certainty and there are multiple influences on lung cancer (e.g., smoking rates)	It has only been advised that the results of the CHA be considered.
p. 8-6 second paragraph discusses the HHRA Model and the US EPA IEUBK Model. "...the HHRA model estimated HQ values associated with Pb exposures and were less than 1.0 under all exposure..." also using the IEUBK model "Predicted BLLS...were well below 10 ug/dL"	But from Chapter 5 Table 5-26 p. 5-42 and p. 5-40 for the HHRA Model "a soil concentration of 870ug/g produces an exposure of 3.6 µg/kd/day and results in a total HQ of 1.0" Furthermore, p. 5-40 for the IEUBK Model an outdoors soil concentration of 375 µg/g...was found to result in ...BLL 10 µg/dL" So the statement needs to be made clear on the differences between a population based (community) HQ and a STC – PRG level (e.g., the yard specific HQ<1 is only there when the STC is <870 µg/g (for the HHRA Model) and the BLL is <10 (for the IEUBK model) only when the STC is less than 375 µg/g. )	This discussion has been revised to help improve clarity.
p. 8-6: second last paragraph: states approx 40% of West FF properties contain levels of Pb that may have adverse effects, and 3% for East FF and 13% for Creighton.	It looks like approx 40% is derived from Table 8-10. Where does the 3% and 13% come from where on the table it looks like approx 9% and 18%.	These percentages are reflective of information provided in Table 8-15; calculated as the percentage of all properties sampled in each COI, that exceed the residential STC for lead (i.e., > 375 µg/g). Reference to Table 8-15 has been added to the text for clarification.
p. 8-7 3 <sup>rd</sup> paragraph "...HQs were below acceptable value of 1.0 <b>indicating</b> ..."	TYPO replace <b>indicating</b> with <b>indicating</b> .	Revision has been made as suggested.
p.8-7 3 <sup>rd</sup> paragraph...	ATSDR states that inorganic Hg is expressed in breast milk, as well as Me Hg to a limited extent – can we get more discussion on this please (perhaps under previous sections), especially as to why or why not this route of exposure may not apply to this HHRA?	Please refer to the responses to previous comments on the potential for exposure to mercury via breast milk.
p. 8-8 top sentence "... value of 1.0 <b>indicating</b> ..."	TYPO replace <b>indicating</b> with <b>indicating</b> .	Revision has been made as suggested.
p. 8-9 to 8-11 Explaining the PRGs and STCs for each chemical	Recommend that for each chemical where they state "A comparison of the...STC...with...residential soil sampling program..." that they state what the CCME soil levels was for sake of a quick comparison.	Given that one of the primary products of the HHRA is the derivation of area-specific PRGs and STCs that considered the unique environmental conditions of the Flin Flon/Creighton area, it is preferred not to re-introduce the CCME guidelines which are based on highly generic and conservative assumptions.

<b>Section</b>	<b>Comment</b>	<b>Response</b>
Page 8-9: Table 8-11: C	Review this table in light of the new calculations for water EPC for arsenic in Creighton. Is there any impact on changing CR EPC from 10 to 2.2 for arsenic on the number of homes in CR above the STC or would that just impact STCs for metals not considered carcinogenic.	As per the methodologies used by CCME, the derivation of guidelines for carcinogens are based on the protection of a one-in-one hundred thousand ILCR per environmental medium. Therefore, the residential arsenic STC is based on exposure to soil only and is not influenced by the concentration in drinking water.
<b>APPENDICES</b>		
Appendix E: 3.4 – Table 3.1	Highlight only mercury in water samples that exceeded guidelines, BUT presently all the mercury results are bolded – means that all exceed environmental water quality guidelines (MB Water Quality Objectives).	Stantec is currently in the process of revising this report. The updated report will replace the draft report in Appendix E.
Appendix M – No methyl Hg table for Creighton – explanation?	There is no equivalent of Table 5-34 for Creighton – following M27 - is there a Methyl Mercury – Creighton table.	A table for methyl mercury for Creighton residents has been added.
Appendix O: 0-9 – game meat equation ‘se dim ent’	Should be reformatted to read ‘ <i>sediment</i> ’	It is unclear why, but Microsoft Equation automatically adjusts “sediment” and “constant” to be split into regular and italic letters. Revision could not be made as suggested.

## HHRA of Flin Flon, Manitoba, and Creighton, Saskatchewan (Draft Report December 2009): Comments Saskatchewan Health, Population Health Unit February 12, 2010

Here is a synopsis of the comments on the December 2009 version of the HHRA for FF/CR. Overall our comments and suggestions are minor. Any substantive issues have been communicated to you already (though included in this spreadsheet as well) and most of the remaining comments are typos, and suggestions for clarity. The report is well written and has incorporated or addressed our concerns from previous versions. Thank you for responding to our questions during the course of this latest review.

