



Published in final edited form as:

J Expo Sci Environ Epidemiol. 2020 May ; 30(3): 504–514. doi:10.1038/s41370-020-0206-6.

Seafood, wine, rice, vegetables, and other food items associated with mercury biomarkers among seafood and non-seafood consumers: NHANES 2011–2012

Ellen M. Wells, PhD^{1,2,*}, Leonid Kopylev, PhD³, Rebecca Nachman, PhD³, Elizabeth G Radke, PhD³, Deborah Segal, MEHS³

¹School of Health Sciences, Purdue University, West Lafayette, Indiana, USA

²Department of Public Health, Purdue University, West Lafayette, Indiana, USA

³National Center for Environmental Assessment, Office of Research and Development, US Environmental Protection Agency, Washington DC, USA

Abstract

Fish/seafood consumption is a source of mercury; other dietary sources are not well described. This cross-sectional study used National Health and Nutrition Examination Survey (NHANES) 2011–2012 data. Participants self-reported consuming fish/seafood (N=5427) or not (N=1770) within the past 30 days. Whole blood total mercury (THg), methylmercury (MeHg) and urinary mercury (UHg) were determined. Diet was assessed using 24-hour recall. Adjusted regression models predicted mercury biomarker concentrations with recent food consumption while controlling for age, sex, education, and race/ethnicity. Geometric mean THg was 0.89 µg/L (95% confidence interval (CI): 0.78, 1.02) (seafood consumers) and 0.31 µg/L (95% CI: 0.28, 0.34) (non-seafood consumers); MeHg and UHg concentrations follow similar patterns. In adjusted regressions among seafood consumers, significant associations were observed between mercury biomarkers with multiple foods, including fish/seafood, wine, rice, vegetables/vegetable oil, liquor and beans/nuts/soy. Among non-seafood consumers, higher THg was significantly associated with mixed rice dishes, vegetables/vegetable oil, liquor and approached statistical significance with wine ($p<0.10$); higher MeHg was significantly associated with wine and higher UHg was significantly associated with mixed rice dishes. Fish/seafood consumption is the strongest dietary predictor of mercury biomarker concentrations; however, consumption of wine, rice, vegetables/vegetable oil, or liquor may also contribute, especially among non-seafood consumers.

Keywords

mercury; methylmercury; biomarker; diet; seafood; National Health and Nutrition Examination Survey

Users may view, print, copy, and download text and data-mine the content in such documents, for the purposes of academic research, subject always to the full Conditions of use:http://www.nature.com/authors/editorial_policies/license.html#terms

*Corresponding author: Ellen M. Wells; 550 Stadium Mall Drive, West Lafayette, IN 47906, United States; wells54@purdue.edu.

Conflict of Interests: The authors declare they have no actual or potential competing financial interests.

Introduction

The World Health Organization (WHO) has identified mercury as one of top ten chemicals of public health concern (1). Numerous reports suggest that exposure to mercury poses a significant threat to human health because mercury is toxic to multiple organ systems including the nervous, renal, respiratory, immune, and cardiovascular systems (2–4). There are different chemical forms of mercury (elemental, inorganic, and organic): the most common form of mercury that humans are exposed to is methylmercury, a form of organic mercury. The different chemical forms of mercury are recognized to have different typical routes of exposure and toxicological impact, as described in more detail below.

The nervous system and renal system are the primary targets of chronic exposure to elemental mercury and/or inorganic mercury (5,6). Neurotoxicity is also a major concern of methylmercury exposure (7–9). Moreover, the developing nervous system is more sensitive to the neurotoxicity of methylmercury than the mature nervous system (10,11). Therefore, exposure to methylmercury, especially among pregnant women and infants and children, is a major concern (12,13). Additional noted health effects of methylmercury exposure include cardiovascular (14–16) and immune system toxicity (17).

Human exposure to mercury is the result of a complex global patterns of environmental release, fate, and transport (18). Workers in industrial operations or mining (either formal or informal/artisanal) operations may be exposed to elemental or inorganic mercury (19–21). Elemental mercury is used in dental amalgams, which has been traced to exposure among dentists (22) and persons with dental fillings (23–25). Mercury exposure has been identified from use of inorganic mercury-containing consumer products, such as skin lightening cream (26) or herbal medicines (27). Numerous studies support the observation that the majority of mercury exposure in humans occurs via consumption of methylmercury via fish or seafood (12,28).

An increasing amount of research suggests that human exposure to total mercury or methylmercury may occur via consumption of items other than fish or seafood. Several studies in China (29–32) and one in the United States (33) suggest rice consumption may be associated with methyl or total mercury exposure. Baby rice cereals and other rice-containing baby products also contain methylmercury (34). Mercury exposure may also be associated with consumption of vegetables (35–38), grains (35,36,39), alcoholic beverages (35,38,40,41), herbal tea (41), and high fructose corn syrup (42). Some studies have reported a negative association of blood mercury with specific foods, such as foods containing tomatoes, potatoes, or meats (41,43); it is possible this negative association reflects overall dietary patterns, i.e., that people eating tomatoes, potatoes or meats also tend to eat fewer foods that have high mercury content (41).

