PREFACE

The technical memo that follows provides preliminary estimates of human exposure and risk for total mercury (THg) and methylmercury (MeHg) associated with the consumption of Ringed Seal meat and liver in the Lower Churchill Hydroelectric Generation Project study area.

The memo was prepared by Dillon Consulting Ltd. as part of the project’s Human Health Risk Assessment, following baseline results of the project’s 2015 aquatic effects monitoring program and using results obtained in the dietary survey and human biomonitoring program conducted in Labrador communities throughout 2014-15. Data collected through the aquatic monitoring program showed elevated mercury concentrations in some seal liver samples, particularly in older specimens.

In response to this observation, the Lower Churchill Management Corporation requested a review – and the technical memo that follows – to determine if the elevated seal mercury measurements represent a risk to human health.

As noted in the technical memo, the review by Dillon Consulting concludes:

*Overall, while the consumption of seal meat and liver may contribute significantly to periodic acute MeHg and inorganic Hg exposure within the study area, there is no evidence that a potential acute human health risk is imminent or even likely. Thus, at this time, there is not believed to be a need to consider seal meat or liver consumption advisories in relation to potential acute MeHg and inorganic Hg exposures and risks. Although, it may be prudent to recommend that only younger seals be harvested for human consumption (as older seals tend to have higher THg concentrations in both muscle and liver tissue). (pg. 14)*

Results of the project’s aquatic effects monitoring and HHRA programs, as well as additional information about current mercury levels in the project area, are available on the Muskrat Falls Project website at [www.muskratfalls.nalcorenergy.com](http://www.muskratfalls.nalcorenergy.com).

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1 In fall 2014 and winter 2015 a baseline dietary survey and human hair sampling program was conducted for the Lower Churchill Project (LCP). The program was conducted in communities adjacent to the Churchill River, and was designed to identify food consumption habits among residents and to measure current mercury levels among those living in these areas.

1.0 Introduction

This technical memo provides preliminary estimates of human exposure and risk for total mercury (THg) and methylmercury (MeHg), that are associated with the consumption of Ringed Seal meat (muscle tissue) and liver in the Lower Churchill Hydroelectric Generation Project (LCHGP) study area.

As the HHRA program is in its relatively early stages, the exposure and risk estimates may change somewhat as further information becomes available on study area seal THg and MeHg tissue concentrations, and as refinements are made to key exposure and risk assumptions, such as:

- seal meat and liver consumption rates;
- the bioavailability and bioaccessibility of THg and MeHg in seal meat and/or liver, including consideration of literature on the effects of cooking methods on MeHg and THg bioaccessibility in fish and other aquatic animal foods (a number of papers indicate that cooking can substantially reduce the bioaccessibility and bioavailability of THg and MeHg to human consumers);
- the proportions of MeHg and inorganic mercury species (relative to THg) in seal meat and liver (i.e., mercury speciation); and,
- the influence of toxicological interactions between THg/MeHg and other key substances present in seal meat and liver, as well as other substances that are present in various food and beverage items common to Labrador Inuit diets, either naturally, or as contaminants (e.g., selenium, iron, zinc, PCBs, polyunsaturated fatty acids, sulfur-rich proteins and amino acids, and other sulfur-rich ligands, polyphenol compounds (such as in tea)).

As information on these items is currently in the process of being researched and reviewed (as part of the HHRA program), the preliminary exposure and risk estimates presented herein are conservative values that intentionally overestimate potential exposure and risk. Thus, they should not be viewed as definitive or representative estimates of exposure and risk at this time. Rather, they are simply indicators of potential exposure and risk.
2.0 Seal Consumption Patterns in the LCHGP Study Area

Data were obtained on seal meat and seal organ consumption patterns and habits in the study area communities (i.e., Churchill Falls, Happy Valley-Goose Bay, Mud Lake, Sheshatshiu, and North West River) in late 2014 to early 2015 (i.e., Baseline Dietary Survey (DS); reported in Golder, 2015). The community of Rigolet was not included in the baseline DS as the Nunatsiavut Government Research Advisory Committee did not approve the application that was filed by Golder Associates on behalf of Nalcor.

Roughly a third of DS respondents reported eating seal meat or organs. For those individuals that reported consumption of seal meat and/or organs, spring and winter were the seasons when consumption was most likely to occur, but consumption (albeit to a lesser extent) was also reported by some respondents in the summer and fall. Of the study area communities, the greatest percentage of respondents who reported seal meat and/or organ consumption were from the predominantly Inuit communities of Mud Lake and North West River. Very little seal meat and/or organ consumption was reported by DS respondents in Churchill Falls, Happy Valley-Goose Bay and the Innu community of Sheshatshiu. DS results showed that seal meat and/or organ consumption is limited almost entirely to adults, although some adolescent, child and toddler respondents reported infrequent consumption of seal meat or organs.

Seal meat and/or organ consumers in Mud Lake and North West River reported that most seal harvesting occurs in the Goose Bay area or from various locations within Lake Melville.

Seal preparation and cooking methods reported by respondents in the baseline DS suggest that seal meat (muscle tissue) is typically trimmed of skin and fat prior to cooking. Very few respondents reported eating seal meat (or organs) in a raw (uncooked) state. Seal meat was reported to be cooked primarily by boiling, frying and roasting. Seal organs were reported to be cooked primarily by frying, boiling and roasting. A very small number of respondents reported that seal organs are dried prior to consumption.

The majority of respondents who reported consuming seal meat and/or organs indicated a consumption frequency of much less than once per week. A very small number of respondents reported consumption frequencies that ranged from 1 to 3 times per week, and no respondents reported consumption of seal meat or organs 4 or more times per week. Based on reported seal meat and organ consumption frequency by DS respondents and information obtained from the DS on typical seal meat and/or organ serving sizes, Golder (2015) developed maximum and average consumption frequency and serving size statistics that can be used to estimate seal meat and seal organ consumption rates.