<b>Page No. (Location)</b>	<b>Comment</b>	<b>Response</b>
<b>EXECUTIVE SUMMARY</b>		
p. (i) first paragraph, 3rd sentence	wording - replace Saskatchewan Environment and Saskatchewan Health with Ministry of Environment and Ministry of Health	Revision has been made as suggested.
p. (v) 2nd paragraph, 1st sentence	wording - add the term "blood" "...Health Canada for <u>blood</u> lead is ..."	Revision has been made as suggested.
p. (vi) 3rd paragraph 5th sent	wording or sentence order for clarity - "This indicates that a number of residential ...exposure" should this sentence be after "...West Flin Flon contained concentrations in excess of this value" b/c it sounds like EFF, CR & CH were above but they were actually below (unlike WFF).	Revision has been made as suggested.
p. (vi) 5th paragraph	wording - RA - for the executive summary recommend this be spelled out	Revision has been made as suggested.
General comment for Exec Summary	It would be good to have an explanation of why a HHRA was required when there already are values from the CCME for residential soil levels for various metals?	This is explained within the main body of the report.
<b>CHAPTER THREE</b>		
p. 3-3 Figure 3-1	wording - For the figure to match the wording - advice to add Hwy 10 to the Figure	The original figure was taken from the Manitoba Conservation (2007) report. As this figure was manipulated from an adobe file and we are unable to add the Hwy 10 label to the figure as requested.
p. 3-6/3-7 re: soil sampling	clarify - Jacques Whitford had 369 samples, 334 samples for COIs (seen in Table 3-2) but 35 extra samples were taken from undisturbed areas, how were these 35 samples used?	These samples were not used in the assessment. Regional (non-residential) data available from Manitoba Conservation (2007) and provincial background data from the Geological Survey of Canada (Smith <i>et al.</i> , 2005), were instead utilized in the screening of COC.
p.3-7 re: boron 2nd paragraph	wording - paragraph appears incomplete, suggest adding a qualifier as to why it was excluded from the COC process.	Boron was not excluded from the COC process. This paragraph is meant to describe how the maximum concentration of boron was calculated. The statement "The calculated maximum total boron concentration was used in the COC screening process for the selection of COC in soil" was added to clarify that boron was not excluded from the COC process.
p.3-10 Table 3-4	There is a column for the MB background concentration data, which comes from the major Geological Survey, is it possible to add an extra	Revision has been made as suggested.

Page No. (Location)	Comment	Response
	column for the Baker's Narrow/Cranberry Portage data for the regional background information (as stated on p.3-11 2nd paragraph) - for ease of comparison?	
p. 3-12 Table 3-5	Table 3-5 for <b>Aluminum</b> is missing from the Table of Contents and this would then bump up all the other table numbers afterwards.	Revision has been made as suggested.
p.3-17	Can you add the definition of Central Tendency Estimate to the glossary?	Revision has been made as suggested; CTE also added to abbreviations list.
p. 3-19 Table 3-9	Discrepancy - re: Toddler Receptors, when comparing the Health Canada "Part I: Guidance Document from Health Canada (2004)" and Richardson 1997 document for comparing the Market Basket Food Consumption Rates for root veg, other veg and fish, the numbers are 105, 67, and 56 respectively; while the HHRA has 79, 48 & 4.7 respectively but both Health Canada and Intrinsic site Richardson 1997, please explain discrepancy.	The receptor parameters presented in Health Canada (2004) and Richardson (1997) for a sample considering "eaters" only. We have adjusted consumption rates to account for both "eaters" and "non-eaters" as would be expected in the general population ( <i>i.e.</i> , the intake rates presented in Richardson (1997) were adjusted to represent per capita intake rates). An explanation of this has been added to Ch. 3 text.
p. 3-26 Figure 3-2	Suggest footnoting the difference between Figure 3-2 and what was actually done in the study in that the additional routes of exposure for this study were: a. swimming - ingestion & dermal; b. snow - ingestion; c. inhalation - indoors & outdoors qualifiers; & suggest commenting that <b>local</b> beef and dairy were not assessed.	Figure has been revised as suggested.
	<b>Outdoor / Commercial Worker</b>	
p.3-28	1. Clarify why they used only the MB soil study data and not all the soil data - is it because MB study data is strictly for public grounds where workers would more likely be involved?	MB data was used because it was from public lands rather than residential properties.
	2. Clarify why it appears that the outdoor worker scenario is done in such a way that they come and leave the area for 14 hours a day ( <i>i.e.</i> , just work in the area for 10 hours) vs. commercial workers who are actual residents of the Flin Flon/Creighton area with other exposures?	If it was assumed that the worker lived within the Flin Flon area, then risks would essentially be equal to those predicted for the residential scenario.
	3. p. 5-73: Table 5-42 does summarize the HQ for an outdoor worker for the COC and routes of exposure, but can we get the exposure levels added to this table or have an additional table like Table 5-39?	Revision has been made as suggested. Additional table added to text ( <i>i.e.</i> , "Table 5-42 Exposure Estimates for an Outdoor Worker ( $\mu\text{g}/\text{kg}/\text{day}$ )")
p.3-28	wording - re: Residential Typical Background Scenario - since the North American food was mentioned suggest adding a sentence by way of introduction re: (a) Cranberry Portage, Baker's Narrows soil data was for soil; (b) air data was taken from Toronto, Winnipeg & Saskatoon; & (c) water data was taken from Ontario OR a reference made here to 4.1.2 p. 4-22 for more details.	Revision has been made as suggested. The statement " <i>The data used in the residential typical background scenario are described in Section 4.1.2</i> " was added for clarification.
<b>CHAPTER FOUR</b>		
Table of contents	wording - There is a lot of detail in this chapter and to allow the reader to follow the information, recommend adding all subtitles from the text into the table of contents ( <i>i.e.</i> , 4.1.1.1, 4.1.1.2,	Revision has been made as suggested.

