Managing mercury exposure in northern Canadian communities

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Mercury is a toxic metal that can cause deleterious neurologic effects at any age, although the fetus is especially at risk.1 Most Canadians are exposed to mercury through diet, specifically fish consumption.2 Several Canadian clinical guidance publications assist health professionals in recognizing clinically important mercury levels and appropriate interventions.2-4 However, the information is poorly adapted to the circumstances of geographically remote northern communities, for whom mercury exposure may be greater.5

In this article, we review the literature on the effects of mercury exposure and its management, in the context of other challenges that northern—primarily indigenous—Canadian communities face. These challenges include highly prevalent food insecurity associated with a rapid transition toward store-bought foods and decreasing access to local natural resources.5,7 We also provide guidance for promoting patient health, especially during pregnancy, while addressing mercury exposure in at-risk populations. Our search strategy is summarized in Box 1.8-10 The principles and suggestions in this paper apply directly to practitioners working in northern Canadian communities, where wild foods are important dietary components. They are more broadly applicable to practitioners working with patients or in communities with high fish consumption.

How do people in northern Canada become exposed to mercury?

Some local ecosystems in northern Canada and the wild foods that come from those ecosystems are contaminated by mercury, which increases the risk of exposure. Despite limited industrial activity,11 the environmental burden of mercury may be greater in northern Canada than further south because mercury released to the atmosphere and oceans, primarily from coal combustion and small-scale gold mining in emerging economies, is subject to long-distance transport and accumulation at the higher latitudes.12 Moreover, wild foods from recently impounded reservoirs (e.g., hydroelectric reservoirs in Quebec, Manitoba and Labrador) can contain very high mercury concentrations because of the mobilization of naturally occurring mercury in soils into aquatic ecosystems.13 Industrial activities in the Athabasca oil sands region also appear to be releasing mercury into the surrounding environment.14

Mercury’s three common forms are elemental, inorganic and methylmercury. Methylmercury is encountered through diet, and because of its ubiquity, it is of most concern to health professionals.12 When either elemental or inorganic mercury enters aquatic ecosystems, it is transformed into methylmercury by aquatic microbial activity. Methylmercury bioaccumulates over time in the flesh and organs of living organisms and is biomagnified in top predators of aquatic food webs, such as predatory fish and marine mammals. When humans consume these fish and mammals, they become exposed to high concentrations of methylmercury.12 Unless otherwise noted, we discuss dietary exposure and are therefore referring to methylmercury with the word mercury.

Dietary mercury is absorbed efficiently by the gut, distributed throughout the body and actively transported through blood–brain and blood–placental barriers.12 Mercury accumulation is greater in the fetus than in the mother: concentrations are about 1.5 times higher in umbilical cord blood samples than in maternal blood samples at term postnatal mercury exposure in both children and adults is limited.16

Key points

- Many northern communities in Canada are deeply connected to the sea, rivers and lakes for food, medicine, livelihood and identity, but they are often exposed to mercury through their diets, which incorporate wild foods from these ecosystems.
- Because of large geographic differences in the availability of wild foods and sources of mercury exposure in some northern regions, more location-specific information is needed to guide health promotion activities.
- Prevalent food insecurity and nutritional concerns can conflict with efforts to reduce mercury exposure in northern communities; patient counselling efforts around mercury exposure thus require a more nuanced and interdisciplinary approach.
- A large body of Canadian and international evidence documents adverse neurodevelopmental outcomes in children exposed prenatally to mercury.
- The evidence base that addresses the health effects associated with long-term postnatal mercury exposure in both children and adults is limited.
Box 1: Evidence used in this review

We searched Embase, MEDLINE, Scopus and Google Scholar from 1994 to 2015 for English- and French-language epidemiologic studies conducted in Canada using the following search terms: “mercury” or “methylmercury,” and “Canada,” “Nunavik,” “Nunavut,” “Northwest Territories,” “Yukon,” “Inuit,” “Cree,” “Dene,” “Metis,” “First Nations,” “indigenous,” “Aboriginal” and “fishermen.” We conducted a cited reference search on all review articles on the topic. In addition, we reviewed books and technical reports from the National Academy of Sciences, the Arctic Monitoring and Assessment Programme and the World Health Organization.