There are still unanswered questions on non-seafood dietary predictors of mercury exposure. Dietary patterns and source of foods vary greatly by region; thus, studies completed in one population may not necessarily reflect the experience of others. While there are a few existing studies of the United States population using National Health and Nutrition Examination Survey (NHANES) data (33,44), these are limited by evaluating only total

mercury biomarkers and not incorporating methylmercury biomarkers. Although there is ample data suggesting that the vast majority of mercury humans are exposed to is in the form of methylmercury, recent work has highlighted the fact that total mercury may not always be a good proxy measurement for the effects of methylmercury (45). An additional limitation is that given the high amounts of mercury in seafood, if the population under study has any fish or seafood consumption, it is extremely difficult to rule out the possibility that the fish or seafood may confound results for other food items.

Therefore, the goal of this study is to identify foods associated with elevated total mercury in whole blood (THg), methylmercury in whole blood (MeHg) and urinary mercury (UHg) among those who report consuming fish and seafood and those who do not. UHg is a measure of total mercury, but unlike total mercury in blood, total mercury in urine is thought to reflect a much higher proportion of inorganic mercury compounds. We use NHANES data for this analysis because the survey is a large, representative study of the United States population that includes a highly detailed dietary assessment and speciated mercury biomarkers. Additionally, the survey determines typical seafood consumption in the month prior to the survey, which allows for identification of those who commonly eat fish and seafood versus not.

Methods

Study design and population.

This analysis uses 2011–2012 data from the United States Center for Disease Control and Prevention's National Health and Nutrition Examination Survey. NHANES is a cross-sectional study that utilizes a complex multistage probability sampling design of noninstitutionalized civilians in order to obtain a representative sample of the United States population in consecutive two-year cycles, which can be combined to increase analytical sample size. Selected subpopulations are oversampled in order to increase the precision of estimates among these subgroups. Analyses incorporated appropriate survey weights and utilized estimation procedures for survey samples. NHANES operates with approval from the National Center for Health Statistics Ethical Review Board; all participants completed a written informed consent process prior to participation in the study. More details about NHANES design and methods can be found online at www.cdc.gov/nchs/nhanes.

Data from NHANES 2011–2012 were selected for this analysis because both total and methylmercury data are available on the full examination sample. Prior to 2011, methylmercury was not included; after 2012 methylmercury was included on a 1/3 subsample of the eligible population. Different weights are needed for a subsample compared to the full population; however, if different weights are combined the sample would not reflect a representative sample of the United States. Thus, we limited this analysis to 2011–2012 data. There were 9756 persons included in NHANES 2011–2012 (see Supplemental Figure 1). We excluded participants who did not have complete THg or MeHg data (N=1919), did not complete the 24-hour dietary recall (N=608), or did not complete the dietary questionnaire (N=32); this leaves a total of N=7197. Those who were included in analyses were more likely to be 20 years old and non-Hispanic white (data not shown). Participants who either reported eating fish or shellfish within the past 30 days or reported a

food item containing fish or seafood in their 24-hour recall were classified as seafood consumers (5427/7197); otherwise participants were classified as non-seafood consumers (1770/7197). UHg was measured on a randomly selected one-third subset of eligible participants; thus, analyses with UHg are conducted separately and include a smaller population (N=2135). Using the same criteria as above, there were 1614/2135 persons who were seafood consumers and 521/2135 persons who were non-seafood consumers.

Assessment of mercury biomarkers.

THg, MeHg and UHg concentrations are included in this analysis. NHANES did quantify whole blood inorganic mercury; however, >70% of values were below the limit of detection limit (LOD) and therefore blood inorganic mercury is not used in the current analysis. Whole blood and urine samples were collected using standard procedures by trained study staff. Trace-free equipment was used for collection; blood samples were stored at -30°C or lower. Mercury concentrations were determined in separate analytic runs using inductively coupled plasma mass spectrometry (LOD=0.16 $\mu\text{g/L}$ [THg], 0.12 $\mu\text{g/L}$ [MeHg], and 0.05 $\mu\text{g/g}$ creatinine [UHg] (46). There were 573/7197 (8.0%) and 1302/7197 (18.1%) samples < LOD for THg and MeHg, respectively. Within the subsample of those assessed for UHg there were 118/2135 (5.5%) samples < LOD. Values <LOD were replaced with LOD/ 2 for analyses. UHg values were divided by urinary creatinine to adjust for differences in dilution, thus UHg is reported in $\mu\text{g/g}$ creatinine.