Page No. (Location)	Comment 4.1.1.3....4.2.3.6)	Response
p. 4-3 Table 4-1	clarify - it states there were 29 samples taken in Creighton, but Jacques Whitford Original report and p.5-25 Table 5-11 states 30 properties were sampled - clarify?	A total of 68 soil samples (from 30 properties) in Creighton were collected as part of the JW sampling program. In the derivation of EPCs only surface soil samples were considered (i.e., samples collected in the 0-2.5 or 0-5 cm bgs profile). A total of 29 of the 68 samples collected fell into this depth range (as presented in Table 4-1, n=29).  The value 29 refers to the number surface soil samples collected in Creighton; the value of 30 refers to the total number of properties sampled in Creighton.
p.4-6 Table 4-4	clarif - Footnote "b" says 13.4 factor applied but text says 12.4 - clarify please?	Revision has been made as suggested (footnote "b" has been corrected).
Page 4-8 Table 4-6	ARSENIC WATER - from the JW Sampling Program the maximum was 2.7 µg/L mean of 2.0 µg/L and 95% of 2.2 and an EPC selected at 2.2 µg/L, however, p. 4-1 Section 4.1.1 of the JW Report for the CR data they state there are 11 samples for CR, but note that samples CS 106, 107 and 108 were in SK BUT were taken from the City of Flin Flon water supply. It is CS 106, 107 and 108 that actually had measured the maximum of 0.0026 mg/l (2.6 µg/L), 0.0027 mg/L (2.7 µg/L) and 0.0026 mg/L (2.6 µg/L) which then seems to give a max of 2.7 µg/L; a mean of 2.0 µg/L; and a 95% of 2.2 µg/L as used by the current HHRA. For CR itself, with CS 106, 107 and 108 removed the max would appear to be 0.0019 mg/L (1.9 µg/L) and a mean of 0.0017 (1.7 µg/L) and a 95% of .... please clarify & adjust??	The notes provided by the reviewer are correct. For conservatism drinking water samples CS106, CS107, and CS108, collected in SK but sourced from the City of Flin Flon water supply, were included in the determination of a drinking water EPC for Creighton. This assumption was made to ensure that the Creighton drinking water EPC was protective of Creighton residents accessing drinking water from the City of Flin Flon water supply.
p. 4-11 Table 4-7	typo? - For Arsenic in "below grd vegetables" there is a superscript "b", should this be a superscript "c" for inorganic fraction only?	Revision has been made as suggested.
p. 4-12 Table 4-8/4-9	wording - Is mercury total mercury - if so can "total" be added to the column?	Concentrations are total mercury. Revision has been made as suggested.
p. 4-12 Table 4-8	wording - Recommend rewording to "95% UCLM Concentrations for <u>each</u> COC in local fish for individual species <u>from all lakes</u> "	No revision made.
p. 4-12 Table 4-9	wording - Recommend rewording to "95% UCLM Concentrations for <u>each</u> COC in <u>all local</u> fish <u>species</u> for <u>each</u> individual <u>lake</u> "	No revision made.
p.4-15 & 4-17,	Typo ? - Comparing Tables 4-12 & 4-14 the 95% UCLM for measured indoor dust for Creighton Pb is 264 µg/g and West FF is 265 µg/g but Table 4-14 & 4-15 the measured EPC at 260 µg/g for both COIs - clarify?	Two significant digits were used for EPCs. This explains the discrepancy between measured indoor dust values and the EPCs presented in Tables 4-14 and 4-15.
p. 4-17 Table 4-14	wording - wording seems a little confusing - recommend: "Comparison of indoor dust EPCs derived based on measured dust concentrations; and <u>Predicted concentration of COC in indoor dust based on EPC</u> outdoor soil	Title has been revised for clarity. "Table 4-14 Indoor Dust EPCs Derived Based on Measured Dust Concentrations <i>Versus</i> Predicted Indoor Dust EPCs Derived Based

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	concentration and site-specific regression equations; and the IEUBK MSA Module for <u>Lead</u> "	on EPC Outdoor Soil Concentrations and Site-Specific Regression Equations and the IEUBK MSA Module for Lead ( $\mu\text{g/g}$ )"
p. 4-18, 4-15	Typo ? - Table 4-15: The EPC for Indoor Dust selected for Pb in WFF & CR was 260 (which are measured values) - however, according to Table 4-12 the 95% UCLM measured indoor dust values for WFF and CR are 265 & 264 respectively - clarify (Just rounding off)?	See response above.
p. 4-19/20 Table 4-18	typo? - re: blueberries, "...and mercury was below an MDL...at 12 sample locations...", but Table 4-18 indicates there were 13 samples (with a footnote) - does text need correcting?	The text is correct. The footnote was edited for clarity ( <i>i.e.</i> , "All samples were below the MDL, with a single exception in which mercury was at the detection limit, therefore, mean, maximum, and 95% UCLMs could not be generated. The EPC is the MDL.")
p.4-25 Table 4-24	clarify ? - footnote indicates a value for Cd in drinking water was not identified within the DWSP, so what values was ultimately used for the background water Cd level?	It was assumed that cadmium was absent from background drinking water.
Appendix F - Comment Response Table	The older Appendices we currently have do not have comments on the previous versions of the HHRA document - we presume this will be updated for the final version to have all comments submitted?	This assumption is correct.
p.4-32 Table 4-29 & p.5-56	wording - Recommend that Mercury be titled Total Mercury, and then a footnote stating the qualifier as outlined on p.5-56, 3rd paragraph, of Hg (inorganic) being 80% of the Air Concentration values, Hg (inorganic) being 96% of the fish values and Hg (inorganic) being 75 % of the drinking water values.	Title kept as "Mercury" to be consistent with the rest of chapter 4. Footnote added to table as recommended.
p.4-32 Table 4-29	typo? - note: why is Cu indoor dust concentration double the outdoor Cu soil concentration, typo or was this actually seen in the measured data?	This trend was observed for the measured indoor dust data (see Table 4-14). Note: the Cu indoor dust concentrations presented in Table 4-29 are predicted concentrations.
p. 4-37	For the equations, does the C for soil, dust <i>etc.</i> ( $C_{\text{soil}}$ ) represent the EPC? Please clarify in footnote or comment in the text near the equations?	The equation sections have been updated to include "exposure point concentration" in the definition of "C" values.
p.4-38	Clarify ? - Top paragraph, it mentions 10 hr/d for the Commercial scenario - however, Equation 3.0 does not seem clear how the 10 hrs/d is accounted for.	Soil ingestion is not a time-based parameter. The soil ingestion rate is event driven and applied to every day or partial day spent on site.
p. 4-39	clarify ? - Equation 4.0 for outdoor dermal soil exposure accounts for hands, arms & legs; while Equation 5.0 indoor dermal dust exposure only accounts for arms & hands, why isn't legs accounted for in indoor dust?	Exposure to indoor dust occurs during the winter months. It was assumed that receptors would not be wearing shorts during winter months.
p. 4-41	typo - Equation 8.0 has COA but it is not defined - is CIA in the definitions a typo?	Revision has been made as suggested.
p.4-43	wording - Equation 12.0, IRDW is this age specific? If so can it be added to the definition box?	The comment "(age-specific)" has been removed from all equation boxes. Almost all receptor parameters ( <i>e.g.</i> , consumption rates) are age-specific, but this