Prospective longitudinal cohort study designs provide the highest quality evidence for examining mercury-related health outcomes in humans, because experimental studies are not ethical. In Canada, the Nunavik Child Development Study (NCDS) is the first long-term prospective birth cohort study examining the growth, developmental and behavioural effects of exposure to mercury and other environmental contaminants. The Maternal–Infant Research on Environmental Chemicals (MIREC) study, which began in 2008 in 10 Canadian cities, followed infants to 6 months of age; the results are being published. Most other Canadian research on prenatal and postnatal mercury exposure is cross-sectional in design. Therefore, we prioritized the NCDS findings. Because of limited longitudinal studies in Canada, we corroborated the NCDS and MIREC findings (the results of which are still being published) with results from other birth cohorts that are considered critical epidemiologic studies in the area. When comparative longitudinal studies were lacking, we also examined well-known cross-sectional research from outside of Canada. For all exposure groups, most Canadian epidemiologic research on mercury-related health effects originates from northern Quebec.

delivery. Breast milk may contain mercury, but because the metal is not lipophilic, infant exposure from breastfeeding is negligible compared with fetal exposure.

Although mercury exposure is a global health issue, putting any population that consumes large amounts of fish or marine mammals at risk, people in northern Canadian communities may have higher blood mercury levels compared with other populations (Table 1), depending on the types and frequency of wild foods consumed.

Not all wild foods are equal. Although most are low in mercury (≤0.2 μg/g), flesh from long-lived top predators, such as beluga whales, can accumulate very high mercury concentrations (>1.0 μg/g). Freshwater predatory fish, including lake trout, sauger, walleye and northern pike, contain highly variable mercury concentrations depending on the fish’s age and characteristics of the local ecosystem. Box 2 shows calculations for consumption recommendations for fish with different levels of mercury concentration, according to Health Canada’s provisional tolerable daily intake of mercury. These calculations highlight that the local composition of diet substantially contributes to mercury exposure;

Table 1: Blood mercury concentrations in select Canadian surveys of women of child-bearing age and children

<table>
<thead>
<tr>
<th>Study</th>
<th>Design</th>
<th>Sampling period</th>
<th>Population</th>
<th>% with blood Hg concentration above guidance value (8 μg/L)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nunavik Health Survey&lt;sup&gt;16&lt;/sup&gt;</td>
<td>Representative survey</td>
<td>2004</td>
<td>Inuit women aged 18–39 yr in Nunavik (northern Quebec)</td>
<td>53.3</td>
</tr>
<tr>
<td>NCDS&lt;sup&gt;8&lt;/sup&gt;</td>
<td>Birth cohort</td>
<td>2005–10</td>
<td>Inuit school-aged children in Nunavik</td>
<td>16.9*</td>
</tr>
<tr>
<td>Canadian Health Measures Survey&lt;sup&gt;17&lt;/sup&gt;</td>
<td>Representative survey†</td>
<td>2007–09</td>
<td>Women aged 16–49 yr; Canada-wide study</td>
<td>2.2</td>
</tr>
<tr>
<td>NCDS&lt;sup&gt;8&lt;/sup&gt;</td>
<td>Birth cohort</td>
<td>1995–01</td>
<td>Pregnant Inuit women from Hudson Bay, Nunavik§</td>
<td>10.4 ± 0.4</td>
</tr>
<tr>
<td>Maternal biomonitoring study&lt;sup&gt;18&lt;/sup&gt;</td>
<td>Convenience sample</td>
<td>2005–07</td>
<td>Pregnant Inuit women in Baffin region</td>
<td>4.0 (3.4–4.7)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pregnant Inuit women in Inuvik</td>
<td>1.1 (0.85–1.5)</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Pregnant Dene and Metis women in Inuvik</td>
<td>0.70 (0.45–1.1)</td>
</tr>
<tr>
<td>Canadian Health Measures Survey&lt;sup&gt;17&lt;/sup&gt;</td>
<td>Representative survey</td>
<td>2007–09</td>
<td>Women aged 16–49 yr; Canada-wide study</td>
<td>0.72 (0.50–0.94)</td>
</tr>
<tr>
<td>MIREC study&lt;sup&gt;19&lt;/sup&gt;</td>
<td>Birth cohort</td>
<td>2008–11</td>
<td>Pregnant women in 10 Canadian urban regions</td>
<td>0.86 ± 2.84</td>
</tr>
</tbody>
</table>

Note: CI = confidence interval, Hg = mercury, MIREC = Maternal-Infant Research on Environmental Chemicals study, NCDS = Nunavik Child Development Study, SD = standard deviation.

