Modeling tribal exposures to methyl mercury from fish consumption

Jianping Xue⁎, Valerie Zartariana, Bruce Mintza, Marc Weberb, Ken Bailey c, Andrew Gellerd

a U.S. Environmental Protection Agency, Office of Research and Development, National Exposure Research Laboratory, 109 T.W. Alexander Drive, Research Triangle Park, NC 27709, United States

b U.S. Environmental Protection Agency, Office of Research and Development, National Health and Environmental Effects Research Laboratory, United States

c U.S. Environmental Protection Agency, Office of Science Policy, United States

d U.S. Environmental Protection Agency, Office of Research and Development, Sustainable and Healthy Communities Research Program, United States

HIGHLIGHTS

• The paper identifies key factors of methyl mercury (MeHg) exposure
• MeHg exposures from fish in tribes are much higher than the US general population
• ~50% of MeHg dietary exposures can be reduced just by avoiding some fishes with high MeHg

GRAPHICAL ABSTRACT

Exposure sensitivity analyses reducing average fish intake for different scenarios: As much as ~50% of MeHg dietary exposures can be reduced just by replacing several species of fish with high MeHg concentration (e.g., walleye, bowfin), substituting species with lower concentrations.

ABSTRACT

Exposure assessment and risk management considerations for tribal fish consumption are different than for the general U.S. population because of higher fish intake from subsistence fishing and/or from unique cultural practices. This research summarizes analyses of available data and methodologies for estimating tribal fish consumption exposures to methyl mercury (MeHg). Large MeHg fish tissue data sets from the Environmental Protections Agency’s (EPA’s) Office of Water, USGS’s EMMMA program, and other data sources, were integrated, analyzed, and combined with fish intake (consumption) data for exposure analyses using EPA’s SHEDS-Dietary model. Results were mapped with GIS tools to depict spatial distributions of the MeHg in fish tissues and fish consumption exposure patterns. Contribution analyses indicates the major sources for those exposures, such as type and length of fish, geographical distribution (water bodies), and dietary exposure patterns. Sensitivity analyses identify the key variables and exposure pathways. Our results show that MeHg exposure of tribal populations from fish are about 3 to 10 times higher than the US general population and that exposure poses potential health risks. The estimated risks would be reduced as much as 50%, especially for high percentiles, just by avoiding consumption of fish species with higher MeHg concentrations such as walleye and bowfin, even without changing total fish intake. These exposure assessment methods and tools can help inform decisions regarding meal sizes and

E-mail address: xue.jianping@epa.gov (J. Xue).

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1. Introduction

Concerns of health risks from fish consumption are a priority tribal issue (Donatuto and Harper, 2008). Exposure assessment and risk management considerations for tribal fish consumption are different than for the general U.S. population because of higher fish intake from subsistence fishing and/or from unique cultural practices (USEPA, 2004; Donatuto and Harper, 2008). Tribal populations are vulnerable to methyl mercury (MeHg) which may lead to impairment of the developing central nervous system as well as pulmonary and nephrotic damage (Cohen et al., 2005). It is well documented that serious health effects of mercury resulted from high-level exposures in Minimata and Nigata, Japan (Irukayama et al., 1977) and in Iraq (Bakir et al., 1973). Though it is very unlikely for people in the general population to have those high-level exposures, the effects of exposure to low levels MeHg are well documented and include developmental deficits, particularly in children exposed prenatally (Grandjean et al., 1997; NRC, 2000). The toxic effects of MeHg are irreversible and severe enough that the potential risk to the United States population from consuming a variety of fish should be reviewed on a continuing basis (Mahaffey and Mergler, 1998). At the same time, it is important to note that eating fish has many health benefits (Daviglus et al., 2002; Mozaffarian and Rimm, 2006).