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		comment is only included in equation boxes 9, 10, 11 and 14.
p. 4-47	clarify ? - Table 4-34 - A fraction of the MB Intake rates was allocated for local foods - the wild game (meat) and vegetables - is there a reason why this approach wasn't this done for fish? The text does say that fish is a major part of the diet so it had its own calculations independent of MB Intake, but given high local fish intake rates would one also eat the same amount of fish based on the MB EDI? Is this an over estimation for MeHg exposure?	The market basket fish intake rate is very low relative to other food consumption rates. It was not considered to be appropriate to assume that receptors in the Flin Flon area would only consume local fish at this rate or a fraction of this rate. Exposure to methyl mercury was likely overestimated as a result of this assumption, however, it is the goal of the HHRA to err on the side of conservatism.
p. 4-47	clarify ? - Where does the fraction # come from for meats, p. 4-41 explains the fraction for root veg, but it's not clear about meat?	The fraction is equal to the wild game consumption rate (g/day) divided by the market basket food (for meats and eggs) consumption rate (e.g., adults: 32/158 (g/day) = 0.2 or 20%). As statement to this effect was added to the text.
p.4-48 Paragraph b/w equations last sent.	Typo - equailant - replace equivalent?	Revision has been made as suggested.
p.4-48 Paragraph b/w equations last sent.	Clarify ? - "The relative absorption factors were also assumed to be equivalent to those selected for soil" the original Stantec data for sediment (p.3.5-3.7 & table 3.3) does not seem to speciate for MeHg content in sediment is this correct? And if so how was MeHg accounted for in the swimming exposure route?	You are correct, methyl mercury content in sediment was not measured. For the purposes of exposure and risk calculations related to swimming, total mercury measured in sediment was assumed to be 100% inorganic mercury. This is consistent with methods used for soil.
p. 4-102 mid 3rd parag.	"In situations where risks are predicted to be w/i the same order of magnitude as the acceptable level, re-evaluation of certain model parameters...is conducted before the potential risks to health are fully characterized" Cd ILCR for CR = 4.5E-05, (WFF=6.9-04), and p.5-30 2nd par last sent indicates "...Cd in ambient air may have potential to result in unacceptable in the risk of developing lung cancer for receptors ...in the Flin Flon area" but would this be more specifically for WFF, EFF, CH but CR a little different?	You are certainly correct in noting that ILCR for Creighton are predicted to be within the same order of magnitude as the acceptable level, while ILCRs for other COI exceed the benchmark by more than an order of magnitude. However, the statement in Chapter 5 is not worded in such a way (i.e., "concentrations of cadmium in ambient air <u>may</u> have the <u>potential</u> to result in an unacceptable increase in the risk of developing lung cancer for receptors...") as to indicate there is a high potential for risk. The addition of "risk terminology" has been included in Chapter 5 to put risk into context – (e.g., Creighton considered "very low" and WFF considered "low" risk.)
<b>CHAPTER FIVE</b>		
p. 5-1	Add "short-term acute exposure" to the glossary	Definitions of acute and chronic are already included in glossary. These specific terms are not used within Chapter 5 and thus, were not added to the glossary.
p.5-2	Add "long-term acute exposure" to the glossary	
p.5-7	wording - Under "Acute Ingestion of Health	