*Not published; proportion calculated directly from the NCDS mercury results.
†Distributions of environmental concentrations are often skewed to the right and log-normally distributed. Consequently, many studies use geometric means to estimate central tendency, although this measure may be biased low.
§The contemporary NCDS contains more than 1 birth cohort. These estimates come from one of the cohorts, and the sample comprises pregnant women recruited from the 3 largest communities in the Hudson Bay region of Nunavik.


therefore, it is not possible to calculate a general safe level for fish consumption.

Box 3 lists governmental websites with useful information about mercury levels in food, including advisories about freshwater fish consumption from the Government of Quebec. However, more location-specific resources are needed, especially in northern Canada, where there are substantial data gaps.\textsuperscript{13}

What is known of the health effects of dietary mercury exposure?

Two clusters of organic mercury poisoning, in Japan and Iraq, constitute the basis of our knowledge of clinical symptomology in humans.\textsuperscript{22,23} Symptoms of the poisoning and histopathologic changes occurred primarily in the central nervous system. They included sensory impairment of the extremities, constriction of the visual field, cerebellar ataxia, hearing and visual loss, muscle weakness, tremor, cognitive deterioration and death.\textsuperscript{22,23} Among children in Japan exposed prenatally to mercury, the incidence of intellectual disability and cerebral palsy increased substantially.\textsuperscript{10} The incidence of cerebral palsy among children with congenital Minimata disease was 9\% of 188 births in villages affected by the poisoning in Japan compared with a national incidence of 0.2\%—2.3\%.\textsuperscript{10} Also, even among those without known congenital Minimata disease, the prevalence of intellectual disability among children born between 1955 and 1958 in the contaminated area was 29\%.\textsuperscript{10} Although less well documented, mercury poisoning may have occurred in Canada, such as in Grassy Narrows during the 1970s.\textsuperscript{24} Manifestations of mercury intoxication are often nonspecific, vary between individuals and require assessment by an expert (medical toxicologist or neurologist).\textsuperscript{25}

In the last 30 years, research has focused on the subclinical effects of mercury in populations habitually exposed to lower levels of mercury than observed in Japan and Iraq. Most work is on the effects of prenatal exposure in children. In Canada, the Nunavik Child Development Study (NCDS) provides the highest quality longitudinal results on these effects (Box 4).\textsuperscript{8}

What are the effects of prenatal mercury exposure in children?

Evidence from Canada and abroad strongly indicates that prenatal, and possibly postnatal, exposure to mercury negatively affects multiple domains of child development. The NCDS has looked extensively at the neurodevelopmental effects of prenatal exposure to mercury through maternal diet. The findings have included poorer early high-contrast visual processing,\textsuperscript{26} changes in attentional processing of sensory information,\textsuperscript{27} poorer immediate memory and long-term recollection memory,\textsuperscript{28} increased prevalence of symptoms of attention-deficit/hyperactivity disorder in the classroom,\textsuperscript{29} poorer estimated intellectual quotient and increased risk of intellectual disability.\textsuperscript{30} These results are corroborated by findings from birth cohorts in the Faroe Islands and New Zealand.\textsuperscript{31,32}

Increased exposure to mercury during pregnancy might affect obstetric and perinatal outcomes. According to the NCDS, moderate to ele-

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**Box 2: How mercury levels in fish translate into fish consumption guidelines**

Health Canada’s recommended provisional tolerable daily intake (pTDI) of mercury:\textsuperscript{21}

- **Adults:** 0.47 g/kg body weight per day
- **Women of child-bearing age and children:** 0.2 μg/kg body weight per day*

A person’s probable daily intake of mercury (in μg/kg body weight per day) can be calculated with the following formula: [fish portion (g) × frequency of consumption (times/d)] ÷ mercury in fish [μg/g] ÷ body weight (kg)

For example, a 60-kg pregnant woman can eat 150 g of a given fish species at the following maximum frequencies to avoid exceeding the recommended pTDI:

- **Fish low in mercury (0.2 μg/g)**
  - No. times/d = 0.40
  - No. times/wk = 2.8
  - No. times/mo = 12.4

- **Fish high in mercury (0.5 μg/g)**
  - No. times/d = 0.16
  - No. times/wk = 1.1
  - No. times/mo = 5.0

- **Fish very high in mercury (1.0 μg/g)**
  - No. times/d = 0.08
  - No. times/wk = 0.6
  - No. times/mo = 2.5

- **Fish extremely high in mercury (4.0 μg/g)**
  - No. times/d = 0.02
  - No. times/wk = 0.1
  - No. times/mo = 0.6

As a general rule, because fish is important to good health, practitioners in northern Canada may consider counselling their patients with the following: “Eat wild foods, including smaller fish and fish that don’t eat other fish. When possible, eat less larger fish that eat other fish.”