Dietary assessment.

The dietary assessment in NHANES is conducted in collaboration with the United States Department of Agriculture (USDA). The in-person dietary interview is completed by a trained interviewer and is conducted on the same day as the blood and urine collection. A proxy respondent, most often a parent or guardian, completed this section for children <6 years old; for children 6–11 years old, the child completes the section with assistance from the proxy respondent. The dietary assessment includes a 24-hour recall component followed by a questionnaire component. During the questionnaire component, participants are asked if they ate fish or shellfish within the past 30 days.

For the 24-hour recall component, participants are asked to report all foods eaten within the past 24 hours. After the initial response, interviewers ask specifically about foods that are frequently forgotten in initial reporting (such as beverages and snacks) and use neutral probing methods to obtain more details when answers are incomplete or unclear. This is done to increase completeness and specificity of overall responses. The United States Department of Agriculture (USDA) What We Eat In America (WWEIA) Survey classifies individual foods into approximately 150 distinct categories based on nutrient and overall consumption patterns within the United States. See www.ars.usda.gov/ba/bhnrc/fsrg for more details on these food categories. We used these categories as a starting point, and then further combined similar foods into a smaller number of food groups (see Supplemental Table 1). Our group classification was based on recommendations from WWEIA, knowledge of which foods have been associated with mercury biomarkers previously (29,35,36,38), and our own initial descriptive analyses.

Other variables.

Data on age, sex, race/ethnicity, and education were obtained via questionnaire. Age was categorized for presentation of descriptive statistics (1 to 19.9 years/20 to 39.9 years/40 to 59.9 years/ 60 years) but was included as a continuous variable in regression models. Race/ethnicity was categorized as non-Hispanic (NH) white, NH black, Hispanic, NH Asian, or other race/multiracial. Education was classified as < high school, high school or equivalent, some college or 2-year degree, 4-year college degree or higher; persons <20 years old, reporting 'don't know', or missing data on educational attainment were grouped into a separate category.

Statistical analyses.

Statistical analyses were completed using Stata 13.1 (College Station, Texas, USA); a p -value <0.05 was considered statistically significant. This is an exploratory analysis as opposed to a confirmatory data analysis; therefore, any results should be verified with other investigations and no adjustments for multiple comparisons are included. Analytical procedures which incorporate the complex survey design and appropriate weights were used. We used Mobile Examination Center weights (MEC) for analyses with THg or MeHg as this represented the smallest sampling unit for these data. As UHg was assessed using a random 1/3 subsample, analyses involving urinary mercury incorporated appropriate subsample weights instead of MEC weights.

All mercury biomarkers were approximately lognormally distributed; therefore, geometric means (95% confidence interval) are presented and natural log transformed variables are used in statistical analyses. The United States Environmental Protection Agency's current reference dose for MeHg is based on a cord blood total mercury measurement of 5.8 µg/L (47). Studies have demonstrated that cord blood Hg is, on average, 1.7 times higher than maternal blood Hg concentrations (48); using this ratio, 5.8 µg/L in cord blood would be equivalent to 3.4 µg/L in maternal blood. Therefore, we created variables to indicate whether whole blood THg or MeHg were >5.8 or >3.4 µg/L.

Descriptive analyses include presentation of demographic characteristics and mercury concentrations among the total population and stratified by seafood consumption. Pearson's chi-square and Wald tests from unadjusted linear regressions were used to evaluate the statistical significance of the variation across demographic characteristics or mercury concentrations by seafood consumption. Geometric mean THg, MeHg, and UHg for persons reporting eating food in each food category were calculated for seafood consumers and non-seafood consumers, separately. Wald tests from unadjusted regression models were used to determine if there was a significant difference in Hg among those reporting eating the specific food vs. not eating that food. Data are not shown when N for a specific food category is <10.

Adjusted linear regression models were constructed to determine the independent associations between specific food categories and each mercury species (THg, MeHg, UHg) within the population groups (the entire population, seafood consumers within past 30 days, non-seafood consumers within past 30 days). All models were adjusted for age, sex,

education, and race/ethnicity. Specific categories for food reported eaten within the past 24 hours were selected because the category had exhibited a significant association with at least one mercury species in unadjusted analyses. Some food categories that were similar were further combined due to collinearity. Food groups used as model covariates were fish, shellfish, or mixed seafood dishes; beans, nuts, or soy (including milk substitutes); Asian foods; soup; mixed rice dishes; rice; red vegetables, leafy vegetables, or vegetable oil; beer; wine; and liquor. It is likely that some seafood might be a minor ingredient in some of these categories such as Asian foods, soups, or mixed rice dishes; this is discussed in more detail below. Regression coefficients (95% confidence intervals) are reported.