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	Risks" IR acute is defined as "... normal children" but table is for toddlers, is the US EPA definition for children equivalent to toddler?	child ( <i>i.e.</i> , of ages six months through 84 months) is close to Health Canada's definition of a toddler ( <i>i.e.</i> , 7 months to 4 years). This value was selected to be consistent with ingestion rates used in the IEUBK model.
p.5-8	"Acute Ingestion of Snow" - explain why snow was not added to the overall exposure estimation with soil/dust or water intake?	The ingestion of snow is considered an infrequent (or intermittent) event, and is thus assessed for short-term (acute) effects. As discussed in Chapter 5, acute exposure estimates for snow are based on short-term transient exposure levels related to extreme activities. Short-term exposures are those defined as occurring over a less than lifetime duration or on an intermittent basis, and are typically characterized by a <u>single pathway</u> exposure only. For this reason, snow was not added to the overall exposure estimate with soil/dust or water intake.
p.5-8 Table 5-5	typo ? - The footnote explains "Short-term TRVs were not available for Cu, Pb & Se, as a result chronic TRVs were conservatively utilized..." however, Cu TRV according to Table 4-42 p. 4-66 is 90 but Table 5-5 lists 10 - while Pb and Se are kept at the Table 4-42 values of 3.6 & 6.2 respectively. Is this an error or why was Cu adjusted? If 90 is kept it would appear to lower the HQ overall from 0.5 to 0.055?	An acute oral TRV of 10 µg/kg/day was adopted from ATSDR (2004). The footnote has been updated accordingly.
General Comment re: Consumer Products	With the exception of lead, none of the COCs are given a consumer product fraction of 0.2 in the hazard quotient as presented on p. 4-101. For example Selenium p.5-73 Table 5-39 a fraction 0.2 for the HQ was not given for consumer products & Cd seems to be found in several consumer products, - can there be more of an explanation as to why this was not done. There is a section on p.7-8 to 7-10 reviewing the consumer products but the text in Ch. 5, for the development of the PRGs and PTCs, is not linked well to Ch. 7, and Ch. 5 or Ch. 7 doesn't explain clearly why the HQ of 0.2 was not used for each COC except lead.	Exposure to COC other than lead through consumer products is generally not considered to be a significant pathway that requires quantitative assessment within the HHRA. Exposure to these COC from consumer products is considered to be minor and highly uncertain.
General Comment - Discussion Point	The Pb PTC (370), was developed based on the IEUBK Model, and on the air data from WFF (p.5-45 mid 1st para & last para). For CR the soil EPC (250) is less than PTC its means that 4/30 CR properties are in exceedance, but... what difference would it make if a PTC were developed based on CR air data since it appears the HQ for air Pb would be ~ 0.00533 a factor of 10 less than the WFF HQ air Pb = 0.0532.	Using air and drinking water concentrations specific to Creighton, the PTC for Creighton would be approximately 405 µg/g.

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<b>ARSENIC</b>		
p.5-9	wording - "...and the consumption of drinking water 24, 19, 16 & 24%." can add something like "for - - - and - respectively" to indicate clearly what COI this is referring to?	Revision has been made as suggested.
p.5-10	Clarity and ease of reading: it would be very useful to have a Table for Creighton like Table 5-6 near the Creighton pie chart for view as with WFF.	Addition of this tables was deemed unnecessary, as the information is provided in Appendix M.
p.5-11 Table 5-6 (applies to all the COCs)	typo ? - Exposure Pathway Column- for the Home Garden - other Vegetable should 0.012 be 0.014 (based on the EPC Summary table on p.4-33?)	Revision has been made to the Table 4-29.
	There are no equations in Appendix M (as outlined on p.5-10) for this table, presume that the equations will be coming in the final draft - hard to follow how the "average % of Total EDI" were calculated?	No reference to equations is made on pg. 5-10.
p.5-12 (applies to all the COCs)	<p>Se, Hg (inorganic), Pb, Cu, Cd have Tables such as <b>Table 5-15 p. 5-31 "Exposure and Risk Contribution per media assumed within the derivation of the residential Cd Soil PRG"</b> that summarize the "exposure and risk contributions per media assumed w/i the derivation of the residential Cd Soil PRG" however, <b>As and MeHg do not</b> - can these be added to applicable sections please?</p> <p>Also for the final document can there be reference made to an Appendices of where all these <b>Hazard Quotient (for chronic exposure) calculations will be laid out, with worked examples and with summation tables?</b></p>	<p>Inclusion of this type of table is not relevant for MeHg or As. As MeHg content in residential soils is assumed to be negligible, exposure to methyl mercury through direct soil/dust pathways was not assessed and a residential soil PTC was not derived. Unlike the other COCs, the PTC for arsenic was derived based on CCME (1999) methods to protect against an ILCR of <math>1.0 \times 10^{-5}</math>, thus, the suggested exposure and risk table is not relevant to the derivation process.</p> <p>Sample calculations for HQs and ILCRs are presented in the worked example (Appendix H) and summed HQ/ILCR results are presented in Appendix M.</p>
p.5-12 (applies to all the COCs)	wording - "Hazard Quotients for Non-cancer Risks" recommend rewording "Hazard Quotients for Chronic Oral Non-Cancer Risks for Arsenic" - helps the flow and to more readily identify what TRV is being used & readily identifies it as a Chronic assessment. This would also apply to the "Hazard Quotient" titles for Cd (p.5-28); Cu (p.5-34); Pb (p.5-40); Hg (p.5-60) and Se (p. 5-70) as well.	No revision made.
p.5-12/13	Presume that Tables & Figures on p.5-12 to 5-14 may be reworked as a result of the lower Creighton water As exposure, after Flin Flon, SK data has been removed and only Creighton water data is used for Creighton.	The EPC for Creighton drinking water is addressed above.
p.5-13 2nd sentence top para. & 2nd para. [also see p.8-5 comment]	Reference to drinking water being one of the "...largest sources of overall arsenic risk to the residents of the COI." Some minor changes are needed for the Creighton 95%UCLM: after re-adjustment the 95% UCLM for Creighton, is 1.8 µg/L for arsenic as opposed to 2.2 µg/L originally used (Table 4-6 p.4-8); and given that the Creighton Water Works data for May 19, 08; June 4th, 06; Feb 6th, 05 were 2, 1.4 & 2.2	<p>The EPC for Creighton drinking water is addressed above.</p> <p>Discussion regarding the contribution of arsenic in drinking water to total exposure and risk has been revised to explain that the incremental contribution above the Typical Background exposure from drinking water is minor.</p>