In aquatic environments, MeHg bio-accumulates up the food chain. Fish contain traces of MeHg; however, it accumulates more in certain types of fish, depending on what the fish eat, resulting in varying MeHg levels. Also, larger fish (swordfish, shark, king mackerel and tilapia) that eat smaller fish, have the highest levels of MeHg due to bioaccumulation. In general concentrations of MeHg vary ~2 orders of magnitude between species (Mahaffey et al., 2011). Only a few species of fish could have MeHg levels of 1 ppm or greater. This occurs most frequently in some large predator fish, such as shark and swordfish and in certain species of large tuna, typically sold as fresh steaks or sushi (Fletcher and Gelberg, 2013).

Reliable estimates of MeHg exposures from fish consumption, and the major contributors, can inform decisions of tribal populations and the general US population regarding types and quantities of fish that are both safe to eat and nutritionally beneficial. Fish MeHg concentrations can be highly variable, even within the same species. Therefore, it is important to have a large dataset of MeHg in fish tissues and reliable fish consumption data. The EPA's Stochastic Human Exposure and Dose Simulation model (SHEDS) has been well evaluated with biomarkers for arsenic, MeHg, chlorpyrifos, and pyrethroids (Xue et al., 2012a, 2012b). It has gone through external peer review by EPA's Federal Insecticide Fungicide, Rodenticide Act Scientific Advisory Panel and has been used to support regulatory decisions on organophosphate, carbamates, pyrethroids, chromated copper arsenate (CCA) and others (SAP, 2007; SAP, 2010).

Xue et al. (Xue et al., 2012a, 2012b) using the SHEDS-Dietary model with national data, reinforced and expanded upon previous observations that dietary exposure via fish consumption is an important route for MeHg intake by the general population, and especially for racial/ethnic groups with higher fish consumption such as tribes. That paper concluded that probabilistic dietary modeling approaches could be applied for local populations (e.g., tribes) and other chemicals and foods, if data are available, and that many research and data needs remain for local-scale assessments involving fish consumption exposures/risks (Xue et al., 2012a). Because that study used national rather than tribal-specific fish consumption and residue data, and Americans Indians are grouped with Asians, Pacific Islanders, and multiracial groups (APNM) in the National Health and Nutritional Examination Survey (NHANES), it is difficult to draw tribal-specific conclusions or suggest specific risk reduction recommendations. Future research recommendations included 1) collecting detailed consumption and residue data at the local scale to identify the specific type of fish consumed and the concentrations of MeHg in those fish for specific community or tribal assessments; and 2) conducting dietary exposure analyses to answer questions of interest related to risk mitigation (e.g., identification of key fish contributing to local exposures; maximum meal sizes relevant to reference doses).

Questions being addressed by the research presented in this paper include the following:

- What fish tissue data sets and tribal fish consumption data sets are available for exposure modeling?
- What are major factors for fish contamination and exposures?
- How can tribes minimize exposures and potential health risks from contaminated fish on tribal lands, while maintaining current dietary practices?
- How can exposure assessment tools inform those decisions?

2. Methods

EPA's SHEDS-Dietary, an important module of EPA's SHEDS-Multimedia model, was used for the analysis. SHEDS-Dietary can generate population percentiles of dietary exposure predictions by source and age/gender group; quantify contribution to total exposure predictions by food, commodity, and chemical; and be used for eating occasion, sensitivity, and uncertainty analyses. In general terms, this model combines information about food and drinking water consumption data for each reported eating occasion with corresponding chemical residue/concentration data to estimate human dietary exposures. SHEDS-Dietary can use the NHANES/WWEIA dietary consumption data (1999–2010), along with EPA/USDA recipe translation files (FCID; Food Commodity Intake Database), and available food and water concentration data and detailed methods can refer to the earlier publications (Xue et al., 2010; Xue et al., 2012a).

To conduct the exposure analyses, we compiled and analyzed available fish tissue data sets and tribal fish consumption data from key studies as listed below. We then mapped fish tissue concentrations and analyzed for key exposure factors. We also compared tribal fish consumption data to NHANES consumption data and then used those data as inputs to the EPA SHEDS model (http://www.epa.gov/hea/sheds/research/sheds/user_information.html). With the SHEDS model, we conducted sensitivity analyses to better understand the impact of modifying fish intake for different species.