Examples of piscivorous fish with elevated median mercury concentrations that are relevant to northern Canada include lake trout, walleye and northern pike. In contrast, brook trout, rainbow trout and lake whitefish\textsuperscript{13} tend to have much lower median mercury concentrations and are appropriate substitutes.

*This recommended pTDI is based on 10 μg/g in maternal hair (equivalent to 200 nmol/L or 40 μg/L in blood) as the approximate threshold for adverse neurodevelopmental outcomes with a fivefold uncertainty factor to account for variability between individuals.\textsuperscript{11}
After the studies used cross-sectional designs and had low response rates (≤ 50%), they had large samples (> 600) and adjusted extensively for confounders. Other cross-sectional studies conducted outside of Canada have also documented neurologic abnormalities in fish-eating populations with comparable mercury exposure levels.

In contrast to children, in whom the developing brain is a critical target of mercury toxicity, the cardiovascular system may be most sensitive in adults. Representative surveys of Nunavik Inuit and Cree adults documented associations between increasing blood mercury concentrations and risk markers for cardiovascular disease. Although the studies used cross-sectional designs and had low response rates, they had large samples (> 600) and adjusted extensively for confounders. It is possible that chronic conditions influence mercury metabolism and excretion, and thus affect exposure concentrations observed in cross-sectional studies (e.g., reverse causality). In addition, cross-sectional studies cannot determine whether the observed mercury-induced cardiovascular damage occurred prenatally or postnatally. Two well-conducted nested case–control studies analyzing data from large prospective US cohorts found no association between mercury exposure and hypertension, nonfatal myocardial infarction, coronary artery disease or stroke in adults free of cardiovascular disease at baseline; however, exposure levels in these cohorts were lower than those typically observed in northern Canada. In the absence of prospective studies among adults with greater exposure levels, it may be justifiable to advise people with elevated cardiovascular risk to reduce mercury exposure as a precaution.

What are the effects of long-term mercury exposure in adults?

Although high mercury levels (> 100 μg/L in blood) have occasionally been reported in Canadian adults since the 1970s, mercury-induced signs and symptoms have never been comprehensively studied. Consequently, it is virtually impossible to establish diagnostic criteria for mercury intoxication at exposure levels relevant to northern Canada. Moreover, the evidence base for the health effects of long-term mercury exposure in Canadian adults is limited in scope and methodology. For example, among 135 Quebec Cree adults less than 40 years old, tremor was significantly associated with increasing mercury concentrations when measured by a general clinical examination but not by a specialized neurologic examination. The study was the result of a lawsuit, which may have compromised internal validity because participants with symptoms may have self-selected into the study. In a cross-sectional study with a control group of non-exposed Japanese residents, First Nations adults from Grassy Narrows, Ontario, exhibited neurologic symptoms consistent with mercury poisoning, but the study lacked biomarker data of actual mercury exposure. Other cross-sectional studies conducted outside of Canada have also documented neurologic abnormalities in fish-eating populations with comparable mercury exposure levels.

In contrast to children, in whom the developing brain is a critical target of mercury toxicity, the cardiovascular system may be most sensitive in adults. Representative surveys of Nunavik Inuit and Cree adults documented associations between increasing blood mercury concentrations and risk markers for cardiovascular disease. Although the studies used cross-sectional designs and had low response rates (≤ 50%), they had large samples (> 600) and adjusted extensively for confounders. It is possible that chronic conditions influence mercury metabolism and excretion, and thus affect exposure concentrations observed in cross-sectional studies (e.g., reverse causality). In addition, cross-sectional studies cannot determine whether the observed mercury-induced cardiovascular damage occurred prenatally or postnatally. Two well-conducted nested case–control studies analyzing data from large prospective US cohorts found no association between mercury exposure and hypertension, nonfatal myocardial infarction, coronary artery disease or stroke in adults free of cardiovascular disease at baseline; however, exposure levels in these cohorts were lower than those typically observed in northern Canada. In the absence of prospective studies among adults with greater exposure levels, it may be justifiable to advise people with elevated cardiovascular risk to reduce mercury exposure as a precaution.
How should mercury exposure be assessed?