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	<p>respectively  <a href="http://www.saskh2o.ca/mydrinkingwaterdata.asp">http://www.saskh2o.ca/mydrinkingwaterdata.asp</a>                      With Creighton' s levels are less than the SK provincial mean for As is 3.0 µg/L (Guidance for CDWQ Tech Doc - Arsenic, p.5 <a href="http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/arsenic/arsenic-eng.pdf">http://www.hc-sc.gc.ca/ewh-semt/alt_formats/hecs-sesc/pdf/pubs/water-eau/arsenic/arsenic-eng.pdf</a>) a qualifier may need to be added to the statement that although the major contributor may be water its water supply still has less As than the provincial mean and is less than the national MAC of 10 µg/L. (Sask data provided)</p>	
p.5-21 & 5-19	<p>clarify - Inconsistency between 2nd para, 3rd sentence "...Falconbridge, Ontario, mean and maximum soil arsenic concentrations of 79 µg/g and <b>555 µg/g</b> were reported..." but Table 5-10 (p.5-21) indicates Flaconbridge Arsenic soil concentration range (µg/g) levels were between <b>2.5-400?</b></p>	<p>Revision has been made to ensure consistency (values provided in the table were confirmed in SARA, 2008).</p>
<b>CADMIUM</b>		
p. 5-29/30 (Discussion / Qualifier ?)	<p>Table 5-14/ "overall, using the Health Canada cancer unit risk value...result in an unacceptable increase in the risk of developing lung cancer for receptors...in the Flin Flon area" Creighton is 4.5E-05, but according to p. (v) its worth noting in the 6th paragraph b/w 1/100,000 and 1/10,000 risk is very low vs. WFF 6.9 E-04! To put the risk of "unacceptable" into some kind of context can some wording around the risk being "very low" be added keeping with standard nomenclature?</p>	<p>Qualitative terminology has been added to express the varying levels of numerical risk.</p>
p.5-31 (applies to all COCs)	<p>wording ? - recommend rewording <b>Table 5-15 (and all similar COC tables - Table 5-20; 5-26; 5-33 &amp; 5-39 )</b> to include that, these <b>values are based Toddler Receptor Characteristics and are for West Flin Flon.</b> Then in the text a reference to an appendices of where to find the other similar tables for the other 3 COIs.</p>	<p>This is already included in the text leading up to the mentioned tables (<i>i.e.</i>, "The predicted daily exposure of a toddler living in West Flin Flon via drinking water..."). These tables were only provided for West Flin Flon; PTCs were not derived for each COI.</p>
<b>LEAD</b>		
p.5-44	<p>"Secondly the assumed soil/dust ingestion rates set as default in the IEUBL model are significantly higher than those recommended by Health Canada (2006)" can a reference to p. 7-21, Table 7-10 be made here?</p>	<p>Revision has been made as suggested (<i>i.e.</i>, added "(see Table 7-10)" to the text).</p>
p.4-18 & p.5-46	<p>clarity - 2nd paragraph, an indoor Pb dust concentration of 290 was used to generate the PTC, why wasn't the EPC of 260 or 265 (if correction is needed) be used instead of the 290 - or a footnote may need to be added to the table 4-15 on p.4-18?</p>	<p>A dust EPC of 290 µg/g was not used to generate the PTC; as described in the text, the PTC of 370 µg/g was back-calculated using the IEUBK model based on established to be protective of a 5% probability of exceeding BLLs of 5 and 10 µg/dL. Using the IEUBK MSA module, concentrations of lead in indoor dust are calculated as a function of the concentrations in outdoor soil.</p>
	<p>clarity - Note: Is it correct that the measured indoor Pb dust levels, 260 for ex in WFF, (265 if correction is needed) were used to predict the BLLs w/i each COI (p. 4-58 (mid 4th par) but</p>	<p>As noted above however, the overall PTC was developed to be protective of a 5% probability of exceeding BLLs of 5 and 10 µg/dL,</p>

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	<p>PTC overall was developed using WFF indoor dust predicted of 290 (p. 4-17 mid)?</p>	<p>not based on a the WFF indoor dust EPC. To do this, an iterative process was used (using the IEUBK MSA module) in which concentrations of lead in air and drinking water, and total dietary lead intake, remained constant <u>while the concentration in soil</u> was adjusted until each of the two criteria were met. This was done using environmental parameters associated with West Flin Flon. The PTC for lead was not developed using the indoor dust EPC.</p>
	<p>clarity - Since the 290 (based on the IEUBK model) was ultimately used to generate the PTC for all the COIs, what was the measured indoor dust concentrations for each COI used for?</p>	<p>See discussion above.</p>
	<p>clarity - Finally, how accurate can the PTC of 370 for CR be using the IEUBK Model generated numbers when "...the MSA module (IEUBK) under predicted concentrations in the other COIs." (p. 4-17 md section) - Any theories as to why the MSA Module did not work for Cr and the Other 2 COIs?</p>	<p>For the community of WFF, indoor dust lead concentrations predicted using the MSA module (and the EPCs for outdoor soil and outdoor air) are similar to the measured EPC for indoor dust in West Flin Flon. While differences between the measured and predicted concentrations were observed for other COI, the PTC to be applied to all COI was derived using the WFF parameters. Given that concentrations of lead in outdoor air in Creighton are lower than in WFF, the application of a single PTC to all communities is considered to be conservative.</p>
<p>p. 5-54 , Table 5-30</p>	<p>clarity - Can we have a footnote added to this Table stating that 1 of the 4 properties in exceedance was from a garden that had brought in soil from outside the area? Previously discussed to explain circumstances of this outlier</p>	<p>Details regarding the sample locations of other exceedances are not available, therefore, it is not considered to be appropriate to selectively comment on specific samples.</p>
<p><b>MERCURY</b></p>		
<p>p. 5.60 1st para</p>	<p>typo - "...below the acceptable value of 1.0 <u>indicting</u>..."</p>	<p>Revision has been made as suggested.</p>
<p>p.5-61 1st sent last para</p>	<p>wording - recommend re-wording "Given that the <u>chronic oral</u> TDI for..."</p>	<p>Revision has been made as suggested.</p>
<p>p.5-63 mid 1st para.</p>	<p>wording - "In the blood, Hg has a short half -life thereby making it useful..." for consistency with Pb, which is defined on p. 5-56 as being 36 days can Hg 1/2 life also be define?</p>	<p>Revision has been made as suggested (<i>i.e.</i>, "In the blood, mercury has a short half-life (<u>3 days</u>) thereby making it a useful medium for determining short-term exposures").</p>
<p>p.5-59, 5-62 &amp; 5-65</p>	<p>wording - Tables 5-31 &amp; Table 5-35 can add a footnote stating that the Hg inorganic and MeHg values were adjusted for in air, water and fish are based no the CCME doc and the local fish sampling program? It is in the text...</p>	<p>Specifics regarding derivation of environmental media concentrations are provided in Chapter 4.</p>