National fish tissue data sets used here were the following: EPA National Listing of Fish Advisories (NLFA); EPA National Lake Fish Tissue Study; EPA National Rivers and Streams Study; EPA National MeHg Survey; and USGS EMMA (Environmental Mercury Mapping, Modeling, and Analysis). State/local fish tissue data sets used were as follows: Washington State, tribally-provided data, including Columbia River Inter-Tribal Fish Commission (CRITFC) (EPA Region 10), Winnebago Tribe Kelly Pond (EPA Region 7), and Pyramid Lake (EPA Region 9).

Tribal fish consumption surveys used in this analysis were the following:

- A fish consumption survey of the Umatilla, Nez Perce, Yakama, and Warm Springs Tribes of the Columbia River Basin (CRITFC, 1994)
- A fish consumption survey of the Tulalip and Squaxin Island Tribes of the Puget Sound Region (Toy, 1996)
• Fish consumption survey of the Suquamish Indian Tribe of the Port
Madison Indian Reservation, Puget Sound Region (Suquamish, 2000)
• Estimated per capita fish consumption in the US (EPA, 2002).

All data were carefully checked and combined for mapping and
fitting distributions for SHEDS-Dietary.

Only some percentiles and summary statistics from these studies
were available for our analyses; raw data on fish consumption from trib-
al populations are lacking in the literature. Similar statistics and percent-
iles from the U.S. population from the NHANES data were generated for
daily fish intake, to compare between U.S. general population and tribal
populations. Those statistics and percentiles of intake data were com-
bined in SHEDS with inputs of fish MeHg concentrations. SHEDS-
Dietary concentration inputs were either lognormal or empirical distri-
butions fitted from the large data set of MeHg fish tissue concentrations
described above. 1000 SHEDS-Dietary simulations generated variability
distributions of MeHg exposures from fish intakes for the general U.S.

Total blood Hg concentration from 1999 to 2010 was downloaded
from NHANES and the total sample size is 30,260. The Hg exposures
from only study subjects of the study periods with those biomarkers
were used for model evaluation.

SHEDS-Dietary was applied to estimate MeHg exposures for short
duration. Cohort studies were not available to us to conduct a chronic
exposure assessment. Therefore, the Diversity and Autocorrelation
(D & A) method (Glen et al., 2008) was used to construct longitudinal
food consumption diaries. Total caloric consumption was used as the
key variable, with D and A statistics set to 0.3 and 0.1, respectively
based on longitudinal data from Lu, C. et al. (Lu et al., 2006). Dietary expo-
sures of MeHg from fish were simulated for a one year period, and expo-
sure durations of 1 day, one week, two weeks, one month, three months,
six months, and twelve months were calculated for comparison.

3. Results

Fig. 1 shows the spatial distribution of averaged MeHg (ppm) in fish
tissues of the 12 most common fish species across the U.S., and several
with the highest MeHg concentrations. Note the pattern is similar, and
there is good coverage overall from all available data sets. MeHg con-
centrations are higher in the Northeast and South. Bowfin and catfish
have limited data, mainly in the south. Bass concentrations are higher in
Northeast, South and Michigan areas.

Fig. 2 and Table 1 show that bass, bowfin, and walleye have the
highest mean MeHg concentrations among 12 common fresh water fish
species. Bowfin has the highest MeHg concentration with 0.87, 1.16 and
3.11 ppm for mean, 75th and 99th percentiles respectively. Carp has the
lowest MeHg concentration in fish tissue with 0.14, 0.18 and 0.61 ppm
for mean, 75th and 99th percentiles respectively. The ratios between
the highest and lowest are 6, 6, and 5 for mean, 75th and 99th percentiles
respectively. Note the variability in Table 1: carp has the lowest standard
deviation at 0.14 ppm; bowfin the highest at 0.78 ppm.