To ensure a conservative estimate of potential THg and MeHg exposure and risk from the consumption of seal meat and/or organs at this time, maximum estimates of consumption frequency across all four seasons (i.e., the number of times a respondent indicated they consume seal meat and/or organs in a given season) were used to develop consumption rates. For both seal meat and organs, this maximum frequency was 3 times per season, for each season of the year. Maximum reported seal meat and organ serving sizes from the DS were also used to develop the seal meat and organ consumption rates. The serving sizes represent how much is typically eaten on a per meal basis, when seal meat and/or organs
are consumed (as reported by DS respondents). These maximum serving sizes were as follows, for each relevant human life stage:

- Toddler – Seal meat (75 g)
- Toddler – Seal organs (0 g; DS toddler respondents had no reported consumption of seal organs)
- Child – Seal meat (150 g)
- Child – Seal organs (75 g)
- Adolescent – Seal meat (225 g)
- Adolescent – Seal organs (225 g)
- Adult – Seal meat (300 g)
- Adult – Seal organs (300 g)

Infants are excluded as this life stage does not consume seal meat or organs.

Consumption rates for human health risk assessment (HHRA) purposes are typically expressed as a quantity (mg, g, kg, etc.) consumed per day. To develop consumption rates for seal meat and/or organs for each relevant human life stage, the maximum consumption frequency was expressed on a per day basis (i.e., 3 times a season equals 12 times per year). Assuming that consumption events occur on different days of the year, it was assumed that seal meat and/or organs are consumed on 12 days of the year. To express this consumption frequency on a per day basis, 12 days per year is divided by the total number of days in a year (365) to yield a daily unitless amortized consumption frequency of \( \frac{12}{365} = 0.033 \). It was also assumed that seal meat and/or organ serving sizes (as noted above) correspond to the amount that is ingested per each day that these food types are consumed.

The HHRA program is in the process of researching and reviewing literature on seal meat and organ consumption rates reported in other arctic and subarctic regions of the world. The outcome of this literature review effort will help determine whether or not the consumption rates based on the DS outcomes appear reasonable and generally consistent with what others have reported. It is possible that consumption rates based on the DS outcomes could be refined or modified to some degree, if deemed necessary and supported by the scientific literature that is reviewed.

### 3.0 Seal Muscle (Meat) and Liver Concentrations

The LCHGP aquatic environmental effects monitoring program (EEMP) has been collecting data on THg concentrations in ringed seal (*Pusa hispida*) muscle tissue since 2011 (N=129) and THg in ringed seal liver tissue since 2012 (N=115). Data on MeHg concentrations in muscle tissue started to be collected in 2015 (N=10, currently). Data are also collected on seal weight, length, blubber thickness (since 2013), and age. To date, mostly young seals have been sampled (<1 year) but seal ages have ranged up to 22 years.

Summary statistics for muscle and liver THg concentrations are provided in Table 1.
Table 1 Seal Muscle and Liver THg Concentration Summary Statistics

<table>
<thead>
<tr>
<th>Statistic</th>
<th>Seal Muscle THg</th>
<th>Seal Liver THg</th>
</tr>
</thead>
<tbody>
<tr>
<td>N (# of samples)</td>
<td>129</td>
<td>115</td>
</tr>
<tr>
<td>Detection Frequency - # of samples &gt;RDL (%)</td>
<td>44.2</td>
<td>96.5</td>
</tr>
<tr>
<td>Minimum Concentration (mg/kg ww)</td>
<td>0.01</td>
<td>0.04</td>
</tr>
<tr>
<td>Maximum Concentration (mg/kg ww)</td>
<td>6.3</td>
<td>110</td>
</tr>
<tr>
<td>Median (50\textsuperscript{th} Percentile) Concentration (mg/kg ww)</td>
<td>0.05</td>
<td>0.29</td>
</tr>
<tr>
<td>Arithmetic Mean Concentration (mg/kg ww)</td>
<td>0.16</td>
<td>3.57</td>
</tr>
<tr>
<td>Standard Deviation (mg/kg ww)</td>
<td>0.58</td>
<td>12.8</td>
</tr>
<tr>
<td>Standard Error of Arithmetic Mean (mg/kg ww)</td>
<td>0.051</td>
<td>1.2</td>
</tr>
<tr>
<td>Coefficient of Variation (unitless)</td>
<td>3.5</td>
<td>3.6</td>
</tr>
<tr>
<td>75\textsuperscript{th} Percentile (mg/kg ww)</td>
<td>0.11</td>
<td>0.55</td>
</tr>
<tr>
<td>90\textsuperscript{th} Percentile (mg/kg ww)</td>
<td>0.23</td>
<td>8.7</td>
</tr>
<tr>
<td>95\textsuperscript{th} Percentile (mg/kg ww)</td>
<td>0.37</td>
<td>17.8</td>
</tr>
<tr>
<td>Data distribution type</td>
<td>Non-parametric (no discernible distribution type)</td>
<td>Non-parametric (no discernible distribution type)</td>
</tr>
</tbody>
</table>

While seal muscle MeHg data are limited (N=10), these data suggest that almost all of the THg present in seal muscle tissue is in the form of MeHg. This finding is consistent with what is commonly reported in the scientific literature. Thus, for preliminary exposure and risk calculation purposes, it is assumed all of the mercury present in seal muscle tissue (100%) is MeHg.