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<b>METHYL MERCURY</b>		
p. 5-63	It would be useful/valuable when reading the information that again, for <b><u>methyl mercury and arsenic</u></b> there be a table presented for <b><u>"Exposure and Risk Contribution per media assumed within the derivation of the residential inorganic ..."</u></b> as was done for the other COCs?	As noted above, inclusion of this type of table is not relevant for MeHg or arsenic. MeHg content in residential soils is assumed to be negligible, and therefore, exposure to methyl mercury through direct soil/dust pathways was not assessed and a residential soil PTC was not derived.
p.5-67 3rd sent, 1st para	wording - "This assumes that these receptors...at the 95% UCLM concentration (0.43 µg/g MeHg or 0.45 µg/g total Hg)" recommend adding 'ww' with the values for consistency?.	Revision has been made as suggested.
<b>SELENIUM</b>		
p.5-70	wording - Again the title "Hazard Quotients" can it be re-worded to something like "HQ for Chronic Oral Exposure" also for the other COCs?	This comment is addressed above.
p.5-71 2nd para, 1st sent	wording - recommend re-wording "Given that the <u>chronic oral TDI</u> for..."	Revision has been made as suggested.
p.5-73 Table 5-39	wording - Table 5-39 again, as with the similar Tables for the other COCs - recommend rewording the title to show it is for a toddler in West Flin Flon.	This comment is addressed above.
<b>CHAPTER SEVEN</b>		
p.7-2 3rd sent, 6th para	wording - "In addition, the Flin Flon Technical Advisory Committee (TAC)...", suggest adding that a Community Advisory Committee was also formed and provided feedback to the whole process.	Revised as suggested: "In addition, the Flin Flon Technical Advisory Committee (TAC) is comprised of stakeholders knowledgeable about the local environment, health issues and risk assessment, <u>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 ."</u>
p.7-5 top para.	clarity - "...chemical-specific correlating factors based on historical paired TSP and PM10 data were used to predict concentrations of COC associated with the respirable PM <sub>10</sub> fraction - important to be clear and to note (p.4-5) that this only worked for As, Cd, Cu, and Pb. - <b>recommend stating that the Hg &amp; Se data for the PM<sub>10</sub> fractions for West Flin Flon is based on the Creighton data (Table 4-4 p. 4-6) with a fraction of 12.4 being used.</b>	Revision has been made as suggested. Edited paragraph (underlined text was added): "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 <u>various COC (i.e., arsenic, cadmium, copper, and lead</u> 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,

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		<p>whereas the HHRA evaluated exposure to COC associated with the PM<sub>10</sub> fraction. Although only TSP data is currently available for this location, chemical-specific correlating factors based on historical paired TSP and PM<sub>10</sub> data were used to predict concentrations of <u>arsenic, cadmium, copper, and lead</u> associated with the respirable PM<sub>10</sub> fraction (as described in detail in Appendix I).</p> <p><u>Added paragraph:</u> "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<sub>10</sub> 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."</p>
p.7-7 last sent	clarify - "...large amounts of local moose meat...may be subject to higher levels of mercury exposure than those who consume..." reference.	Revision made as suggested (i.e., added, "(see Table 4-16)."
<b>LEVEL OF COC IN CONSUMER PRODUCTS</b>		
p.7-8 1st sent, 1st para	Explain - "Background concentrations of the COC in consumer products were not elevated in the current assessment" - reference?	The report states that "Background concentrations of the COC in consumer products were not <u>evaluated</u> in the current assessment" not " <u>elevated</u> ".
p.7-8	readjust wording - "The use of lead-based paints was banned in the USA in 1978." Recommend this be made clear/re-worded - that the US in 1977 went to a 0.06% for lead-based paints, but it wasn't until Jan 1991 before Canada negotiated a voluntary lead-based level of 0.06% with the industry, and the 0.06% was not regulated until 2005. In 1976 Canada was at a 0.5% lead-based paint level for interior surfaces, furniture and surfaces frequented by children but the lead-based paint levels could be greater than 0.5% for exterior surfaces or interior surfaces not frequented by children [Hazardous Products Act (Surface Coating	Revision has been made as suggested.  Edited text: " A well known source of lead is the presence of lead-based paints in homes. <u>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</u>