Table 2 illustrates how much higher fish intake is for tribal popula-
tions compared to other ethnicities. The 2 tribal-specific data sets had
the highest values: 2.71, 6.19, 10.09 g/kg bw/day for mean, 90th and
95th percentiles with Suquamish (2000) data, and 0.89, 2.31 and 2.94
with Toy, K.A. (1996) data. These were 2–5 times higher than the fish in-
takes of the APNM group from 1999 to 2010 NHANES, and the APNM
group fish intakes were ~2 times higher than other NHANES groups in
the general population.

Table 3 shows that CRTFC exposures were much higher than APNM
exposures simulated with SHEDS. We used available percentiles from
the CRTFC study since raw data were not available. MeHg exposures
from fish consumption for tribal populations are highest: 1.09, 2.37 and
4.16 μg/kg bw/day for mean, 90th and 95th percentiles with
Suquamish (2000) and 0.35, 0.94 and 1.17 with Toy, K.A. (1996). MeHg exposures from fish for the APNM population from 1999 to
2010 NHANES were 0.18, 0.64 and 1.01 μg/kg bw/day for ages 20+
years and 0.13, 0.39 and 0.88 μg/kg bw/day for ages 0–19 years kg
bw/day kg bw/day for mean, 90th and 95th percentiles respectively
(Table 3). The Non-Hispanic White group had the lowest MeHg expo-
sure from fish consumption, with 0.06, 0.00 and 0.34 μg/kg bw/day for mean, 90th and 95th percentiles. Ratios of averaged MeHg exposures
between the highest and lowest are approximately 20.

Lognormal distribution were used for SHEDS modeling, since there
was a better fit to MeHg fish tissue concentrations than Normal, Weibull
and other distributions, as shown in Fig. 2. A2 (in Appendix A) shows the

Fig. 1. Spatial distribution across the U.S. of MeHg (ppm) in fish tissues of the 12 most common fish species.
MeHg exposure results with empirical distributions from original large fish tissue data sets in comparison with Table 3 (with fitted lognormal distribution for MeHg in fish tissues), and the ratio between A2 and Table 3 is in A3. With empirical distributions of MeHg in fish tissue as inputs (instead of lognormal distribution), MeHg exposure from fish are very similar to the exposures using lognormal distribution as inputs for all groups (Table 3 and A2). The ratios of exposures from latter method to the former are 1.10, 0.96 and 1.22 for mean, minimum and maximum, indicating that about 10% and 23% over-estimate with lognormal distribution as inputs for mean and maximum, respectively, and the method with lognormal distribution is a conservative choice (A3).

Fig. 3 shows that the key factors for MeHg fish tissue data in water bodies are species, location, weight, and length in the General Linear Model (GLM) analysis. Year of fish tissue sample was not significant.
The dominant factor affecting MeHg in fish tissues is fish type; 62% of the total variance is explained by this factor in the GLM. Other factors are location/state (24%), fish weight (7%), fish length (6%), and year (1%).

Fig. 4 illustrates the results of sensitivity analyses. The average fish intake of Suquamish (2000) data was reduced for different scenarios, and 1000 SHEDS simulations were conducted. A 25% simulation reduced fish intake decreases exposure by about 24%; 50% intake reduction decreases exposure by 59%. Removing high-concentrated MeHg fish decreases the dietary exposure by 44%. A 25% and 50% reduction of fish intake and removal of high-concentrated fish leads to 57% and 68% decrease in exposure, respectively.

Fig. 4 shows the MeHg concentration is reduced by ~50% by removing high-concentration MeHg fish from the diet, such as bowfin, walleye, and largemouth bass.

Table 1: Hg concentrations in fish tissue concentrations of 12 common species.