Code availability.

Code used in data analysis will be available as a Stata do-file on the EPA Science Hub website, <https://catalog.data.gov/dataset/epa-sciencehub>.

Results

Demographic characteristics among seafood consumers and non-seafood consumers are presented in Table 1. There was a significant difference in age between the two groups with non-seafood consumers tending to be younger. A higher proportion of seafood consumers had higher education (at least some college) versus non-seafood consumers; this was also statistically significant. There was no significant difference in sex or race/ethnicity between seafood consumers and non-seafood consumers.

Geometric mean mercury concentrations, stratified by seafood consumption, are presented in Table 2. Mercury concentrations were significantly higher among seafood consumers versus non-seafood consumers: for THg and MeHg, the geometric mean among seafood consumers was more than twice as large as that among non-seafood consumers. The ratio of MeHg/THg was also significantly higher among seafood consumers (0.808) versus non-seafood consumers (0.631); overall the MeHg/THg ratio was 0.766 (95% confidence interval: 0.727, 0.805). An estimated 3.8% of seafood consumers had THg higher than 5.8 µg/L; 9.4% had concentrations higher than 3.4 µg/L. In terms of the US population, this suggests roughly 7.7 million and 18.7 million persons have THg > 5.8 µg/L or > 3.4 µg/L, respectively. These percentages were similar, but slightly lower, for MeHg. Less than one percent of non-seafood consumers had blood mercury concentrations higher than these thresholds (Supplemental Table 2).

Geometric mean mercury concentrations for seafood consumers who reported eating specific foods within the past 24 hours are presented in Supplemental Table 3. For seafood consumers, substantially higher THg or MeHg concentrations were observed among those whose diet included fish or shellfish in the past 24 hours, Asian foods, rice, and alcoholic beverages. Higher UHg concentrations were observed among those who consumed fish, beans/nuts/soy, cooked grains and cereal, or wine. Corresponding data for non-seafood consumers are presented in Supplemental Table 4. For non-seafood consumers, somewhat higher THg or MeHg concentrations were observed among those who reported eating rice, vegetables, and substantially higher THg or MeHg among those consuming alcoholic

beverages. Higher UHg concentrations were observed among those whose diet included mixed dishes with rice, and vegetable oils.

Tables 3–5 present results from regression models predicting mercury biomarkers among seafood and non-seafood consumers. Supplemental Table 5 presents the difference in all of these regression models' R^2 between the full model R^2 minus the R^2 from a model without a specific dietary component. In adjusted regression models among seafood consumers, THg was associated with the majority of foods included in the model (Table 3). Consumption of fish or seafood, wine, soup, rice, and red vegetables/leafy vegetables/vegetable oil within the past 24 hours was associated with higher MeHg (Table 4). Consumption of fish or seafood as well as beans, nuts, or soy in the past 24 hours was associated with higher UHg (Table 5).

In adjusted regression models among non-seafood consumers, there was a significant association of consumption of mixed rice dishes, red vegetables/leafy vegetables/vegetable oil, and liquor within the past 24 hours with higher concentrations of whole blood total mercury; wine was approaching statistical significance ($p=0.085$) (Table 3). Consumption of wine within the past 24 hours was significantly associated with higher MeHg (Table 4). Consumption of mixed rice dishes within the past 24 hours was associated with higher UHg (Table 5).

Discussion

In this cross-sectional analysis of a representative sample of the United States population, we identified associations between multiple food categories with THg, MeHg, and UHg in seafood consumers and non-seafood consumers, after adjusting for age, sex, education and race/ethnicity. Overall, seafood consumers had significantly higher concentrations of THg, MeHg, and UHg compared to non-seafood consumers. In adjusted models among self-reported non-seafood consumers, THg was associated with consumption of mixed rice dishes, red vegetables/leafy vegetables/vegetable oil, liquor within the past 24 hours; there was a borderline association with wine. Adjusted models among non-seafood consumers also found significant associations of MeHg with consumption of wine in the past 24 hours, and UHg with consumption of mixed rice dishes in the past 24 hours.

The association of mercury biomarkers with demographic variables in our adjusted models are largely consistent with previous reports. Consistent with our results, higher mercury biomarker concentrations have been associated with older age (49,50), higher education (51) and Asian race/ethnicity (51,52). Interestingly, in our analysis the association with Asian race/ethnicity is strongest among seafood-consumers; among non-seafood consumers, those of Hispanic or non-Hispanic black race/ethnicity have significant associations with THg and Hispanics have a significant association with UHg, whereas Asians who report being non-seafood consumers do not. Prior studies have suggested that a high consumption rate of fish and seafood among those of Asian race/ethnicity may be a major driver of higher mercury concentrations among this group overall (53,54); although we saw no difference by race/ethnicity in whether or not individuals reported eating fish or seafood within the past month, it is still likely that there are different patterns in the type and quantity of seafood consumed which could contribute to these differences in observed Hg biomarker concentrations. The

reason for the association of mercury biomarkers with non-seafood consuming non-Hispanic blacks and Hispanics is less clear. It is possible that these groups may be more likely to be exposed to mercury via non-dietary routes such as occupation, residence in environmentally contaminated areas, or use of mercury-containing cosmetics including skin-lightening creams (55).

