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Methyl mercury exposure from fish consumption in vulnerable racial/ethnic populations: Probabilistic SHEDS-Dietary model analyses using 1999–2006 NHANES and 1990–2002 TDS data ☆

Jianping Xue *, Valerie G. Zartarian, Shi V. Liu, Andrew M. Geller

U.S. Environmental Protection Agency, Office of Research and Development, National Exposure Research Laboratory, 109 T.W. Alexander Drive, Research Triangle Park, NC 27711, United States

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ABSTRACT

NHANES subjects self-identified as "Asian, Pacific Islander, Native American, or multiracial" (A/P/N/M) have higher levels of blood organic mercury than other racial/ethnic groups; however, the reasons for this have been unclear. This research uses exposure modeling to determine the reasons for elevated blood methylmercury (MeHg) levels, and also extends previous analyses of observed NHANES blood levels. The probabilistic SHEDS-Dietary model was applied, using MeHg fish residue data from FDA's Total Diet Study (1990–2002) combined with NHANES/WWEIA (1999–2006) fish consumption data, to generate exposure estimates by race/ethnicity, age group, and fish type. Statistical analyses of blood methylmercury levels in the (6 times larger) 1999–2006 NHANES data were compared against previous published results for 1999–2002 data.

The A/P/N/M group has higher fish intake, modeled MeHg exposures, and blood levels than the general population and other racial/ethnic groups. Tuna, other saltwater fish, and other freshwater fish are key food types driving dietary MeHg exposure. The 1–<3 years-old A/P/N/M group has the highest mean dietary MeHg intake per body weight (0.06 μg/kg/day; ~2.3 times higher than the rest of the population). Fish intake and modeled exposure predictions correlate well with NHANES blood biomarker levels. This study, using the SHEDS-Dietary model with national data, reinforces and expands upon previous observations that dietary exposure via fish consumption is an important route for methylmercury intake by the general population, and especially for racial/ethnic groups with higher fish consumption. These probabilistic dietary modeling approaches could be applied for local populations (e.g., tribes) and other chemicals and foods, if data are available.

1. Introduction

Blood organic mercury, or methyl mercury (MeHg), is a form of mercury (Hg) with potent neurotoxicity and teratogenicity effects (WHO, 1990; Mahaffey, 1998). Since the recognition of Minamata disease as an outcome of high-level MeHg exposure, efforts have been made in reducing the uncontrolled release of mercury compounds (Harada, 1995). However, MeHg is still present in many environments due to historical pollution and ongoing use of mercury compounds (Harada et al., 1998; U.S. EPA, 2001). Some studies suggest that exposure to low concentrations of MeHg can still pose some health concerns (Mancini et al., 2009: Yokoo et al., 2003).

For the general human population, the major route for MeHg exposure is dietary exposure via consuming seafood, especially fish (Bjornberg et al., 2005; Carvalho et al., 2008; Diez, 2009; Knobeloch et al., 2007; Sirot et al., 2008; Zhang et al., 2009). Two studies based on the 1999 and 2000 National Health and Nutrition Examination Survey (NHANES) data found that the geometric mean of hair Hg concentration was 0.12 μg/g in children and 0.20 μg/g in women in the general U.S. household population (McDowell et al., 2004), and that blood organic mercury reflects MeHg intake from fish and/or shellfish (Mahaffey et al., 2004). A small scale study containing 14 people (Knobeloch et al., 2006) and a later expanded study including more than 2000 adults (Knobeloch et al., 2007) from Wisconsin in the

Abbreviations: A/P/N/M, Asian, Pacific Islander, Native American or multiracial; CDF, cumulative distribution function; EPA, U.S. Environmental Protection Agency; FDA, Food and Drug Administration; GLM, general linear model; Hg, mercury; MeHg, methyl mercury; NATA, National Air Toxics Assessment; NERL, National Exposure Research Laboratory; NHANES, National Health and Nutrition Examination Survey; ORD, Office of Research and Development; PBPK, physiologically-based pharmacokinetic; RAC, raw agricultural commodity; SHEDS, Stochastic Human Exposure and Dose Simulation (model); TDS, Total Diet Study; US, United States; WWEIA, What We Eat in America.

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* Corresponding author at: U.S. EPA, 109 T.W. Alexander Drive, MD E205-02, Research Triangle Park, NC 27711, United States.

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U.S. found the average hair Hg level to be 0.714 μg/g, and concluded that MeHg exposure may be correlated with fish consumption (Knobeloch et al., 2007).