<table>
<thead>
<tr>
<th>Fish types</th>
<th>n</th>
<th>Mean</th>
<th>Std</th>
<th>Min</th>
<th>p5</th>
<th>p25</th>
<th>p50</th>
<th>p75</th>
<th>p99</th>
<th>Max</th>
</tr>
</thead>
<tbody>
<tr>
<td>N. pike</td>
<td>11,029</td>
<td>0.35</td>
<td>0.28</td>
<td>0.01</td>
<td>0.08</td>
<td>0.17</td>
<td>0.28</td>
<td>0.45</td>
<td>1.4</td>
<td>4.0</td>
</tr>
<tr>
<td>Bass</td>
<td>35,884</td>
<td>0.47</td>
<td>0.42</td>
<td>0.00</td>
<td>0.07</td>
<td>0.20</td>
<td>0.33</td>
<td>0.61</td>
<td>2.0</td>
<td>7.4</td>
</tr>
<tr>
<td>Bluegill</td>
<td>6449</td>
<td>0.20</td>
<td>0.19</td>
<td>0.00</td>
<td>0.03</td>
<td>0.08</td>
<td>0.17</td>
<td>0.25</td>
<td>0.8</td>
<td>4.5</td>
</tr>
<tr>
<td>Bowfin</td>
<td>6998</td>
<td>0.87</td>
<td>0.78</td>
<td>0.00</td>
<td>0.20</td>
<td>0.37</td>
<td>0.68</td>
<td>1.16</td>
<td>3.1</td>
<td>23.2</td>
</tr>
<tr>
<td>Carp</td>
<td>7075</td>
<td>0.14</td>
<td>0.14</td>
<td>0.00</td>
<td>0.02</td>
<td>0.06</td>
<td>0.11</td>
<td>0.18</td>
<td>0.6</td>
<td>3.0</td>
</tr>
<tr>
<td>Carfish</td>
<td>9914</td>
<td>0.24</td>
<td>0.28</td>
<td>0.00</td>
<td>0.02</td>
<td>0.09</td>
<td>0.17</td>
<td>0.26</td>
<td>1.5</td>
<td>4.5</td>
</tr>
<tr>
<td>Craggie</td>
<td>6813</td>
<td>0.26</td>
<td>0.50</td>
<td>0.00</td>
<td>0.03</td>
<td>0.10</td>
<td>0.21</td>
<td>0.33</td>
<td>1.1</td>
<td>26.0</td>
</tr>
<tr>
<td>Drum</td>
<td>2892</td>
<td>0.34</td>
<td>0.31</td>
<td>0.00</td>
<td>0.04</td>
<td>0.12</td>
<td>0.25</td>
<td>0.46</td>
<td>1.5</td>
<td>1.9</td>
</tr>
<tr>
<td>Sucker</td>
<td>4160</td>
<td>0.15</td>
<td>0.17</td>
<td>0.00</td>
<td>0.02</td>
<td>0.06</td>
<td>0.10</td>
<td>0.19</td>
<td>0.8</td>
<td>3.7</td>
</tr>
<tr>
<td>Sunfish</td>
<td>8614</td>
<td>0.26</td>
<td>0.26</td>
<td>0.00</td>
<td>0.03</td>
<td>0.12</td>
<td>0.25</td>
<td>0.28</td>
<td>1.0</td>
<td>2.9</td>
</tr>
<tr>
<td>Trout</td>
<td>3238</td>
<td>0.21</td>
<td>0.22</td>
<td>0.01</td>
<td>0.02</td>
<td>0.08</td>
<td>0.14</td>
<td>0.27</td>
<td>1.0</td>
<td>2.8</td>
</tr>
<tr>
<td>Walleye</td>
<td>12,024</td>
<td>0.42</td>
<td>0.34</td>
<td>0.00</td>
<td>0.08</td>
<td>0.18</td>
<td>0.32</td>
<td>0.55</td>
<td>1.1</td>
<td>4.0</td>
</tr>
</tbody>
</table>

Table 2: Daily fish intake* for NHANES and tribal populations.