Preliminary correlation analysis conducted on the seal muscle and liver THg data indicates the following:

- Muscle and liver THg concentrations in seals have a strong positive correlation (r=0.84).
- Muscle THg and seal age have a moderate positive correlation (r=0.50).
- Liver THg and seal age have a moderate positive correlation (r=0.58).
- Muscle THg and seal weight have a weak positive correlation (r=0.49).
- Liver THg and seal weight have a moderate positive correlation (r=0.62).
- Muscle THg and seal length have a weak positive correlation (r=0.40).
- Liver THg and seal length have a moderate positive correlation (r=0.51).

As is expected and commonly reported for seal mercury tissue concentrations, the relationships between muscle and liver THg concentrations, and between muscle and liver THg concentrations and seal weight and length, appear to be linear and positively correlated.

Human health risk assessments (HHRAs) that evaluate country food consumption do not generally assume that maximum concentrations represent typical or expected exposure. Clearly, the study area seal muscle and liver THg concentrations are highly variable, which suggests it is not reasonable to
assume a maximum or even an upper bound concentration represents typical or expected exposure. HHRAs commonly estimate exposure point concentrations (EPCs) for chemicals in the media of interest that reflect the variability and dispersion in the data, and that also provide an upper estimate of central tendency within the data. It is widely recognized in numerous North American regulatory jurisdictions that the most appropriate statistic for an EPC in a HHRA is the upper 95% confidence limit on the arithmetic mean (UCLM95). The UCLM95 is sometimes termed the “true mean”, or “the concentration most likely to be contacted over time”.

It is important in any human exposure and risk calculations to ensure that calculated UCLM95 values are accurate and statistically robust. Thus, to derive the UCLM95 EPCs for seal muscle and liver THg concentrations, the U.S. EPA computer program, ProUCL™ Version 5.0 was used. The U.S. EPA strongly recommends the use of ProUCL when calculating EPCs for use in HHRAs. ProUCL determines the most appropriate UCLM95 value for a dataset, given its distribution and characteristics. A number of statistically valid methods to calculate a UCLM95 can be run simultaneously, with the program recommending the most appropriate or statistically robust value(s) to select. However, according to its user guidance, ProUCL can only determine robust and reliable UCLM95 values if the sample size is at least eight (8). As the sample size for THg concentrations within the seal muscle and liver datasets is much larger than this (i.e., N=129 for muscle tissue; N=115 for liver tissue), it was possible to calculate adequately robust UCLM95 EPCs with a high degree of confidence.

In calculating the UCLM95-based EPCs for THg in seal muscle and liver, the following tasks/conditions were conducted/applied, all of which tend to bias the UCLM95-based EPCs high:

- For any seal muscle or liver samples with analytical results for THg below the laboratory reported detection limits (i.e., <RDLs), the <RDL values were assumed to equal the RDL.
- Data quality was verified prior to calculating UCLM95-based EPCs.
- As the measured THg concentrations in seal muscle and liver samples represent potential concentrations that human receptors could be exposed to, no attempt was made to conduct statistical outlier tests to remove extreme values (high or low) from the seal muscle and liver chemistry datasets. Thus, the EPC calculations for THg included the presence of potential extreme values.
- As the calculated options for a UCLM95 generated by ProUCL 5.0 can vary considerably (as a function of the underlying assumptions in the statistical models, and the data distribution type), some degree of professional judgment is typically necessary in selecting the most appropriate UCLM95 value for use as the EPC. Key considerations often include the data distribution type, the significance level associated with the UCLM95 calculation methods (i.e., ProUCL-recommended values are not always at the 95% significance level), any warnings generated by the ProUCL 5.0 software, and the magnitude of the calculated UCLM95 options.

The UCLM95-based seal muscle and liver THg EPCs are 0.39 mg/kg ww and 8.8 mg/kg ww, respectively. Both EPCs are a 95% Chebyshev (Mean, Sd) UCL, which reflects non-parametric UCLM95 estimation approaches. As noted, these EPC values represent upper central tendency for seal THg concentrations in muscle and liver tissues and are conservative estimates of potential exposure (i.e., the UCLM95 for THg in muscle is similar to the 95th percentile, and the UCLM95 for THg in liver is similar to the 90th percentile; Table 1). ProUCL 5.0 statistical output for the UCLM95-based EPCs is provided in Appendix A.
4.0 MeHg Proportions in Seal Muscle and Liver Tissue

As noted above, the limited seal muscle MeHg data collected to date suggests that almost all of the THg present in seal muscle tissue is in the form of MeHg. This finding is consistent with what is commonly reported in the scientific literature. Thus, for exposure and risk calculation purposes, it is assumed that all (100%) of the mercury present in seal muscle tissue is MeHg.

In seal liver, a number of studies have consistently reported that most of the mercury is not present as MeHg, but rather, occurs as inorganic Hg forms (e.g., Lemire et al., 2015; Eaton et al., 1980; Dietz et al., 1990; Cappon and Smith, 1981; Wagemann et al., 2000). In these studies, the reported range for MeHg content, out of the THg content present in seal liver, is <2% to 12%. Older seals tend to have a lower MeHg proportion in their livers than younger seals (Wagemann et al., 1998). A recent human health risk assessment conducted for the Nunavik region of Quebec (Lemire et al., 2015) conservatively assumed that 25% of the THg present in seal liver is MeHg. This is likely a substantial overestimate based on the literature reviewed to date.

A number of these studies also report that the Hg present in seal liver is not readily bioavailable (i.e., not readily absorbed) when consumed by humans or experimental animals (Eaton et al., 1980; Lemire et al., 2015; Clarkson, 2002; Ikemoto et al., 2004; Lemes et al., 2011; Wagemann et al., 1998). This is believed to be a function of liver Hg being bound tightly to selenium complexes, as well as MeHg demethylation processes that occur in the liver of mammalian species. Many of these same studies note that Hg-Se complexes are poorly absorbed in the human intestine, relative to MeHg.