A study using the 1999–2000 NHANES data found that the geometric means of hair mercury concentrations in frequent fish consumers were higher than others, and non-Hispanic black and Mexican–American children had higher hair Hg levels than non-Hispanic white children (Mahaffey et al., 2004). Another study using 1999–2002 NHANES data found that participants self-identified as Asian, Pacific Islander, Native American, or multiracial (abbreviated here as the A/P/N/M group) have higher percentage over certain blood mercury levels than others (Hightower et al., 2006). This A/P/N/M group also seemed to have higher mean levels of consumption of fish and shellfish, but the 95% confidence intervals overlapped with the remaining groups except Mexican Americans. Hightower et al. (2006) recommended that future studies should explore reasons for the high mercury levels in this group to help inform risk reduction actions. This paper explores the reasons by using human exposure modeling with large national data sets for fish consumption and MeHg residues.

This paper uses the expanded 1999–2006 NHANES to extend findings from previous studies in two ways:

1) Fish consumption data from NHANES are used as model inputs to the U.S. Environmental Protection Agency (EPA)'s physically-based, probabilistic model called SHEDS-Dietary (Stochastic Human Exposure and Dose Simulation — Dietary model) (Xue et al., 2010a,b) to produce estimates of MeHg exposure stratified by age (including young children), sex, and race/ethnicity, concentrating on A/P/N/M groups and attributing estimated MeHg exposure to consumption of specific fish and shellfish.
2) SHEDS-Dietary model estimates are evaluated against blood biomarkers for MeHg collected by NHANES from the same individuals who were surveyed for the fish consumption data.

Using large data sets of food consumption from NHANES/What We Eat in America Survey (WWEIA), fish organic MeHg residue data (i.e., bioaccumulated MeHg levels measured in fish) from the U.S. Food and Drug Administration (FDA)'s Total Diet Study (TDS) (FDA, Food, Drug Administration, 1990–2002 and most data from 1991 to 1997), and blood biomarkers from NHANES (same individuals as for fish consumption data), we demonstrate that fish consumption can be an important MeHg exposure route, especially for A/P/N/M populations.

2. Methods

2.1. Fish consumption data

Data from the 1999–2006 NHANES/WWEIA survey for A/P/N/M subjects were used for model inputs regarding the amount of fish consumed by individuals. This database contains 55,000 person-days of real-time dietary consumption data, i.e., amounts of foods (including fish) recorded instantly by individuals for each separate eating occasion. The NHANES sample size used here for participants who designated themselves as A/P/N/M was >6 times larger than in earlier studies (Hightower et al., 2006; Mahaffey et al., 2004).

2.2. MethylMercury (MeHg) concentrations in fish

MeHg residue data from the FDA's TDS (FDA, Food, Drug Administration, 1990–2002), also known as the market basket study (1990–2002), were combined with consumption data in SHEDS-Dietary. TDS collects and analyzes ~280 foods for pesticide residues, industrial chemicals, and toxic and nutrient elements. Foods collected in the TDS are prepared as "table-ready," i.e., as would be consumed, for realistic estimates of dietary intake of those targeted components. Approximately 40 different fish and shellfish were measured for MeHg.

2.3. Exposure modeling

To estimate the exposure of MeHg from fish by race/ethnicity, age group, and food type, we used EPA's population-based probabilistic dietary exposure model. SHEDS-Dietary simulates individual exposures to chemicals in food and drinking water over different time periods (e.g., daily, yearly) (Xue et al., 2010a,b). The SHEDS-Dietary model is one module of EPA's more comprehensive human exposure model, SHEDS-Multimedia, which can simulate aggregate or cumulative exposures over time via multiple routes of exposure (dietary and non-dietary) to multiple types of chemicals (http://www.epa.gov/heasd/products/sheds_multimedia/sheds_mm.html; Zartarian et al., 2006; Xue et al., 2006a, 2006b). SHEDS-Dietary can produce 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 either USDA's Continuing Survey of Food Intake by Individuals (CSFII) (1994–96, 1998) or the NHANES/WWEIA dietary consumption data (1999–2006), along with EPA/USDA recipe translation files (FCID; Food Commodity Intake Database), and available food and water concentration data. Combining this information requires a number of technical considerations, such as translating foods reported as eaten into raw agricultural commodities using recipe files, sampling residues within a day and over time, considering non-detects, and allocating total drinking water consumption into within-day drinking water events (Xue et al., 2010a). As another example, about 40 types of fish and shellfish were mapped from TDS into food items in the NHANES/WWEIA data.