<table>
<thead>
<tr>
<th>Group</th>
<th>g/kg bw/day</th>
<th>g/day</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean 90th 95th</td>
<td>Mean 95th 99th</td>
</tr>
<tr>
<td>Mexican American (0–19)</td>
<td>0.18 0.00 1.05</td>
<td>6.0 36.7 146.5</td>
</tr>
<tr>
<td>Mexican American (20+)</td>
<td>0.24 0.73 1.74</td>
<td>17.9 128.0 297.8</td>
</tr>
<tr>
<td>Non-Hispanic White (0–19)</td>
<td>0.14 0.00 0.86</td>
<td>5.1 31.8 127.6</td>
</tr>
<tr>
<td>Non-Hispanic White (20+)</td>
<td>0.21 0.73 1.45</td>
<td>16.7 113.4 260.5</td>
</tr>
<tr>
<td>Non-Hispanic Black (0–19)</td>
<td>0.22 0.41 1.55</td>
<td>8.3 55.8 168.7</td>
</tr>
<tr>
<td>Non-Hispanic Black (20+)</td>
<td>0.26 0.91 1.59</td>
<td>21.7 133.0 291.6</td>
</tr>
<tr>
<td>Other Hispanic (0–19)</td>
<td>0.18 0.00 0.99</td>
<td>5.8 40.0 150.7</td>
</tr>
<tr>
<td>Other Hispanic (20+)</td>
<td>0.24 0.81 1.56</td>
<td>16.9 110.6 262.8</td>
</tr>
<tr>
<td>APNM (0–19)</td>
<td>0.33 0.96 2.20</td>
<td>10.3 62.8 184.1</td>
</tr>
<tr>
<td>APNM (20+)</td>
<td>0.46 1.69 2.53</td>
<td>30.7 167.8 327.9</td>
</tr>
<tr>
<td>Toy, K.A. (1996)</td>
<td>0.89 2.31 2.94</td>
<td></td>
</tr>
<tr>
<td>Suquamish (2000)</td>
<td>2.71 6.19 10.09</td>
<td></td>
</tr>
<tr>
<td>Columbia River Inter-Tribal Fish Commission (1994)</td>
<td>58.7 170.0 389.0</td>
<td></td>
</tr>
</tbody>
</table>

Table 3: Averaged MeHg exposures by percentiles for NHANES and tribal populations.*

<table>
<thead>
<tr>
<th>Group</th>
<th>90th 95th</th>
<th>99th 99th</th>
</tr>
</thead>
<tbody>
<tr>
<td>APNM (0–19)</td>
<td>0.069 0.000 0.391</td>
<td>2.2 14.2 60.3</td>
</tr>
<tr>
<td>APNM (20+)</td>
<td>0.096 0.286 0.692</td>
<td>7.7 53.6 113.5</td>
</tr>
<tr>
<td>APNM (0–19)</td>
<td>0.057 0.000 0.337</td>
<td>1.9 12.5 49.5</td>
</tr>
<tr>
<td>APNM (20+)</td>
<td>0.082 0.288 0.518</td>
<td>6.5 42.9 102.8</td>
</tr>
<tr>
<td>APNM (0–19)</td>
<td>0.080 0.146 0.636</td>
<td>3.3 22.1 65.9</td>
</tr>
<tr>
<td>APNM (20+)</td>
<td>0.096 0.352 0.652</td>
<td>8.0 50.6 112.2</td>
</tr>
<tr>
<td>APNM (0–19)</td>
<td>0.069 0.000 0.400</td>
<td>2.0 14.9 56.4</td>
</tr>
<tr>
<td>APNM (20+)</td>
<td>0.096 0.298 0.603</td>
<td>6.7 42.6 97.3</td>
</tr>
<tr>
<td>APNM (0–19)</td>
<td>0.125 0.392 0.884</td>
<td>4.2 24.8 72.1</td>
</tr>
<tr>
<td>APNM (20+)</td>
<td>0.182 0.637 1.009</td>
<td>11.6 66.8 121.0</td>
</tr>
<tr>
<td>Toy, K.A. (1996)</td>
<td>0.350 0.539 1.167</td>
<td></td>
</tr>
<tr>
<td>Suquamish (2000)</td>
<td>1.085 2.365 4.156</td>
<td></td>
</tr>
<tr>
<td>Columbia River Inter-Tribal Fish Commission (1994)</td>
<td>23.2 64.6 151.0</td>
<td></td>
</tr>
<tr>
<td>Columbia River Inter-Tribal Fish Commission (1994)*</td>
<td>18.5 52.1 122.4</td>
<td></td>
</tr>
</tbody>
</table>