For preliminary exposure and risk calculation purposes, it was conservatively assumed that the MeHg proportion of the THg present in seal liver is 20%, with the remaining 80% comprised of inorganic Hg forms.

The HHRA program continues to research and review the literature on Hg speciation in seal liver. It is possible that the current 20% MeHg assumption will be adjusted as additional studies are reviewed. The program is also considering collection of study area-specific data on the MeHg proportion of THg in seal liver.

5.0 Oral Bioavailability and Bioaccessibility of THg and MeHg in Seal Meat/Organs

The HHRA program is in the process of researching and reviewing studies on Hg bioaccessibility and bioavailability from fish and other aquatic animal-based food items, including seals. Bioaccessibility refers to the amount of a substance that is solubilized in the human gastrointestinal tract and available for absorption. Bioavailability refers to the quantity of the bioaccessible fraction that actually gets absorbed into the systemic circulation. Thus, the bioaccessibility of a chemical within food sets an upper limit on bioavailability. References for such studies will be provided in the HHRA documentation. In general, a number of studies have found that the gastrointestinal absorption of MeHg and inorganic Hg in fish or other aquatic animal foods (such as marine mammals and shellfish) is typically much lower than 100%, which is the common default assumption in HHRA. THg bioaccessibility (which includes MeHg bioaccessibility) from various fish and shellfish foods has been reported to range from 9% to 83%. Variability is high depending on the fish species and its trophic position and location (which influences the Hg speciation profile in tissues).
A recent study reports that the gastrointestinal bioavailability of THg (which includes the MeHg proportion) in seal liver, measured using an **in vitro** model that simulates the physiological conditions of the human gastrointestinal tract, was <25% (Laird, personal communication, Cited in Lemire et al., 2015). Laird et al., (2009) previously reported that the **in vitro** bioaccessibility of THg in ringed seal liver was 19%. Hg bioaccessibility information on seal meat has not been identified to date, and no other data on the bioaccessibility or bioavailability of MeHg or THg in seal tissues has been identified at this time.

It is well established in the literature that inorganic Hg compounds (which, as noted above, dominate in seal liver tissue) are poorly absorbed in humans and in experimental animals. Estimates of gastrointestinal absorption of inorganic Hg range up to 15% in humans and from 3% to 40% in experimental animals (ATSDR, 1999). The variability is largely dependent on the animal species tested and on the different Hg dosing vehicles and dosing approaches used in the studies.

A number of studies have also found that cooking methods can substantially alter the bioaccessibility and bioavailability of all forms of Hg in food items. Numerous studies also report that certain foods and beverages, that are often consumed with fish and other aquatic animal food items (such as tea, coffee, certain grains), can substantially reduce the amount of MeHg and other Hg forms that are absorbed from the gastrointestinal tract into the systemic circulation (i.e., reduce the oral bioaccessibility and bioavailability of Hg).

The findings from these types of studies may have profound impacts on the exposure and risk estimates for MeHg and inorganic Hg in the HHRA. At the very least, these studies will provide important context and perspective on calculated MeHg and inorganic Hg exposure and risk estimates.

While it is premature to select definitive oral relative absorption factors (RAFs) for use in the HHRA at this time, it is clear from the literature reviewed to date that the gastrointestinal absorption of MeHg and other forms of Hg present in seal meat and/or liver is not complete and is likely considerably lower than the gastrointestinal absorption of MeHg and inorganic Hg that occurred in the principal studies upon which current human toxicological reference values (TRVs) are based. Thus, conservative interim oral RAFs were applied to the seal meat and liver MeHg and THg preliminary exposure estimates, as follows:

- For seal muscle (meat), where it is assumed all Hg is present as MeHg, the interim RAF=1.0.
- For seal liver, the interim RAF=0.25.

These interim RAF estimates represent the upper end of MeHg and THg gastrointestinal bioavailability reported in the literature.

Consideration may be given to conducting some study area specific THg and/or MeHg bioaccessibility tests on seal muscle and liver samples and potentially on selected fish muscle samples too. However, regulatory acceptance of bioaccessibility testing in Canada is limited at this time, due to limited validation of many of the test protocols that are applied within the scientific literature. Thus, prior to conducting such tests, Nalcor will consult with Health Canada on their level of comfort and acceptance with food bioaccessibility testing and its application to human health risk assessment.
6.0 Human Toxicological Reference Values (TRVs) for MeHg and Inorganic Hg

Human health risk is calculated by dividing estimated exposure by the applicable TRV(s). Published TRVs for both MeHg and inorganic Hg compounds, from a number of North American and European regulatory agencies, were compiled and reviewed. Based on this review, the following TRVs were applied at this time. These TRVs are the most protective of those currently available for both MeHg and inorganic Hg.

MeHg TRV: Oral reference dose of 0.0001 mg MeHg/kg body weight/day (U.S. EPA, 2001a,b; NRC, 2000). This TRV is based on neurological and neurodevelopmental effects observed in human epidemiological studies of populations that consume large amounts of fish and marine mammals, and where children were exposed in utero to MeHg exposure incurred by their mothers. The oral RfD is applicable to lifetime daily exposure for all populations including sensitive subgroups or subpopulations (which includes women of child-bearing age, pregnant women and the developing fetus)( U.S. EPA, 2001a).

Inorganic Hg (represented by mercuric chloride) TRV: Tolerable daily intake and oral reference dose of 0.0003 mg Hg/kg body weight/day (Health Canada, 2010a; U.S. EPA, 1995). This TRV is based on renal (kidney) and autoimmune effects in orally exposed rats.

Pending further review of human TRVs for MeHg and inorganic Hg, the TRVs used in the HHRA may be refined somewhat from those applied herein. The HHRA may also use a range of TRVs to provide context on human health risk estimates. This is a common approach in HHRAs where multiple TRVs for a substance exist from multiple regulatory agencies, and all are scientifically defensible values.