In this paper, SHEDS-Dietary was used for calculating dietary MeHg exposures for each eating occasion of individuals; estimating the ranges of population dietary exposures; and identifying key factors and contributions of food types. Consumption data for all foods was used, but only MeHg residue data for fish were included in the simulations. The unmatched NHANES/WWEIA foods consumed were matched again by raw agricultural commodities (RAC), using the recipe files in EPA's Food Consumption Intake Database (FCID; U.S. EPA, 2000), so that exposures for those foods could be calculated. Each simulated individual's exposure for each fish type was calculated by multiplying total daily consumption with corresponding residues. Aggregate daily exposure was calculated by summing exposures across all commodities. Summation of MeHg exposures from every eating occasion for 1 day yielded the individual's daily MeHg exposure. Details on the probabilistic dietary exposure algorithms in SHEDS are given in Xue et al., 2010a,b. Although SHEDS-Dietary can be used to model longitudinal dietary exposures, this research addressed only the cross-sectional exposures, due to data limitations. The SHEDS-Dietary model was applied to obtain cumulative distribution functions (CDFs) for population dietary MeHg exposure estimates, calculate corresponding summary statistics by age, gender, and race/ethnicity, and conduct analyses for key fish contributors to exposure.

2.4. Biomarker analyses of blood methyl mercury (for exposure model comparison)

A total of 21,278 samples were collected for blood MeHg in the 1999–2006 NHANES. Blood MeHg biomarker data from the same individuals with fish consumption data and 30 day recall fish consumption from the NHANES were used for comparing against SHEDS-Dietary model predictions during the same time period as

the consumption data were collected. A total of 13,344 records of matched data for fish consumption, 30 day recall fish consumption, and biomarkers were used for the general linear model (GLM) analysis conducted.

We analyzed the 1999–2006 NHANES blood biomarker data for MeHg, to compare with the SHEDS-Dietary model MeHg estimates, with a larger NHANES sample size than previously published results (Hightower et al., 2006; Mahaffey et al., 2004) (NHANES 1999–2006 sample size is 6 times larger than the 1999–2002 database). To conduct analyses of variance contributions of blood MeHg, a general linear model (GLM) was applied using the largest and most comprehensive data available to identify key factors. These data were merged for the same subjects and a GLM was applied with blood MeHg as the dependent variable, to identify the risk factors.

3. Results

3.1. Fish consumption

Table 1 shows total fish intake (grams fish consumed) by race/ethnicity and age group, from 1999 to 2006 NHANES/WWEIA data, used in the SHEDS-Dietary exposure analyses. Fish intake is higher for the A/P/N/M group than other groups; it is approximately 1.4–2.9 times greater than the other ethnic groups combined across all age groups, whether calculated as grams consumed per day or adjusted by body weight.

3.2. Methyl mercury exposure

Table 2 presents SHEDS modeled MeHg exposure estimates by race/ethnicity and age group, based on the FDA's 1990–2002 TDS residue data and the 1999–2006 NHANES dietary consumption data. This table indicates that A/P/N/M populations are exposed to higher levels of MeHg from fish consumption than other racial/ethnic groups in the general U.S. population, ranging from factor of 1.3 to 2.8 times higher (means based on mass per day) except for the 11–<16 year age group.

The mean modeled MeHg exposures from fish for A/P/N/M groups ranges from 0.01 to 0.06 μg/kg/day over different age groups, compared to 0.01 to 0.03 μg/kg/day in the general population. For all age groups except 11–15 year-olds, the A/P/N/M groups have higher mean dietary MeHg exposures, ranging from a factor of 1.5 to 2.5 times higher than the general population. *The pattern is similar between total fish intake by ethnicity and age group (Table 1) and SHEDS MeHg exposure predictions by ethnicity and age group (Table 2)*. The 1-year-old A/P/N/M group has the highest ratio (2.8 with exposure as μg/day; 2.5 if μg/kg/day), for SHEDS modeled mean MeHg exposures, compared to the same age group for other ethnicities. The 1–<3 year-old A/P/N/M groups also have the highest dietary modeled MeHg exposure (intake) per body weight (0.27 and 1.31 μg/kg/day for 1 year-olds, 0.26 and 1.44 for 2 year-olds at the 95th and 99th percentiles, respectively). Fig. 1 illustrates the SHEDS modeled MeHg exposure by ethnicity: A/P/N/M is highest, Mexican–American is lowest.

The contribution by fish type to total MeHg exposure in A/P/N/M group is depicted in Fig. 2. The major fish contributors are tuna fish (31%), saltwater fish – other (30%), fresh water fish – other (27%), shrimp (5%), shellfish (4%), and catfish (3%).