Biological specimens that can be used to measure the body burden of mercury are summarized in Table 2. Blood and hair are considered optimal for assessing dietary mercury exposure in adults. Maternal blood and hair sampled during early pregnancy, and cord blood at delivery, are gold standards for measuring prenatal exposure. In 2010, Legrand and colleagues calculated mercury thresholds of concern and appropriate clinical actions depending on an individual’s age and sex. In Table 3, we have adapted these to include Quebec-specific thresholds and other considerations for geographically remote contexts.

How can dietary counselling be tailored for residents in northern Canada?

Mercury exposure is one of many health concerns facing northern Canadian communities. Counselling to reduce exposure must recognize and address wider food security issues and nutritional challenges, or it will unlikely be effective. Linked with a rapid transition from wild foods to a Western diet, food quality and nutrient intakes have declined in many indigenous communities, which has contributed to increasing rates of chronic diseases and obesity among adults.50 Many northern communities also experience exceptional levels of food insecurity, with most people in some communities compromising the quantity and quality of foods they consume.6 These factors complicate counselling strategies, especially during pregnancy when the fetus is most vulnerable to mercury exposure. In addition, past efforts to reduce dietary mercury exposure have frightened and confused some communities about wild foods,51 even though for many, procuring, eating and sharing wild foods promotes a healthy spirit, mind and body.52 Wild foods are also nutritionally rich in proteins, vitamins, minerals and phytochemicals.16

In the following sections, we provide an overview of food security and nutritional considerations related to wild foods and mercury exposure during pregnancy. Specifically, omega-3 fatty acids, selenium and, more recently, iron have been the focus of extensive epidemiologic research into the appropriate interpretation of many health out-

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**Table 2: Summary of recommended biological specimens to evaluate dietary mercury exposure**

<table>
<thead>
<tr>
<th>Specimen</th>
<th>Measurement recommended?</th>
<th>Exposure period</th>
<th>Comments/limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Blood*</td>
<td>Yes</td>
<td>Last 3 mo</td>
<td>• Most common, accurate measure of postnatal methylmercury exposure&lt;sup&gt;10&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Hard to ascertain if there have been acute exposure events that may explain elevated blood level&lt;sup&gt;10&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Possibly affected by hematocrit because mercury binds to hemoglobin&lt;sup&gt;47&lt;/sup&gt;</td>
</tr>
<tr>
<td>Hair†</td>
<td>Yes</td>
<td>About 1 mo for every cm of hair</td>
<td>• Less invasive and sample is easily preserved&lt;sup&gt;10&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Well correlated with blood measurement</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Approximate delay of 20 d between exposure and excretion&lt;sup&gt;10&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• May be analyzed by cm for a retrospective portrayal of mercury exposure over time&lt;sup&gt;10&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Limited usefulness when hair is short</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• May be affected by permanent hair treatments, natural hair colour and structure&lt;sup&gt;48&lt;/sup&gt;</td>
</tr>
<tr>
<td>Fingernail or toenail</td>
<td>No</td>
<td></td>
<td>• Few reference values&lt;sup&gt;10&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Exposure period hard to establish</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Limited usefulness in clinical practice</td>
</tr>
<tr>
<td>Urine</td>
<td>Not for methylmercury</td>
<td></td>
<td>• May be used to confirm recent exposure to inorganic mercury&lt;sup&gt;15&lt;/sup&gt;</td>
</tr>
<tr>
<td>Plasma</td>
<td>No</td>
<td></td>
<td>• Very low mercury fraction in plasma&lt;sup&gt;9&lt;/sup&gt;</td>
</tr>
<tr>
<td>Cord blood at birth</td>
<td>Yes</td>
<td>Last wk of gestation</td>
<td>• Considered best available measure of prenatal mercury exposure&lt;sup&gt;10&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Few reference values&lt;sup&gt;10&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>• Mercury level is about 1.5 higher than in maternal blood at delivery&lt;sup&gt;15&lt;/sup&gt;</td>
</tr>
</tbody>
</table>

*If patient’s hair is long enough and resources permit, combine with measurement in hair sample (analysis by cm if possible) to clarify whether exposure is long term or acute.