7.0 Preliminary MeHg and THg Chronic Exposure and Risk Calculations for Seal Meat and Seal Liver Consumption

The general equation used for calculating estimated exposures to MeHg and inorganic Hg in seal meat and liver is as follows. This equation is based on that provided within Health Canada (2010b).

\[
EXP_{SEAL} = \left[ CR \times CF \times C_{SEAL} \times P_{Hg} \times RAF_{ORAL} \right] / BW
\]

Where:

- \( EXP_{SEAL} \) = MeHg or inorganic Hg exposure via the consumption of seal meat and/or seal liver (mg/kg body weight/day).
- \( C_{SEAL} \) = concentration of MeHg or inorganic Hg in seal meat and/or liver (mg/kg ww); The UCLM95-based seal muscle and liver THg EPCs are 0.39 mg/kg ww and 8.8 mg/kg ww, respectively.
- \( CR \) = consumption rate (kg ww of seal meat or liver eaten per serving (per day)); assumptions were as follows:
  - Toddler – Seal meat (0.075 kg ww/d)
Toddler – Seal organs (0 kg ww/d; DS toddler respondents had no reported consumption of seal organs)
Child – Seal meat (0.150 kg ww/d)
Child – Seal organs (0.075 kg ww/d)
Adolescent – Seal meat (0.225 kg ww/d)
Adolescent – Seal organs (0.225 kg ww/d)
Adult – Seal meat (0.300 kg ww/d)
Adult – Seal organs (0.300 kg ww/d)

**CF** = consumption frequency (days per year when consumption occurs); i.e., 12/365=0.033.

**P**

**Hg** = proportion of MeHg or inorganic Hg in seal meat and/or liver; assumed that all (100%) of the Hg present in seal muscle tissue is MeHg; conservatively assumed that the MeHg proportion of the THg present in seal liver is 20%, with the remaining 80% comprised of inorganic Hg forms.

**RAF** = oral relative absorption factor; for seal muscle (meat), it is assumed all Hg is present as MeHg and that the interim RAF=1.0; for seal liver, the interim RAF=0.25.

**BW** = body weight (kg); at this time, generic female age class body weight values have been taken from the Canadian Exposure Factors Handbook (Richardson and Stantec, 2013); female receptor body weights are used as females tend to incur a higher dose than males due to having lower body weights; also, pregnant females and their developing fetuses are well known to be the most potentially vulnerable human receptors to the neurodevelopmental effects of MeHg.

Toddler: 14.8 kg
Child: 34.7 kg
Adolescent: 61 kg
Adult: 69.8 kg

**ww** = wet weight

Table 2 presents chronic exposure and risk estimates for MeHg (from consumption of seal meat and liver) and inorganic Hg (from consumption of seal liver). Health risk estimates are expressed both as hazard quotients (i.e., HQ=exposure estimate / TRV) and as the % of the TRV that is taken up by the estimated exposure. The latter approach is commonly used when estimating risk from the consumption of food items (e.g., Health Canada, 2007).
<table>
<thead>
<tr>
<th>Receptor Age Class</th>
<th>Female Toddler</th>
<th>Female Child</th>
<th>Female Adolescent</th>
<th>Female Adult</th>
</tr>
</thead>
<tbody>
<tr>
<td>Seal meat MeHg exposure (mg/kg BW/d)</td>
<td>0.000065</td>
<td>0.000055</td>
<td>0.000047</td>
<td>0.000055</td>
</tr>
<tr>
<td>Estimated Risk (HQ; % of TRV)</td>
<td>0.65; 65%</td>
<td>0.55; 55%</td>
<td>0.47; 47%</td>
<td>0.55; 55%</td>
</tr>
<tr>
<td>Seal liver MeHg exposure (mg/kg BW/d)</td>
<td>0</td>
<td>0.000031</td>
<td>0.000053</td>
<td>0.000062</td>
</tr>
<tr>
<td>Estimated Risk (HQ; % of TRV)</td>
<td>0</td>
<td>0.31; 31%</td>
<td>0.53; 53%</td>
<td>0.62; 62%</td>
</tr>
<tr>
<td>Total MeHg exposure if seal meat and liver are consumed together</td>
<td>0.000065</td>
<td>0.000087</td>
<td>0.0001</td>
<td>0.00012</td>
</tr>
<tr>
<td>Estimated Risk (HQ; % of TRV)</td>
<td>0.65; 65%</td>
<td>0.87; 87%</td>
<td>1.01; 101%</td>
<td>1.17; 117%</td>
</tr>
<tr>
<td>Seal liver inorganic Hg exposure (mg/kg BW/d)</td>
<td>0</td>
<td>0.00013</td>
<td>0.00021</td>
<td>0.00025</td>
</tr>
<tr>
<td>Estimated Risk (HQ; % of TRV)</td>
<td>0</td>
<td>0.42; 42%</td>
<td>0.71; 71%</td>
<td>0.83; 83%</td>
</tr>
</tbody>
</table>

The preliminary chronic exposure and risk estimates summarized in **Table 2** only account for seal meat and liver consumption, and do not account for locally caught fish consumption, the consumption of other country foods of aquatic origin, or MeHg and THg exposures that would occur from the consumption of foods purchased at grocery stores. The HHRA will account for all of these potential sources of MeHg and inorganic Hg exposure (MeHg in grocery store foods is typically limited to fish/shellfish and potentially some other meat products; it is not generally present in foods that are of plant origin or highly processed).