Table 1
Total fish intake by ethnicity and age group from 1999 to 2006 NHANES data.

			g/day				g/kg/day			
Ethnicity	Age	N	Mean	Ratio[a]	P95	P99	Mean	Ratio[a]	P95	P99
A/P/N/M	<1	170	0.66	1.5	0.0	31.8	0.07	1.4	0.0	3.3
	1–2	185	7.96	2.9	55.8	101.2	0.61	2.8	4.7	7.4
	3–5	200	6.29	1.5	38.8	114.9	0.35	1.5	2.3	6.7
	6–12	402	11.90	2.0	69.9	195.6	0.39	2.2	2.2	8.6
	13–19	463	12.21	1.8	64.6	274.6	0.22	2.0	1.2	4.8
	20–49	636	26.73	1.8	167.1	327.9	0.40	2.1	2.4	4.1
	50+	366	29.14	1.7	178.4	315.9	0.44	2.0	2.8	5.2
Rest	<1	2517	0.45		0.0	8.5	0.05		0.0	0.8
	1–2	3326	2.71		15.2	75.5	0.22		1.3	6.0
	3–5	3153	4.29		32.0	99.8	0.23		1.7	5.4
	6–12	7390	6.10		45.9	129.6	0.17		1.3	3.8
	13–19	10955	6.95		47.7	160.1	0.11		0.7	2.6
	20–49	13707	15.22		108.8	259.2	0.19		1.4	3.3
	50+	11530	17.46		120.4	255.0	0.22		1.5	3.2

[a] Ratio of (A/P/N/M) over other ethnicities.

Table 2
SHEDS MeHg exposure predictions by ethnicity and age from 1999 to 2006 NHANES data.

			μg/day				μg/kg/day			
Ethnicity	Age	N	Mean	Ratio[a]	P95	P99	Mean	Ratio[a]	P95	P99
A/P/N/M	0<1	170	0.1	2.1	0.0	4.1	0.01	2.2	0.00	0.39
	1 to <2	89	0.7	2.8	3.7	17.8	0.06	2.5	0.27	1.31
	2 to <3	96	0.8	1.9	3.7	18.7	0.06	2.1	0.26	1.44
	3 to <6	200	0.6	1.3	3.0	19.4	0.04	1.5	0.13	1.61
	6 to <11	285	1.7	2.5	3.1	73.4	0.05	2.3	0.13	1.96
	11 to <16	316	0.4	0.5	2.0	11.8	0.01	0.6	0.04	0.19
	16 to <21	296	1.1	1.3	5.6	23.8	0.02	1.5	0.08	0.49
	21 to <50	604	2.5	1.7	13.9	40.8	0.04	1.9	0.24	0.55
	50+	366	2.9	1.6	17.4	54.1	0.04	1.8	0.22	0.71
Rest	0<1	2517	0.0		0.0	0.4	0.01		0.00	0.05
	1 to <2	1704	0.3		0.5	6.3	0.02		0.05	0.53
	2 to <3	1622	0.4		1.5	10.1	0.03		0.09	0.79
	3 to <6	3153	0.5		1.5	12.6	0.03		0.09	0.63
	6 to <11	4815	0.6		1.4	18.1	0.02		0.05	0.62
	11 to <16	7305	0.8		2.1	25.4	0.01		0.04	0.44
	16 to <21	6721	0.8		2.2	23.0	0.01		0.03	0.34
	21 to <50	13211	1.5		6.6	32.8	0.02		0.09	0.43
	50+	11530	1.8		9.5	38.5	0.02		0.13	0.47

[a] Ratio of (A/P/N/M) over other ethnicities.

3.3. Blood methyl mercury

Table 3 presents summary statistics of MeHg blood level by age group and race/ethnicity (ug/L) from the 1999–2006 NHANES data. These blood levels of MeHg are consistent with SHEDS-modeled MeHg exposures presented above. This table also shows that 1-year-old in the A/P/N/M group have the highest ratio of blood MeHg levels (ranging from a factor of 1.4 to 3.0 times higher) compared to corresponding age groups in the general U.S. population.

Table 4 shows the prevalence of high MeHg blood concentrations across racial/ethnic groups derived from NHANES data, using as a criterion the U.S. EPA's reference dose for MeHg (5.8 μg/L) and a more conservative reference level (3.5 μg/L) based on estimates of higher levels of MeHg in cord blood than in maternal blood (Rice, et al., 2003). A/P/N/M populations have the highest prevalence of MeHg blood concentrations exceeding the critical health-based reference doses compared to other racial/ethnic groups (Mexican–Americans have the lowest levels); 8.4% of their MeHg blood levels are higher than U.S. EPA's reference dose.