In adjusted models, recent consumption of rice and/or mixed rice dishes was associated with THg and MeHg among seafood consumers and mixed rice dishes were associated with THg and UHg among non-seafood consumers. Numerous studies have been able to quantify mercury concentrations of concern in rice and rice-containing cereals (29,30,56–58). Several studies have also identified significant associations of rice with mercury biomarker concentrations or modeled mercury exposures in populations with substantial seafood consumption (33,59), although an analysis of the Korean Health and Nutrition Examination Survey did not observe an association between blood mercury and rice consumption (38). Additionally, a series of studies in China focus on regions with high rice but low seafood consumption. These also identified significant associations of rice with methylmercury biomarker concentrations (29,57) or used modeling to estimate a significant contribution to total dietary mercury (30,32). Of note is that a few of these studies are also located in areas near widespread environmental mercury contamination as a result of industrial or mining practices (30,32,57); this likely influences the extent to which rice contributes to total or methylmercury exposure in these populations.

Recent consumption of red vegetables, leafy vegetables, or vegetable oil was associated with higher THg among both seafood and non-seafood consumers and MeHg among seafood consumers, after adjustment for demographic variables. Several studies have suggested that leafy vegetables or other plants are able to uptake mercury as a result of local mercury contamination in soil (60–64), air (65,66), water (67,68); or from mercury-containing biosolids applied to soils (69–72). Epidemiology studies have also identified associations of vegetables with mercury biomarkers among populations living near areas with environmental mercury contamination (37,57), as well as populations in Korea (38) and Finland (35). Modeling studies in China have also suggested that leafy greens or vegetables may comprise a substantial contribution to overall dietary mercury exposure (73,74).

In our adjusted models, those who reported drinking wine or liquor in the past 24 hours had, on average, higher THg concentrations and those who reported drinking wine in the past 24 hours had higher MeHg concentrations; in our study, these observations were observed for both seafood and non-seafood consumers. As results among non-seafood consumers are unlikely to be confounded by the presence of mercury in fish/seafood this highlights the potential importance of wine as a contributor to mercury exposure. Additionally, prior research has identified associations of wine consumption with mercury exposure among Finnish men (35), Viennese coronary artery disease patients (75), pregnant women from the United Kingdom (41), and Austrian women (76). Chung and colleagues used factor analysis to identify typical food consumption patterns among Koreans and found that the ‘alcohol and noodle’ dietary pattern was associated with higher blood mercury (77). While the above studies are similar to ours in identifying an association of wine consumption with mercury, the results from our study do appear to be stronger than in these prior reports. In our

regression models, the strength of the association (i.e., the beta coefficient) between wine consumption was larger than most other food categories, and similar in size to that observed for fish/shellfish and seafood (Tables 3, 4). Meanwhile, additional studies have quantified total mercury within wine (40,78–81). Taken together, more investigation into the potential contribution of wine to mercury exposure, particularly in the US population, may be warranted.

This analysis does have some limitations. First, both the 24-hour recall and dietary questionnaire data rely on participant recall, which can result in some inaccuracy. While this possibility cannot completely be ruled out, we think that any effect would be minimal as NHANES takes extensive steps to ensure dietary data accuracy; as shown through completion of periodic validation and crossover studies (82). Another limitation is that the 24-hour dietary recall may not represent longer-term patterns in food consumption, which may result in some confounding of our results. However, this is somewhat offset by the large sample size available in NHANES, which increases that likelihood that even if some individuals may not have a representative diet in the past 24 hours, that the overall population mean will still be a reasonable representation of the population. We additionally have no reason to anticipate that there would be any association of recent versus typical food consumption patterns with mercury, so any misclassification would likely be nondifferential, resulting in a bias towards the null. At the same time, the prior 24-hours of food consumption is likely to be informative with regards to mercury content in blood and urine, as recent food consumption is likely to be strongly represented in these biological samples.