Given that only seal meat and liver consumption are accounted for at this time, the preliminary chronic exposure and risk estimates presented in **Table 2** are elevated, and suggest there could be potential health concerns associated with MeHg and inorganic Hg concentrations in seal meat and liver tissues. However, this must be balanced against the conservative assumptions and approaches used to estimate exposure and risk. All assumptions and approaches that were used intentionally overestimate exposure and risk. In particular, the maximum reported seal meat and liver consumption frequency (from the DS) was assumed. If the preliminary chronic exposure and risk calculations instead use the average reported seal meat and liver consumption frequency (i.e., most consumption rates and other intake rates used in HHRA are based on reported averages (arithmetic means)), all exposure and risk estimates drop by...
approximately 3-fold, and indicate a much lower potential concern. For example, the highest HQ value for the adult exposed to MeHg from consuming seal meat and liver together (1.17) drops to roughly 0.4 when the average (rather than the maximum) seal meat and liver consumption frequency is assumed. This HQ value of 0.4 would allow for 60% of MeHg exposure to come from sources other than seal meat and/or liver, before the TRV is exceeded. Furthermore, results of the hair sampling program in the communities where seal meat/liver consumption was reported did not show hair Hg concentrations that are suggestive of elevated exposure to MeHg from consuming country foods of aquatic origin.

Although the preliminary chronic exposure estimates suggest that the consumption of seal meat and liver may contribute significantly to MeHg and inorganic Hg exposure within the study area, they do not indicate that a potential human health risk is imminent or likely. Thus, at this time, it is not believed to be necessary to consider consumption advisories for Ringed Seal, though it may be prudent to recommend that only younger seals be harvested for human consumption (as older seals tend to have higher THg concentrations in both muscle and liver tissue). The HHRA will provide more refined estimates of exposure and risk that will include all major sources of MeHg exposure, and the ongoing monitoring program will continue to provide data that enables tracking of MeHg and THg concentrations in seal meat and liver. It is also noted that the need for risk management measures, such as consumption advisories, is not entirely dependent on HHRA outcomes. While HHRA outcomes and/or other risk-based approaches are clearly important in determining if consumption advisories are warranted, the need for advisories (and the form that advisories take) typically considers other factors too, such as the health and nutritional benefits of consuming a certain food item, the social and cultural benefits of consuming a certain food item, and various practical and economic considerations as well. For any future potential consumption advisories related to MeHg in the study area, the literature on toxicological interactions of MeHg with other common dietary substances should also be considered.

8.0 Preliminary MeHg and THg Acute Exposure and Risk Calculations for Seal Meat and Seal Liver Consumption

The preliminary exposure and risk calculations summarized in Table 2 assume that exposures occur over a chronic (long term) duration, which is a traditional HHRA practice. However, the reported consumption patterns for seal meat and seal liver within the study area are much more suggestive of an acute rather than a chronic exposure scenario, as both seal meat and seal liver are consumed episodically (i.e., in single or few events) and infrequently throughout the year.

For an acute exposure scenario, exposure and risk estimates that are amortized over prolonged periods may not be accurate or sufficiently meaningful. When exposure events are not frequent or continuous, the body can generally metabolize and eliminate the accumulated dose between exposure events (which is less likely to occur under conditions of chronic exposure). Also, there may be different types of effects that are of concern between chronic and acute exposure conditions. The ability of any chemical to cause toxicity is a function of both the exposure conditions (including the timing and duration of exposure) as well as the toxicological properties of the chemical. Many chemicals have different effects/endpoints that are of interest or concern under conditions of acute or short term exposure, relative to those that are of interest/concern under conditions of longer term or chronic exposure. Acute toxicity endpoints tend to be more severe and rapid onset (e.g., dizziness, loss of consciousness, respiratory irritation, vomiting and other gastrointestinal distress) than chronic endpoints, which are often more subtle effects that take more time to manifest (e.g., enzyme and other...
biochemical changes, cancer, organ lesions, impaired neurological function, developmental effects, weight loss etc.). In summary, there can be different effects that are of concern when exposure is acute, relative to when exposure is chronic.

However, there are some barriers or limitations to assessing acute exposure events or scenarios in HHRAs. The single biggest limitation is that most available regulatory human health-based TRVs are based on chronic exposure durations and chronic effects. Acute or short term TRVs (expressed as a dose) do not exist for the vast majority of chemicals, which often limits the ability of HHRAs to reliably assess acute exposures and risks. It is generally believed though that focusing on chronic effects is conservative and protective of those effects that may occur under acute exposure conditions. For most chemicals, substantially higher exposures can be tolerated without adverse effects for short or acute durations, while lower exposures can produce adverse effects when the exposure is continuous and/or occurs over prolonged durations. While this premise is generally considered reasonable and is widely accepted, some caution is warranted with respect to categorically assuming that all chronic TRVs will also protect against all potential acute effects. Ideally, different TRVs for both acute and chronic effects would exist for all chemicals evaluated in HHRAs, but this is not the case at this time.

With respect to MeHg and inorganic Hg, an acute regulatory oral TRV was identified for inorganic Hg only (i.e., ATSDR, 1999 acute minimal risk level (MRL) of 0.007 mg/kg BW/d; based on a 14 day rat study where the rats displayed adverse kidney effects following oral exposure to mercuric chloride in deionized water). No acute oral TRV was identified for MeHg. However, acute MeHg (and inorganic Hg) toxicity data were identified in the literature from human poisoning case reports and experimental animal studies. The range of these acute toxicity values was compared to the estimated acute exposures for MeHg and inorganic Hg resulting from the consumption of seal meat and liver. This was also conducted for acute inorganic Hg exposures from seal liver ingestion, in addition to comparing these exposure estimates against the acute oral MRL.

To estimate acute exposures to MeHg and inorganic Hg from the consumption of seal meat and liver, the same equation provided in Section 7.0 was used, but with the CF term removed. This adjustment results in calculation of the daily dose received per exposure event, rather than the amortized daily dose received over a year. In other words, the adjusted equation yields the estimated MeHg and inorganic Hg exposure on a single day when seal meat and/or liver are consumed.