Table 5 presents similar results specific to each age group, comparing A/P/N/M to the rest of the population combined from NHANES. The percentage of high MeHg blood level of A/P/N/M populations is consistently higher than other ethnicities across corresponding age groups. Also, the percentage above the reference values generally increases as age increases: the ratio is lower, however, for two age groups (3–5 year-olds, and 13–19 year-olds).

Fig. 3 illustrates blood MeHg by race/ethnicity from 1999 to 2006 NHANES data. Concentrations of blood MeHg for A/P/N/M populations are highest and for Mexican–Americans are lowest. Blood levels

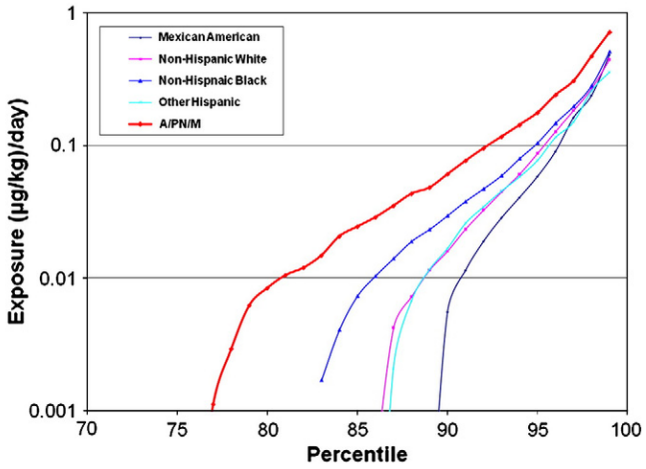

Fig. 1. MeHg exposure predictions of all ages by ethnicity from SHEDS from 1999 to 2006 NHANES data.

Table 3
Summary statistics of blood MeHg level by age group and ethnicity (µg/l) from 1999 to 2006 NHANES data.

Ethnicity	Age (year)	N	Mean	Ratio[a]	P95	P99
A/P/N/M	1 to <2	36	0.76	3.0	4.40	4.40
	2 to <3	42	0.59	2.2	2.21	4.00
	3 to <6	91	0.57	1.5	3.40	6.82
	6 to <11	96	1.01	2.7	4.40	6.80
	11 to <16	94	0.69	1.5	3.03	4.70
	16 to <21	126	1.24	2.0	4.90	6.71
	21 to <50	270	1.69	1.6	5.52	6.63
	50+	108	1.70	1.4	5.03	5.78
Rest	1 to <2	716	0.25		0.92	2.46
	2 to <3	726	0.27		1.10	2.42
	3 to <6	1570	0.38		1.62	3.60
	6 to <11	1336	0.38		1.40	3.63
	11 to <16	2089	0.45		1.76	4.35
	16 to <21	3117	0.62		2.32	5.20
	21 to <50	6339	1.04		3.81	6.30
	50+	3510	1.18		4.00	6.00

[a] Ratio of (A/P/N/M) over other ethnicities.

of MeHg are consistent with MeHg exposures estimated by SHEDS-Dietary, as shown in Fig. 1; modeled exposure predictions correlate well with NHANES blood biomarker levels in terms of age, gender, and ethnicity.

Fig. 4 shows similar results by ethnicity and age group. The MeHg blood levels of A/P/N/M populations are consistently higher than those for other racial/ethnic groups across age groups, and the levels increase as age increases. The similar patterns of modeled dietary exposure predictions and NHANES blood biomarker levels (in terms of age, gender, and ethnicity) also suggest that fish consumption is a key exposure pathway for the A/P/N/M populations.

Table 6 shows MeHg levels by ethnicity and number of fish and shell fish consumed in 30 days. These results indicate that the MeHg blood levels in the A/P/N/M populations are higher than for other racial/ethnic groups, even based on 30 day fish consumption recall data. For other racial/ethnic groups, the blood MeHg level generally increases as fish consumption increases, and the percentage above critical levels generally increases. For the A/P/N/M populations, this trend is not consistent, mainly due to small sample size. The MeHg blood levels in the A/P/N/M populations are higher than other racial/ethnic groups, and the difference is statistically significant with p value less than 0.0001 using the *t*-test.

Based on analyses of variance contributions of blood MeHg, using GLM, age is the biggest contributor, followed by modeled MeHg exposure from SHEDS, 30-day recall fish consumption, and race/ethnicity (overall p-value<0.0001). While exposures to males were estimated to be higher than exposures to females, gender accounted for only a modest portion of the variance.