†There may be important differences in blood-to-hair mercury ratio between people; singular use of hair sample and of hair-to-blood concentration conversion of 250 proposed by the World Health Organization should be discouraged when establishing individual risk in a clinical setting.
comes associated with mercury exposure, because these nutrients may confound observed associations or biochemically interact with the contaminant to influence toxicity. Because our objective is to highlight specific health care concerns often concomitant with elevated mercury exposure in northern communities, we have not reviewed all research on mercury–nutrient interactions. Instead, we focus on food security and nutritional concerns that are especially relevant to northern Canada and for which there is a substantial evidence base upon which guidance to health professionals can be based. Our overarching goal is to improve providers’ messaging to patients about dietary mercury exposure.

### Food security and fetal sensitivity to mercury
A sudden change in food intake during pregnancy might increase fetal sensitivity to toxic substances,

<table>
<thead>
<tr>
<th>Group</th>
<th>Total mercury level</th>
<th>Recommended actions</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>In blood</td>
<td>In hair</td>
</tr>
<tr>
<td><strong>Women of child-bearing age</strong>&lt; 8 µg/L (&lt; 40 nmol/L)</td>
<td>&lt; 2 µg/g</td>
<td>• No follow-up required</td>
</tr>
</tbody>
</table>
| **Children < 18 yr**        | 8–40 µg/L (> 40–200 nmol/L) | > 2–10 µg/g  | • Repeat blood/hair test immediately in pregnant women; otherwise repeat in 3–6 mo  
  • Identify dietary sources of exposure and provide dietary advice, taking into account social importance of wild foods  
  • If food insecurity or micronutrient deficiency is a concern, consult with nutritionist or dietitian for low-mercury, high-nutrient food options |
| > 40–100 µg/L (> 200–500 nmol/L) | > 10–25 µg/g  | • Repeat blood/hair test immediately  
  • Identify dietary sources of exposure and provide dietary advice, taking into account social importance of wild foods  
  • If food insecurity or micronutrient deficiency is a concern, consult with nutritionist or dietitian for low-mercury, high-nutrient food options |
| > 100 µg/L (> 500 nmol/L)   | > 25 µg/g     | • Repeat blood/hair test immediately  
  • Request mercury speciation in whole blood to discriminate dietary methylmercury from inorganic mercury exposure  
  • If inorganic mercury, identify environmental sources of exposure and seek solutions with patient to eliminate exposure  
  • If methylmercury, identify dietary sources of exposure and provide dietary advice  
  • If accompanied by symptoms of mercury exposure, refer patient to an expert (medical toxicologist or neurologist) |
| **Women > child-bearing age**< 20 µg/L (< 100 nmol/L) | < 5 µg/g  | • No follow-up required                                                                                                                                |
| **Men > 18 yr**             | 20–100 µg/L (100–500 nmol/L) | > 5–25 µg/g  | • Repeat blood/hair test in 3–6 mo  
  • Identify dietary sources of exposure and provide dietary advice, taking into account social importance of wild foods  
  • If accompanied by symptoms of mercury exposure, refer patient to an expert (medical toxicologist or neurologist) |
| > 100 µg/L (> 500 nmol/L)   | > 25 µg/g     | • Repeat blood/hair test immediately  
  • Request mercury speciation in whole blood to discriminate dietary methylmercury from inorganic mercury exposure  
  • If inorganic mercury, identify environmental sources of exposure and seek solutions with patient to eliminate exposure  
  • If methylmercury, identify dietary sources of exposure and provide dietary advice  
  • If accompanied by symptoms of mercury exposure, refer patient to an expert (medical toxicologist or neurologist) |
| **All groups (Quebec only)**≥ 12 µg/L (≥ 60 nmol/L) | ≥ 3 µg/g  | • Test result must be declared to regional public health authority (consult steps to be taken at www.msss.gouv.qc.ca/professionnels/mado/demarche_medecins.php) |

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including mercury. One population-based cross-sectional study in Bangladesh concluded that women and children were more at risk than men for both toxic metal exposure and micronutrient deficiency. To our knowledge, no epidemiologic research in Canada and elsewhere has investigated whether mercury toxicity differs according to food security status, sex, general nutrition or micronutrient deficiency. Health advice to women of childbearing age about mercury should factor in the wider context of food insecurity by avoiding messages that scare families away from wild foods low in mercury.