Fig. 3. Key factors for MeHg fish tissue data in water bodies are species, location, weight, and length.

* Using fish tissue Hg data located in tribal area.

4. Discussion

Estimating MeHg exposure for the U.S. general population and tribal populations is challenging, especially for longitudinal exposures, because data from many sources without a cohort study must be assembled for use in a reliable dietary exposure model. Therefore, most estimates for exposure presented here are cross-sectional. The Ojibwe Health Study (OHS) has concluded 10 years of data collection and exposure assessment. Tribes from the states of Wisconsin, Michigan, and Minnesota (822 participants) completed fish consumption and environmental risk perception questionnaires; average fish consumption was ~60 g per day (Dellinger, 2004), which is very comparable to 58.7 g/day in the Columbia River Inter-Tribal Fish Commission study (Table 2). Estimated average exposure for MeHg was 0.04 to 0.06 μg/kg bw/day with 5–8 median meal size fish meals per month for women of childbearing age for U.S. general population (Sunderland, 2007). The FDA has estimated that, on average, the intake rate for total mercury (both inorganic and organic) is 50–100 ng/kg bw/day (equivalent to 0.05–0.1 μg/kg bw/day or 3.5–7 μg/kg bw/day for a 70-kg adult) (ATSDR, 1999). Average and maximum MeHg exposures were 4.8 and 8.75 μg/kg bw/day for children, and 2.2 and 4.0 μg/kg bw/day for adults, for Penobscot Indian Nation with screen model (ATSDR, 2014). Our average SHEDS-modeled exposure for the U.S. general population (6 + years old) is about 0.09 μg/kg bw/day (A4) and 0.35 and 1.11 μg/kg bw/day for the adults of two tribal populations (Toy, 1996; Suquamish, 2000). Our estimates are comparable in terms of the US general population with estimates from FDA and Sunderland’s study, while the exposure of the tribal population exposure estimated.
by SHEDS is lower than the average in Agency for Toxic Substances and Disease Registry (ATSDR) report. The reason is that SHEDS is a higher
tier model using more detailed and larger datasets with more realistic
numbers. Also, there are large variations in tribal population exposures,
due primarily to diverse cultures and geographic locations.

In 2001, EPA revised an oral Reference Dose (RfD) for MeHg intake of
0.1 μg/kg bw/day (http://www.epa.gov/iris/subst/0073.htm).
ATSDR has established a chronic oral Minimum Risk Level (MRL) of
0.3 μg/kg bw/day for MeHg. In 2003, the Joint FAO/WHO Expert
Committee On Food Additives (JECFA), established a Provisional Toler-
able Weekly Intake (PTWI) of 1.6 μg/kg bw (about 0.23 μg/kg bw/day)
for MeHg, based on the most sensitive toxicological end-point (devel-
opmental neurotoxicity) in the most susceptible species (humans)
(FAO/WHO, 2003). Averaged estimated exposure from our results
(Table 3) ranged from 0.06 to 0.1 μg/kg bw/day which is lower than the
EPA RfD (excluding the Tribal, Asian, Pacific 0–19 and 20+ ages).
But average exposures for tribal groups (Toy, 1996; Suquamish, 2000)
are higher than 0.3 μg/kg bw/day. The 95th percentiles for all ethnicities
are higher than 0.3 μg/kg bw/day. Our estimated exposures are all cross-
sectional. Statistically, the average will be similar between acute and
chronic exposures if data sets used for estimates are large. However,
higher percentiles of acute exposures are much higher than chronic
exposures (Fig. 6 and A4). From average and high percentiles of
longitudinal exposure simulations with SHEDS, MeHg in fish is posing a
potentially significant health risk to more highly exposed tribal sub-
populations.