4. Discussion

Significant levels of MeHg in some blood samples from people in the U.S. population participating in the NHANES (Mahaffey et al., 2004; Hightower and Moore, 2003) deserve attention because the survey was conducted after decades of effort to reduce Hg pollution and strengthen food safety against MeHg exposure. Using the probabilistic SHEDS-Dietary model, which simulates exposures for actual eating occasions, we conducted comprehensive analyses on modeled human exposure to MeHg from fish consumption and measured blood MeHg concentrations for different racial/ethnic groups, and identified some information needs. Our study examined the relationship between fish consumption, exposure, and blood levels; it revealed that 1–<3 year olds have highest modeled MeHg exposures (average of 0.06 µg/kg/day); and confirmed the findings of previous studies that the A/P/N/M subjects in the NHANES have higher levels across age groups of modeled MeHg exposure than the rest of the ethnicities which include black, non-Hispanic white, Mexican American, and Hispanic. The main reason that the A/P/N/M group had higher exposures than the other groups is their higher fish intake. Estimated exposure is highest for 1–<3 year-olds because

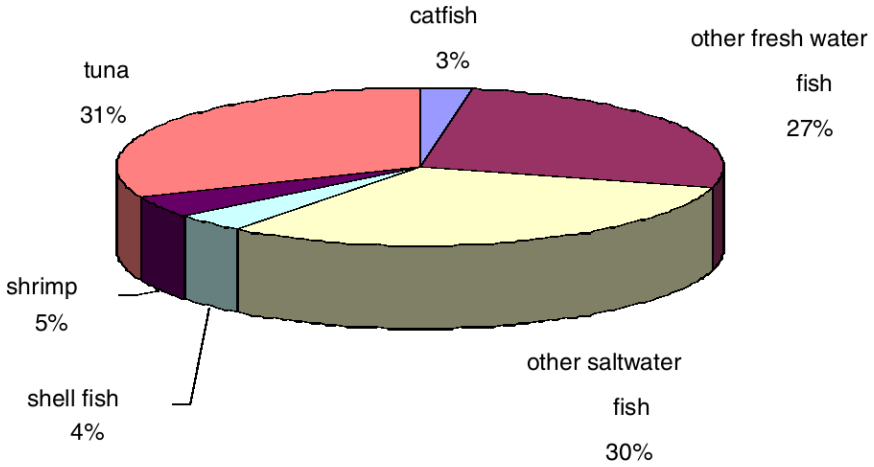

Fig. 2. Contribution of MeHg exposure from different fish types for A/P/N/M group.

Table 4
Percentage with high MeHg blood level by ethnicity from 1999–2006 NHANES data.

Ethnicity	Percentage above	
	3.5 μg/L	5.8 μg/L
Mexican American	2.7	0.7
Non-Hispanic White	7.4	3.2
Non-Hispanic Black	6.6	2.3
Other Hispanic	7.5	2.5
A/P/N/M	18.2	8.4

of higher intake and lower body weight (exposure was weighted by body weight). More research is needed to explore other reasons.

In general, the SHEDS-Dietary MeHg exposure modeling results and the NHANES biomarker data are consistent among age groups. Comparing Figs. 1 and 3, blood dose has higher variability than exposure because of dosimetry considerations and body effects. Their ethnicity pattern, however, is the same. While the percentage of A/P/N/M MeHg blood levels above the reference values generally increases as age increases, the ratio is lower, for two age groups (3–5 year-olds, and 13–19 year-olds); we think this is due to small sample size for the higher criterion of 5.8 μg/L. This research also revealed that tuna, other saltwater fish, and other freshwater fish are the key food types driving dietary MeHg exposure. Even though shark and swordfish had the highest concentrations, their contributions to exposure were not significant because intake of those fish types was lower.

A number of studies hypothesized that seafood/fish is the most prominent source of MeHg in blood (Mahaffey et al., 2004; McDowell et al., 2004; Hightower and Moore, 2003) and identified the A/P/N/M groups as "high-risk" for MeHg blood levels, as compared to the other groups. It was suggested (Hightower et al., 2006) that future studies should address the reasons for this observation. Our results are consistent, in general, with those reported in previous studies. The NHANES/WWEIA consumption data used in SHEDS-Dietary compare well to the EPA Child-Specific Exposure Factors Handbook (U.S. EPA, 2008), Tables 10–20, Fish Consumption Rates among Native American Children. For 3–5 year olds, NHANES A/P/N/M values vs. EF Handbook values: 95th percentile: 40 v.s. 50 g per day; 99th percentile: 115 v.s. 100 g per day (Sample size = 194, data compiled from the Umatilla, Nez Perce, Yakama, and Warm Springs tribes of the Columbia River Basin). Our modeled MeHg exposure results are also consistent with other studies such as Japanese research reporting that fish consumption accounts for approximately 80–90% of the total human mercury exposure in Japan (MHLW, 2004) and that the mean MeHg intake for the Japanese population was estimated to be 6.76 μg/day or 0.14 μg/kg body weight per day (bw/day) with a mean value for the hair Hg level as 2.02 μg/g (Zhang et al., 2009). The estimated 99th percentile exposures of 1 to 10 year olds (see Table 2) are about 3 times as high as the Joint FAO/WHO Expert Committee on Food Additives Provisional Tolerable Weekly Intakes, expressed on a daily basis as 0.47 μg/kg/day (FAO/WHO, 1999). Finally, our analyses of the blood organic mercury data with the expanded NHANES database are consistent with the previous NHANES analyses (Mahaffey et al., 2004). Our research reveals that the significant contributors to MeHg blood levels are the following factors: age, MeHg exposure (SHEDS modeled estimates), fish consumption, and race/ethnicity.