Table 3 presents acute exposure estimates for MeHg (from consumption of seal meat and liver) and acute exposure and risk estimates for inorganic Hg (from consumption of seal liver). Health risk estimates are expressed both as hazard quotients (i.e., HQ=exposure estimate / TRV) and as the % of the TRV that is taken up by the estimated exposure.
### Table 3
Summary of Preliminary Acute Exposure Estimates for MeHg (from the consumption of seal meat and liver) and Acute Exposure and Risk Estimates for Inorganic Hg (from the consumption of seal liver)

<table>
<thead>
<tr>
<th>Receptor Age Class</th>
<th>Female Toddler</th>
<th>Female Child</th>
<th>Female Adolescent</th>
<th>Female Adult</th>
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<tbody>
<tr>
<td>Seal meat MeHg exposure (mg/kg BW/d)</td>
<td>0.0020</td>
<td>0.0017</td>
<td>0.0014</td>
<td>0.0017</td>
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<tr>
<td>Estimated Risk (HQ; % of TRV)</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
</tr>
<tr>
<td>Seal liver MeHg exposure (mg/kg BW/d)</td>
<td>0</td>
<td>0.00095</td>
<td>0.0016</td>
<td>0.0019</td>
</tr>
<tr>
<td>Estimated Risk (HQ; % of TRV)</td>
<td>0</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
</tr>
<tr>
<td>Total MeHg exposure if seal meat and liver are consumed together</td>
<td>0.0020</td>
<td>0.0026</td>
<td>0.0031</td>
<td>0.0036</td>
</tr>
<tr>
<td>Estimated Risk (HQ; % of TRV)</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
<td>NC</td>
</tr>
<tr>
<td>Seal liver inorganic Hg exposure (mg/kg BW/d)</td>
<td>0</td>
<td>0.0038</td>
<td>0.0065</td>
<td>0.0076</td>
</tr>
<tr>
<td>Estimated Risk (HQ; % of TRV)</td>
<td>0</td>
<td>0.54; 54%</td>
<td>0.93; 93%</td>
<td>1.08; 108%</td>
</tr>
</tbody>
</table>

**Notes:**
NC = not calculated as no acute duration TRV was identified for MeHg.

Similar to the preliminary chronic exposure and risk estimates summarized in Table 2, the acute exposure and risk estimates in Table 3 also only account for seal meat and liver consumption, and do not address other potential sources of MeHg or inorganic Hg exposure. For acute exposure and risk estimates though, this is less of an issue as it is possible and even likely that there may not be significant exposure from other sources on single individual days when seal meat and/or liver is consumed.

Similar to what was noted for preliminary chronic exposure and risk estimates, the preliminary acute exposure and risk estimates also appear to be elevated, and suggestive of potential human health concerns. However, all assumptions and approaches that were used to estimate acute exposures intentionally overestimate the exposure (as per the assumptions and approaches for the preliminary chronic exposure and risk estimates).

To help put the estimated acute risks for inorganic mercury from seal liver consumption into context, it is noted that the ATDSR acute MRL is based on a dose (0.93 mg/kg BW/d) that did not cause adverse kidney effects in rats (i.e., the no-observed-adverse effect level, or NOAEL; higher doses in the principal
study (up to 15 mg/kg BW/d) only produced mild to moderate kidney effects), with a 100-fold uncertainty factor applied to it. Thus, exceedance of this acute TRV does not mean that acute risks or adverse health effects should be expected. Rather, a low margin of exceedance over this conservative TRV (as occurs for the adult receptor in Table 3) simply implies that the safety factor beyond the NOAEL is reduced. Exposures would likely need to be at least two orders of magnitude (100 times) higher than those estimated in Table 3 in order for acute adverse effects to have a reasonable likelihood to occur in humans.

Further context is provided by examining the range of acute toxicity values reported in the literature for MeHg and inorganic Hg, and comparing the estimated acute exposures from Table 3 to these ranges. This information is preliminary at this time as a number of papers and regulatory agency documentation are in the process of being obtained and reviewed. However, review of the oral acute toxicity literature to date, for both MeHg and inorganic Hg, suggests that the estimated exposures in Table 3 are generally two to three orders of magnitude (100 to 1000 times) lower than doses that resulted in clear evidence of adverse effects in human poisoning case studies and in acute experimental animal toxicological studies. Based on information reviewed to date, acute oral exposure to MeHg and inorganic Hg can result in a variety of effects in both humans and experimental animals. Reported signs and symptoms of acute oral exposure may include: various renal (kidney) effects; various neurological and neurobehavioural effects; gastrointestinal effects; cardiovascular effects; thyroid hormone changes; lowered thymus weight; body weight changes; various enzyme level and activity changes; hematological changes; reproductive and developmental effects; and, immunological effects. The severity of these effects tends to increase with increasing dose, but variability is high depending on the chemical form of Hg that was administered, test species, test duration, and various test conditions such as dosing regimen and dosing approaches.

Potential acute effects associated with MeHg and inorganic Hg presence in seal meat and liver will continue to be tracked and further evaluated. The HHRA will refine estimates of potential exposure and risk associated with acute oral exposure to MeHg and inorganic Hg, and the ongoing monitoring program will continue to collect data on THg and MeHg in seal meat and THg in liver.