**Omega-3 fatty acids, mercury and health outcomes**

Omega-3 fatty acids from the mother’s diet are essential components of membrane phospholipids and are deposited to the fetus’ central nervous system in relatively large quantities during the last trimester of pregnancy and first months of life. Fatty acids in fish and seafood negatively confound associations between mercury and neurotoxicity. In Nunavik, greater intake of omega-3 fatty acids during the last trimester of pregnancy is associated with better neurophysiologic and neurobehavioural outcomes. The benefits of prenatal exposure to omega-3 fatty acids are maintained throughout childhood and reduce sensitivity to outcomes of mercury exposure, such as intellectual disability.

Although omega-3 fatty acids may not counteract the toxic effects of mercury, they have been positively associated with many of the same health outcomes with which mercury exposure has been negatively associated. Fatty fish (e.g., salmon, sardines and herring) and marine mammal fat are high in omega-3 fatty acids and generally low in mercury, and their consumption can be encouraged during pregnancy.

**Selenium-mercury interactions**

Experimental and cross-sectional studies suggest toxicokinetic interactions between selenium and mercury, with selenium reducing the reproductive, neurologic and cardiovascular toxic effects associated with mercury exposure.

Such findings are unconfirmed in epidemiologic studies from northern Canada. But if true, this would raise concerns of even greater toxic effects from mercury in certain northern communities with previously high selenium intakes from wild foods that are now rapidly being exchanged for Western diets with lower selenium concentrations.

**Iron deficiency and mercury exposure during pregnancy**

The prevalence of iron deficiency is high in some northern communities with elevated food insecurity or ongoing nutritional transition. Positive correlations between iron stores and blood mercury levels have been documented; methylmercury is known to accumulate in red blood cells and bind to hemoglobin. In Nunavik, the correlation was attributed to general consumption of wild foods, because many wild foods have elevated levels of bioavailable iron. Both iron deficiency and prenatal exposure to mercury negatively affect children’s brain development, potentially confounding these associations in populations where both are high.

Dietary advice to reduce mercury exposure should likewise not increase the risk of iron deficiency during pregnancy by scaring women away from iron-rich wild foods, especially terrestrial species that are low in mercury.

**Conclusion**

The Minamata Convention on Mercury, a global treaty to protect humans and the environment from adverse effects of mercury, was approved in January 2013. It includes a ban on new mercury mines, phasing out of old ones, air emissions controls, and regulations for international artisanal and small-scale gold mining. If successful, long-term anthropogenic emissions of mercury may decline, reducing global environmental concentrations.

For now, we expect communities that frequently consume predatory fish or marine mammals to have elevated mercury exposure. It is important for clinicians to be clear on the potential risks of mercury exposure, the complexity of the underlying epidemiologic data and how to counsel, in a culturally sensitive manner, patients in communities where mercury exposure may be a concern. Gaps in knowledge are summarized in Box 5. Regionally specific sources of information on popular wild foods and their nutritional and contaminant properties that are understandable to health care providers and their patients are required.

**Box 5: Gaps in knowledge**

- More location-specific tools are needed to guide health promotion activities in northern Canada, given the large geographic variation in mercury concentrations in wild foods.
- Prospective studies are needed of adverse health effects from long-term exposure to mercury in adults, especially in communities with elevated mercury levels.
- Research is needed that simultaneously examines food security, malnutrition (including obesity), micronutrient deficiencies and mercury toxicity.
References

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Contributors: Catherine Pirkle and Melanie Lemire contributed equally to the conception and outline of the manuscript. Catherine Pirkle reviewed the literature, with substantial input from Melanie Lemire and Gina Muckle. Catherine Pirkle drafted the first version of the manuscript. All of the authors contributed to the interpretation of the literature discussed in the article and to the critical revisions of subsequent drafts, approved the final version of the manuscript to be published and agreed to act as guarantors of the work.

Acknowledgement: Catherine Pirkle was supported by a postdoctoral fellowship award from the Institute of Aboriginal Peoples’ Health, Canadian Institutes of Health Research, during which much of this work was conducted.

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