Another limitation is that, although NHANES collected highly detailed and specific dietary information on a large population, there are still a few specific food items which are difficult to isolate in statistical analyses. For example, several prior studies have suggested that mushroom consumption may be a contributor to dietary total or methylmercury exposure (37,40,70,83), but overall mushroom consumption in the typical US diet is incorporated into other categories (i.e., mixed dishes) so we could not evaluate this separately (see Supplemental Table 1). Additionally, there were a few food categories which we were unable to evaluate in multivariable models because either too few participants reported eating them in the past 24 hours (e.g., wine and liquor among non-seafood consumers with UHg, see Supplemental Table 4) or they were too highly correlated with other food items. For example, in unadjusted analyses consumption of vegetable oil appears to be highly correlated with UHg among non-seafood consumers; however, recent consumption of vegetable oil was highly correlated with consumption of leafy and other vegetables; thus, including them as separate items in regression models would likely have resulted in model collinearity. Our estimates are also somewhat limited by the fact that we evaluated food consumption as a binary variable (consumption vs. not) instead of incorporating data on the quantity of each food consumed. A final consideration is that it is possible that some individuals who report being non-seafood consumers might have some non-zero amount of seafood consumption, either through consuming seafood less frequently than on a monthly basis or via eating food items with “hidden” seafood ingredients. For example, Asian dishes or soups which do not have fish or shellfish as a main ingredient may still contain fish or oyster sauce as a minor ingredient. It is possible that this may have resulted in some

exposure misclassification with Asian foods, mixed rice dishes, or soups. Results for these categories should be interpreted accordingly.

There are also many strengths to this analysis. First, NHANES is a large, representative sample of the United States population. However, the benefit of the representative sampling may be somewhat diminished in this analysis as children and minorities were more likely to be excluded from analysis due to lack of mercury biomarker or dietary data. Another strength is that we were able to use multiple mercury biomarkers (THg in blood, MeHg in blood, and UHg) which represent different proportions of different mercury compounds. THg is dominated by organic mercury, mainly MeHg, whereas UHg has a high proportion of inorganic mercury (3). As noted above, the association patterns of dietary components with these three biomarkers differ, which could suggest potential differences in the source of mercury contamination.

Another substantial strength of this study is the highly detailed dietary information collected as part of this study. While we were not able to explore all potential foods of concern, as noted above, the large sample size and detailed data collected from the 24-hour dietary recall allowed sufficient power to investigate over 30 different food categories. Additionally, NHANES includes extensive questions about long-term dietary patterns, which allowed us to contrast results among seafood consumers and non-seafood consumers. It is possible that with any analysis of seafood consumers, even when controlling for fish/seafood consumption, that associations of non-seafood dietary components with mercury biomarkers may be a result of residual confounding from seafood consumption. This is because fish and seafood are unquestionably the predominant source of dietary mercury exposure and consumption of many types of food are highly correlated. Analyses within a non-seafood consuming population are much less likely to be influenced from residual confounding from seafood consumption, thus, associations observed within this subset are highly informative regarding non-seafood sources of dietary mercury exposure.

Taken together, this work supports and extends existing research that certain non-seafood dietary items such as vegetables, rice, and wine are associated with higher average concentrations of mercury biomarkers. A key strength of this analysis is that we use a large, representative sample of the United States, and demonstrate that the associations of mercury biomarkers with vegetable, rice, and wine intake are observed among both seafood consumers and non-seafood consumers. Although fish and seafood are unquestionably the source of the largest quantity of dietary mercury exposure, contributions from other sources should still be considered, especially among non-seafood- consuming populations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgements:

The views expressed in this article are those of the authors and do not necessarily reflect the views or policies of the US Environmental Protection Agency (EPA). EMW's work was supported under a faculty research participation program between the Oak Ridge Institute for Science and Education (ORISE) and the EPA's National Center for

Environmental Assessment (NCEA/ORD/EPA) as well as the United States National Institute of Occupational Safety and Health under Grant T03OH008615.