Overall, while the consumption of seal meat and liver may contribute significantly to periodic acute MeHg and inorganic Hg exposure within the study area, there is no evidence that a potential acute human health risk is imminent or even likely. Thus, at this time, there is not believed to be a need to consider seal meat or liver consumption advisories in relation to potential acute MeHg and inorganic Hg exposures and risks. Although, it may be prudent to recommend that only younger seals be harvested for human consumption (as older seals tend to have higher THg concentrations in both muscle and liver tissue).
9.0 References Cited


Appendix A: ProUCL Version 5.0 UCLM95-Based EPC Calculations for Ringed Seal Muscle and Liver THg Concentrations
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#### Normal GOF Test

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  - Shapiro Wilk GOF Test: Data Not Normal at 5% Significance Level
- Lilliefors Test Statistic: 0.394408
  - Lilliefors GOF Test: Data Not Normal at 5% Significance Level

#### Gamma GOF Test

- A-D Test Statistic: 16.03465
  - Anderson-Darling Gamma GOF Test: Data Not Gamma Distributed at 5% Significance Level
- K-S Test Statistic: 0.267525
  - Kolmogrov-Smirnov Gamma GOF Test: Data Not Gamma Distributed at 5% Significance Level

#### Lognormal GOF Test

- Shapiro Wilk Test Statistic: 0.859582
  - Shapiro Wilk Lognormal GOF Test: Data Not Lognormal at 5% Significance Level
- Lilliefors Test Statistic: 0.231471
  - Lilliefors Lognormal GOF Test: Data Not Lognormal at 5% Significance Level
- Data Not Lognormal at 5% Significance Level
Minimum of Logged Data -4.60517  Mean of logged Data -2.59661
Maximum of Logged Data 1.84055  SD of logged Data 0.933096

Assuming Lognormal Distribution
95% H-UCL 0.137327  90% Chebyshev (MVUE) UCL 0.147994
95% Chebyshev (MVUE) UCL 0.163108  97.5% Chebyshev (MVUE) UCL 0.184087
99% Chebyshev (MVUE) UCL 0.225294

Nonparametric Distribution Free UCL Statistics
Data do not follow a Discernible Distribution (0.05)

Nonparametric Distribution Free UCLs
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95% Standard Bootstrap UCL 0.246555  95% Bootstrap-t UCL 0.454851
95% Hall's Bootstrap UCL 0.524623  95% Percentile Bootstrap UCL 0.257442
95% BCA Bootstrap UCL 0.323258
90% Chebyshev(Mean, Sd) UCL 0.317466  95% Chebyshev(Mean, Sd) UCL 0.386616
97.5% Chebyshev(Mean, Sd) UCL 0.482593  99% Chebyshev(Mean, Sd) UCL 0.671122

Suggested UCL to Use
95% Chebyshev (Mean, Sd) UCL 0.386616

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). However, simulations results will not cover all Real World data sets. For additional insight the user may want to consult a statistician.
Ringed Seal Liver UCLM95 EPC

General Statistics on Uncensored Full Data

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Full Precision: ON

From File: WorkSheet.xls

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- Shapiro Wilk GOF Test: Data Not Normal at 5% Significance Level
- Lilliefors Test Statistic: 0.08262
- Lilliefors GOF Test: Data Not Normal at 5% Significance Level
- Data Not Normal at 5% Significance Level

Gamma GOF Test

- A-D Test Statistic: 19.53564
- Anderson-Darling Gamma GOF Test: Data Not Gamma Distributed at 5% Significance Level
- K-S Test Statistic: 0.347066
- Kolmogrov-Smirnoff Gamma GOF Test: Data Not Gamma Distributed at 5% Significance Level
- Data Not Gamma Distributed at 5% Significance Level

Assuming Normal Distribution

- 95% Normal UCL: 3.568957
- 95% Student's-t UCL: 5.550686
- 95% UCLs (Adjusted for Skewness): 6.278242
- 95% Adjusted-CLT UCL (Chen-1995): 5.666682
- 95% Modified-t UCL (Johnson-1978): 5.666682
- Assuming Normal Distribution

Gamma Statistics

- k hat (MLE): 0.333229
- Theta hat (MLE): 10.71022
- nu hat (MLE): 76.6427
- MLE Mean (bias corrected): 3.568957
- Adjusted Level of Significance: 0.047913

Assuming Gamma Distribution

- 95% Approximate Gamma UCL (use when n>=50): 4.76554
- 95% Adjusted Gamma UCL (use when n<50): 4.783077

Lognormal GOF Test

- Shapiro Wilk Test Statistic: 0.805098
- Shapiro Wilk Lognormal GOF Test: Data Not Lognormal at 5% Significance Level
- Lilliefors Test Statistic: 0.221764
- Lilliefors Lognormal GOF Test: Data Not Lognormal at 5% Significance Level
- Data Not Lognormal at 5% Significance Level

Lognormal Statistics
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Mean of logged Data:  -0.76189
Maximum of Logged Data:  4.70048
SD of logged Data:  1.57863

Assuming Lognormal Distribution:
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95% Chebyshev (MVUE) UCL:  2.58340
99% Chebyshev (MVUE) UCL:  3.03569

Nonparametric Distribution Free UCL Statistics:
Data do not follow a Discernible Distribution (0.05)

Nonparametric Distribution Free UCLs:
95% CLT UCL:  5.53458
95% Standard Bootstrap UCL:  5.56829
95% Hall's Bootstrap UCL:  6.52272
95% BCA Bootstrap UCL:  6.44391
90% Chebyshev(Mean, Sd) UCL:  7.15400
97.5% Chebyshev(Mean, Sd) UCL:  11.03182

Suggested UCL to Use:
95% Chebyshev (Mean, Sd) UCL:  8.77790

Note: Suggestions regarding the selection of a 95% UCL are provided to help the user to select the most appropriate 95% UCL. These recommendations are based upon the results of the simulation studies summarized in Singh, Singh, and Iaci (2002) and Singh and Singh (2003). However, simulations results will not cover all Real World data sets. For additional insight the user may want to consult a statistician.