Table 5
Percentage with high MeHg blood level by ethnicity and age from 1999 to 2006 NHANES data.

Age (yr)	Above 3.5 μg/L			Above 5.8 μg/L		
	A/P/N/M	REST	Ratio[a]	A/P/N/M	Rest	Ratio[a]
1–2	5.3	0.6	8.4	0.6	0.3	2.0
3–5	3.5	1.2	2.8	0.3	0.6	0.5
6–12	7.9	1.3	5.8	0.5	0.2	3.0
13–19	9.1	1.7	5.4	0.3	0.9	0.4
20–49	22.1	8.3	2.7	4.0	3.9	1.0
50+	25.3	9.6	2.6	4.9	3.7	1.3

[a] Ratio of (A/P/N/M) over other ethnicities.

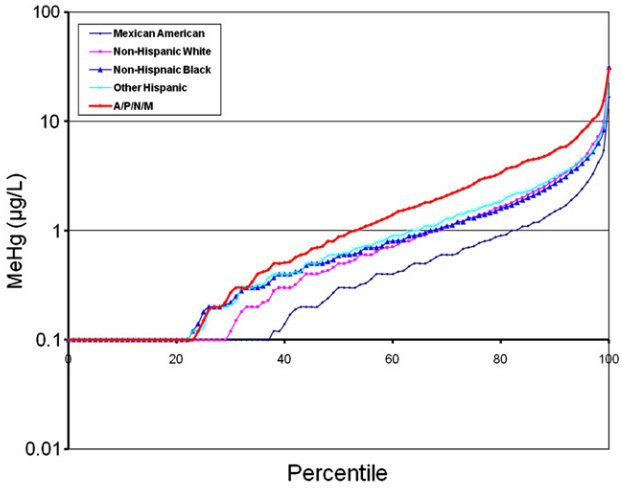

Fig. 3. Blood MeHg by ethnicity from 1999 to 2006 NHANES data.

The focus of the MeHg exposure analyses in this paper was fish consumption exposures; however, we did a screening comparison to inhalation exposures using the EPA's National Air Toxics Assessment (NATA). In the 2002 NATA database, the average exposure to Hg from air for the U.S. population is 5.54E-05 μg/m^3; assuming 20 m^3/day for inhalation rate (a child usually has less than 10 m^3/day), the exposure of Hg from air is about 0.0011 μg/day. In comparison with Table 2, modeled MeHg exposure from fish consumption is about a thousand times higher than from air. In the future we could do more refined air analyses for tribal populations using NATA, but the fish consumption exposure route is much more significant based on the current analyses.

To our knowledge, the dietary exposure modeling analyses presented in this paper for MeHg have not been attempted before for these vulnerable populations in the exposure-related literature. The modeling methods and analyses presented here could be applied to other chemicals, and to assess cumulative risks (incorporating health benefits) from fish consumption. They could also be used to inform risk reduction strategies related to fish consumption, e.g.

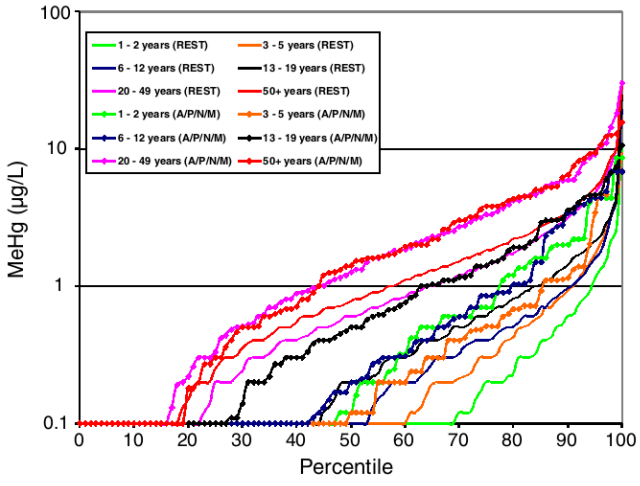

Fig. 4. Blood MEHg by ethnicity and age group from 1999 to 2006 NHANES data.