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	Material Regulations, 2005 & Product Safety Hazardous Products (Liquid Coating Materials) Regulations, 1976].	<u>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 % (5000 mg/kg) to 0.06% (600 mg/kg) until 2005 (Government of Canada, 2005).</u> There are many homes in which this paint is still present... “
p.7-12 2nd para, 3rd sent	clarify - re: breast milk, not clear what model was actually used to assess MeHg in breast milk since as outlined on p. 7-12 to 7-14 since "...PBPK model for MeHg has not yet been recommended or recognised by suitable regulatory agencies (Health Canada, US EPA)"	A reference has been added for the approach used within Chapter 7 (Chiao and McKone, 1995).
p. 7-18, Table 7-4	clarify - 33% RAF for As was used in the HHRA, but p.22 bottom 4th paragraph of the review of the FF/Cr HHRA Vol I by IERP say 34% - is there a typo here or is it just the result of rounding off?	The HHRA originally used an RAF of 34% for arsenic. In response to comments provided by the IERP, a value of 33% was used in the revised HHRA.
p. 7-20 3rd sent.	clarify - "...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)..." seems to <b>contradict</b> the statement on bottom para last sent of p.7-5 which states "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." Please clarify.	The HHRA indicated that exposure to lead and inorganic mercury had the potential to result in elevated health risks and recommended that a biomonitoring study be completed to provide additional information on actual levels of exposure to these COC. Use of the soil ingestion rate of 100 mg/day would result in the same conclusions and recommendations.
<b>CHAPTER EIGHT</b>		
<b>ARSENIC</b>		
Table 8-2, Table 8-3	Typo - Creighton Arsenic HQ of 1.4 should be shaded.	Revision to Table 8-3 has been made as suggested. This comment is not applicable to Table 8-2.
p. 8-5 (also applies to p.4-24/4-25 Background) [also see p.5-13 comment]	RE: Arsenic - Drinking Water. It is important to be clear that the Ministry of Ontario water data was used for the Arsenic background data. This is important for Creighton because it seems that drinking water is the largest contributor for Arsenic exposure (p.5-14 Fig. 5-5), not inhalation, for the composite lifetime receptor. But it's important to put this into perspective since according Health Canada (see reference in the CDWQ Guidelines Technical Doc for Arsenic p. 5 re: Saskatchewan statement) the Saskatchewan background mean appears to be 3.0 vs. the ON 95% UCLM is 0.64. (Saskatchewan data provided)	The arsenic in background drinking water for Creighton has been corrected, and is now based on Saskatchewan-specific data. The HHRA indicates that exposure to arsenic <i>via</i> the consumption of drinking water in the Flin Flon-Creighton area is similar to exposure under the typical background scenario <i>via</i> this pathway.
p.8-5	Arsenic - given that air is the major contributor for WFF, EFF & CH why not make some comment, in addition to the urinary As study, that as was made for Cadmium that "...future	This note has been added to Chapter 8 as requested.

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	reduction in smelter-related emissions, which possibly may have a direct and immediate effect on reducing inhalation related exposure and risks"	
<b>CADMIUM</b>		
p.8-5 5th para; last sent & also p. (vi) 3rd sent	"That said, the ILCRs for cadmium are quite elevated..." However, suggest clarifying for Creighton - the ILCR cancer risk from air source is within the same order of magnitude at 4.5E-05, so for Creighton the risk according to p (v) would be very low and in light of p. 4-102,3rd para, 3rd sent, would Creighton fall into the category of having a "...re-evaluation of certain model parameters..conducted before the potential risk to health are fully characterized" - was this done for Creighton?	This comment is addressed above.
<b>LEAD</b>		
p. 8-6 Table 8-10	Typo - Col 1, row 2 "age categories (years)" mislabeled title?	Column title has been corrected as suggested. The title now reads " <i>Descriptive Statistics</i> "
p. 8-6	wording - Table 8-10 can add to the title the receptor group, assuming toddler?	These statistics are not for a particular age-group, but are calculated for age groups 0 to 7 years. The title has been updated to reflect this ("Blood Lead Concentrations in Children (0 to 7 Years) Predicted by the IEUBK Model (µg/dL)")
<b>INORGANIC MERCURY</b>		
p.8-7	wording ? - "in the blood, mercury has a short half-life thereby ..." please state for consistency with lead what the 1/2 life is?	Revision has been made as suggested, text updated to read "In the blood, mercury has a short half-life (3 days) thereby making it a useful medium for determining short-term exposures".
<b>PRGs and PTCs</b>		
p.8-8 1st sent, 2nd para.	clarify - "Although exposure to COC through the inhalation of particulates in air and from the consumption of drinking water may vary from community to community, these sources have a relatively minor contribution to the total intake." Please clarify given that for Arsenic ILCR for Creighton the major contributor is water and for Cadmium ILCR the major contributor is air..in other parts of the document.	Clarification has been provided.
p.8-12	clarify - Table 8-18 Creighton has 11 properties in total that exceed a PRG/PTC criteria, but add 4 for Pb and 10 for As get 14, but to clarify is there a couple properties that exceed both	The paragraph prior to Table 8-18 indicates that this table "provides the number of properties with concentrations that exceed the residential PTC for at least one COC".
<b>COMMUNITY EDUCATION</b>		
p. 8-13	In previous public messages (Heavy Metal Soil Study in Creighton and Flin Flon - Dec., 2007 Fact Sheet)the following two items were also included: (1) wear certain clothing only for outdoor play, work and gardening, and remove that clothing when going indoors and (2) clean stroller and bike wheels to avoid tracking soil indoors. For consistency can these be added to the HHRA document as well?	Based on other comments from the TAC, this section no longer lists recommendations for risk management other than a follow-up biomonitoring study and continued air monitoring.

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p.8-20 5th para, 2nd sent	" ...and children in this neighborhood had elevated levels of urinary arsenic. Temporary relocation of the children reduced their urinary arsenic levels to background, which confirmed sources related to their environment." Can you indicate the urinary arsenic levels in this study?	The speciated urinary arsenic level (8.6 ug/L) has been added as suggested.