References

1. WHO. Ten chemicals of major public health concern [Internet]. 2010 [cited 2019 Jun 4]. Available from: https://www.who.int/ipcs/assessment/public_health/chemicals_phc/en/
2. Bjørklund G, Dadar M, Mutter J, Aaseth J. The toxicology of mercury: Current research and emerging trends. *Environmental Research*. 2017 11;159:545–54. [PubMed: 28889024]
3. Clarkson TW, Magos L. The toxicology of mercury and its chemical compounds. *Crit Rev Toxicol*. 2006 9;36(8):609–62. [PubMed: 16973445]
4. Syversen T, Kaur P. The toxicology of mercury and its compounds. *Journal of Trace Elements in Medicine and Biology*. 2012 10;26(4):215–26. [PubMed: 22658719]
5. Bridges CC, Zalups RK. The aging kidney and the nephrotoxic effects of mercury. *J Toxicol Environ Health B Crit Rev*. 2017;20(2):55–80. [PubMed: 28339347]
6. Fields CA, Borak J, Louis ED. Mercury-induced motor and sensory neurotoxicity: systematic review of workers currently exposed to mercury vapor. *Crit Rev Toxicol*. 2017 11;47(10):811–44. [PubMed: 28718354]
7. Castoldi AF, Coccini T, Ceccatelli S, Manzo L. Neurotoxicity and molecular effects of methylmercury. *Brain Research Bulletin*. 2001 5;55(2):197–203. [PubMed: 11470315]
8. Davidson PW, Myers GJ, Weiss B. Mercury exposure and child development outcomes. *Pediatrics*. 2004 4 1;113(4):1023–9. [PubMed: 15060195]
9. Farina M, Rocha JBT, Aschner M. Mechanisms of methylmercury-induced neurotoxicity: Evidence from experimental studies. *Life Sciences*. 2011 10;89(15–16):555–63. [PubMed: 21683713]
10. Grandjean P, Budtz-Jorgensen E, White RF, Jorgensen PJ, Weihe P, Debes F, et al. Methylmercury exposure biomarkers as indicators of neurotoxicity in children aged 7 years. *Am J Epidemiol*. 1999 8 1;150(3):301–5. [PubMed: 10430235]
11. Rice D, Barone S. Critical Periods of Vulnerability for the Developing Nervous System: Evidence from Humans and Animal Models. *Environmental Health Perspectives*. 2000 6;108:511. [PubMed: 10852851]
12. Mergler D, Anderson HA, Chan LHM, Mahaffey KR, Murray M, Sakamoto M, et al. Methylmercury Exposure and Health Effects in Humans: A Worldwide Concern. *AMBIO: A Journal of the Human Environment*. 2007 2;36(1):3–11.
13. Sheehan MC, Burke TA, Navas-Acien A, Breyse PN, McGready J, Fox MA. Global methylmercury exposure from seafood consumption and risk of developmental neurotoxicity: a systematic review. *Bulletin of the World Health Organization*. 2014 4 1;92(4):254–269F. [PubMed: 24700993]
14. Guallar E, Sanz-Gallardo MI, Veer P van't, Bode P, Aro A, Gómez-Aracena J, et al. Mercury, Fish Oils, and the Risk of Myocardial Infarction. *New England Journal of Medicine*. 2002 11 28;347(22):1747–54. [PubMed: 12456850]
15. Houston MC. Role of Mercury Toxicity in Hypertension, Cardiovascular Disease, and Stroke: Role of Mercury Toxicity in Hypertension. *The Journal of Clinical Hypertension*. 2011 8;13(8):621–7. [PubMed: 21806773]
16. Virtanen JK, Rissanen TH, Voutilainen S, Tuomainen T-P. Mercury as a risk factor for cardiovascular diseases. *The Journal of Nutritional Biochemistry*. 2007 2;18(2):75–85. [PubMed: 16781863]
17. Crowe W, Allsopp PJ, Watson GE, Magee PJ, Strain J, Armstrong DJ, et al. Mercury as an environmental stimulus in the development of autoimmunity – A systematic review. *Autoimmunity Reviews*. 2017 1;16(1):72–80. [PubMed: 27666813]
18. Driscoll CT, Mason RP, Chan HM, Jacob DJ, Pirrone N. Mercury as a Global Pollutant: Sources, Pathways, and Effects. *Environmental Science & Technology*. 2013 5 21;47(10):4967–83. [PubMed: 23590191]