According to questionnaire results, the percentages of respondents
who recalled eating walleye were 73, 49, 44, 22 and 30 for Inland
Lakes, Lake Superior, Menominee, Lakes Michigan/Superior and
other reservations, respectively (Dellinger, 2004). Based on our SHEDS
analyses, for some tribal populations, MeHg exposures would be
reduced greatly just by reducing or avoiding consumption of high-
concentrated fish species.

Model evaluation is crucial for exposure modeling assessments. For
an indirect evaluation, without a physiologically-based pharmacokinet-
ic (PBPK) model for MeHg, we used SHEDS to compare average and
95th percentile of exposure and blood biomarker levels among 10
groups by age and ethnicities. For means and 95th percentile, the
SHEDS exposure estimates are very consistent with the real blood
MeHg concentrations from NHANES among those 10 groups.

Uncertainty is inherent in all exposure models, and it is important to
c caracterize the uncertainty in regard to model structure and data in-
puts. In comparison with fish MeHg residue data and consumption in-
take for the US general population from NHANES, limited fish intake
data from tribal populations will contribute to uncertainty of modeled
MeHg exposures. Also, because fish intake data was not available for
individual fish species, MeHg concentrations were aggregated for
multiple species for the SHEDS modeling, and separate modeling results
by individual fish species could not be calculated. Therefore, the SHEDS
modeling results cannot be used to characterize what proportion of
total tribal MeHg exposure from fish consumption is due to high fish
intake versus intake from highly contaminated fish species, and this
remains a large uncertainty in the overall analysis and conclusions.
Lack of longitudinal studies on fish intake is another factor for
uncertainty in estimating health effects of MeHg from fish consumption.
To reduce uncertainties, it is important to integrate all studies by tribal
populations and new studies with cohorts including detailed fish
species intake data.

There are a number of key findings in this paper. First, MeHg in fish
poses a potentially significant health risk to more highly exposed tribal
sub-populations. Tribal fish intakes and exposures are greater than for
other ethnicity groups in NHANES, and critical tribal fish consumption
exposure factors are fish species, location, and weight. Reducing con-
sumption of fish species with the highest MeHg concentrations, even
while eating the same total amount of fish, can significantly reduce ex-
posures. Bass, bowfin and walleye are the most contaminated fresh
water species for MeHg of the fresh water species where data were
available. In addition, lognormal distribution is the best fit for MeHg
fish tissue concentrations in our tests and this can be used in future di-
etary exposure assessments; however, it could overestimate MeHg ex-
posure especially for high percentile.

This research has identified key factors for health risks from tribal
fish consumption of MeHg, and provided results to inform risk man-
agement decisions. If tribes do not want to reduce their fish intake,
they could consider eating less contaminated species to minimize
their exposures. Future research can apply exposure and GIS tools
to inform tribal risk mitigation decisions for other persistent pollutants, such as PCBs.

5. Conclusion

MeHg exposures from dietary fish consumption in tribes are about 3 to 10 times higher than the US general population, which implies correspondingly higher potential health risks for tribal populations compared to the general US population. As much as ~50% of MeHg dietary exposures can be reduced just by replacing several species of fish with high MeHg concentration (e.g., walleye, bowfin), substituting species with lower concentrations. The exposure assessment methods we used can inform tribal decisions on how to reduce dietary exposures.

Fig. 5. Modeled MeHg exposures vs. biomarker data.

Disclaimer

This manuscript has been reviewed and approved for publication.

Competing interest declaration

The authors declare no conflict of interest.

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Appendix A. Supplementary data
Supplementary data to this article can be found online at http://dx.
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Fig. 6. Modeled exposure statistics over time.

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References

Fig. 6. Modeled exposure statistics over time.