Table 6
Blood MeHg levels by ethnicity and number of fish and shell fishes consumed in previous 30 days.

Ethnic	Number of fish eaten in 30 days	sample size	Average blood MeHg level (μg/L)	Percent above	
				3.5 μg/L	5.8 μg/L
Others	0	7049	0.78	6.0	2.5
Others	1–5	2244	0.70	3.2	1.3
Others	6–10	941	1.00	6.6	2.3
Others	11–20	858	1.17	9.8	4.1
Others	21–30	463	1.37	11.8	5.1
Others	30+	372	1.95	24.2	13.9
A/P/N/M	0	291	1.41	20.6	7.8
A/P/N/M	1–5	75	0.96	1.8	8.3
A/P/N/M	6–10	51	2.18	29.3	8.5
A/P/N/M	11–20	40	2.50	43.6	30.5
A/P/N/M	21–30	30	1.34	16.9	11.8
A/P/N/M	30+	20	2.87	39.6	13.5

types of fish to avoid such as shark and swordfish (Mahaffey et al. 1994), water bodies for targeted cleanup, recommended meal sizes. Such mitigation approaches could be beneficial to both the general population and specific vulnerable populations such as local communities and tribes who rely on subsistence fishing. However, many research and data needs remain for local-scale assessments involving fish consumption exposures/risks. Because this study used national rather than tribal-specific fish consumption and residue data, and Native Americans are grouped with Asians, Pacific Islanders, and multiracial groups in NHANES/WWEIA, it is difficult to draw tribal-specific conclusions or suggest specific risk reduction recommendations. Future research that would be useful includes the following:

- assessing importance of eating occasions and longitudinal analyses in SHEDS-Dietary for MeHg (with long half-life) and other chemicals in fish;
- linking SHEDS-Dietary exposure analyses with physiologically-based pharmacokinetic (PBPK) models for dose predictions;
- collecting detailed consumption and residue data at the local scale to identify specific type of fish consumed and concentrations in those fish for specific community or tribal assessments; and
- 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).

5. Conclusions

The health effects of methyl mercury and higher blood levels in A/P/N/M populations have been studied previously. This research extends and is consistent with findings from previous studies. Our probabilistic dietary exposure modeling assessment advances the science by using the large and recent data bases from NHANES and TDS to explore reasons for higher MeHg blood levels, and to estimate MeHg exposure via fish consumption by age, gender, race/ethnicity (specifically, A/P/N/M groups), and type of fish. The SHEDS-Dietary model, previously evaluated for arsenic in Xue et al., 2010a, yielded MeHg predictions consistent with the NHANES blood level data. In addition, the blood organic mercury analyses in this paper using the expanded NHANES database are consistent with previous results. We found that A/P/N/M racial/ethnic groups have higher dietary MeHg exposures from fish consumption than other populations. Correlations of modeled dietary exposure predictions with NHANES blood biomarker levels suggest that fish consumption is a key exposure pathway for these populations.

Our study reinforces and expands upon previous observations that dietary exposure via fish consumption is an important route for MeHg intake by the general population and an even more important route for racial/ethnic groups with higher fish consumption rates. Specific results in this paper for updated fish consumption rates, with the most recent NHANES data, could be used in future to update the EPA Exposure Factor Handbooks (U.S. EPA, 2008). The contributing fish analyses for exposure could be used for public dissemination of risk reduction actions regarding fish intake of specific types.

In summary, our conclusions are the following: (1) SHEDS-Dietary modeling confirms that A/P/N/M populations have higher MeHg exposures than the rest of the population, and allows identification of MeHg intakes by age, gender, ethnicity, and type of fish; (2) Correlations of modeled dietary exposure predictions with NHANES blood biomarker levels suggest that fish consumption is a key exposure pathway for these populations; (3) Statistical analyses of blood methylmercury levels in the (6 times larger) 1999–2006 NHANES database were consistent with previous published results for 1999–2002 data; (4) More research with local-scale data is needed to better estimate fish consumption exposure for specific communities and tribes; and (5) This research has important implications for risk reduction recommendations related to fish consumption.

6. Competing interests declaration

All authors declare no potential competing financial interests.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at doi:10.1016/j.scitotenv.2011.10.010.

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