19. Ellingsen DG, Bast-Pettersen R, Efskind J, Thomassen Y. Neuropsychological Effects of Low Mercury Vapor Exposure in Chloralkali Workers. *NeuroToxicology*. 2001 4;22(2):249–58. [PubMed: 11405256]
20. Gibb H, O'Leary KG. Mercury Exposure and Health Impacts among Individuals in the Artisanal and Small-Scale Gold Mining Community: A Comprehensive Review. *Environmental Health Perspectives*. 2014 7;122(7):667–72. [PubMed: 24682486]
21. Li P, Du B, Chan HM, Feng X. Human inorganic mercury exposure, renal effects and possible pathways in Wanshan mercury mining area, China. *Environ Res*. 2015 7;140:198–204. [PubMed: 25863593]
22. Khwaja MA, Abbasi MS. Mercury poisoning dentistry: high-level indoor air mercury contamination at selected dental sites. *Reviews on Environmental Health* [Internet]. 2014 1 1 [cited 2019 Jun 3];29(1–2). Available from: <https://www.degruyter.com/view/j/reveh.2014.29.issue-1-2/reveh-2014-0010/reveh-2014-0010.xml>
23. Homme KG, Kern JK, Haley BE, Geier DA, King PG, Sykes LK, et al. New science challenges old notion that mercury dental amalgam is safe. *Biomaterials*. 2014 2;27(1):19–24. [PubMed: 24420334]
24. Lindberg A, Björnberg KA, Vahter M, Berglund M. Exposure to methylmercury in non-fish-eating people in Sweden. *Environ Res*. 2004 9;96(1):28–33. [PubMed: 15261781]
25. Vieira SM, de Almeida R, Holanda IBB, Mussu MH, Galvão RCF, Crispim PTB, et al. Total and methyl-mercury in hair and milk of mothers living in the city of Porto Velho and in villages along the Rio Madeira, Amazon, Brazil. *Int J Hyg Environ Health*. 2013 11;216(6):682–9. [PubMed: 23340120]
26. Chan TYK. Inorganic mercury poisoning associated with skin-lightening cosmetic products. *Clin Toxicol (Phila)*. 2011 12;49(10):886–91. [PubMed: 22070559]
27. Lee D, Lee K-G. Mercury and methylmercury in Korean herbal medicines and functional health foods. *Food Addit Contam Part B Surveill*. 2013;6(4):279–84. [PubMed: 24779938]
28. Basu N, Horvat M, Evers DC, Zastenskaya I, Weihe P, Tempowski J. A State-of-the-Science Review of Mercury Biomarkers in Human Populations Worldwide between 2000 and 2018. *Environ Health Perspect*. 2018;126(10):106001. [PubMed: 30407086]
29. Hong C, Yu X, Liu J, Cheng Y, Rothenberg SE. Low-level methylmercury exposure through rice ingestion in a cohort of pregnant mothers in rural China. *Environ Res*. 2016;150:519–27. [PubMed: 27423706]
30. Li P, Feng X, Yuan X, Chan HM, Qiu G, Sun G-X, et al. Rice consumption contributes to low level methylmercury exposure in southern China. *Environ Int*. 2012 11 15;49:18–23. [PubMed: 22944358]
31. Rothenberg SE, Yu X, Liu J, Biasini FJ, Hong C, Jiang X, et al. Maternal methylmercury exposure through rice ingestion and offspring neurodevelopment: A prospective cohort study. *Int J Hyg Environ Health*. 2016;219(8):832–42. [PubMed: 27503636]
32. Zhang H, Feng X, Larssen T, Qiu G, Vogt RD. In inland China, rice, rather than fish, is the major pathway for methylmercury exposure. *Environ Health Perspect*. 2010 9;118(9):1183–8. [PubMed: 20378486]
33. Davis MA, Gilbert-Diamond D, Karagas MR, Li Z, Moore JH, Williams SM, et al. A Dietary-Wide Association Study (DWAS) of Environmental Metal Exposure in US Children and Adults. Crawford DC, editor *PLoS ONE*. 2014 9 8;9(9):e104768. [PubMed: 25198543]
34. Rothenberg SE, Jackson BP, Carly McCalla G, Donohue A, Emmons AM. Co-exposure to methylmercury and inorganic arsenic in baby rice cereals and rice-containing teething biscuits. *Environmental Research*. 2017 11 1;159:639–47. [PubMed: 28938205]
35. Airaksinen R, Turunen AW, Rantakokko P, Männistö S, Vartiainen T, Verkasalo PK. Blood concentration of methylmercury in relation to food consumption. *Public Health Nutr*. 2011 3;14(3):480–9. [PubMed: 20529404]
36. Kwon YM, Lee HS, Yoo DC, Kim CH, Kim GS, Kim JA, et al. Dietary exposure and risk assessment of mercury from the Korean total diet study. *J Toxicol Environ Health Part A*. 2009;72(21–22):1484–92. [PubMed: 20077222]
37. Miklav i A, Mazej D, Ja imovi R, Dizdarevi T, Horvat M. Mercury in food items from the Idrija Mercury Mine area. *Environ Res*. 2013 8;125:61–8. [PubMed: 23683522]

38. Park S, Lee B-K. Strong positive associations between seafood, vegetables, and alcohol with blood mercury and urinary arsenic levels in the Korean adult population. *Arch Environ Contam Toxicol*. 2013 1;64(1):160–70. [PubMed: 23011092]
39. Shao D, Kang Y, Cheng Z, Wang H, Huang M, Wu S, et al. Hair mercury levels and food consumption in residents from the Pearl River Delta: South China. *Food Chem*. 2013 1 15;136(2):682–8. [PubMed: 23122114]
40. Filippini T, Malavolti M, Cilloni S, Wise LA, Violi F, Malagoli C, et al. Intake of arsenic and mercury from fish and seafood in a Northern Italy community. *Food Chem Toxicol*. 2018 6;116(Pt B):20–6.
41. Golding J, Steer CD, Hibbeln JR, Emmett PM, Lowery T, Jones R. Dietary predictors of maternal prenatal blood mercury levels in the ALSPAC birth cohort study. *Environ Health Perspect*. 2013 10;121(10):1214–8. [PubMed: 23811414]
42. Dufault R, LeBlanc B, Schnoll R, Cornett C, Schweitzer L, Wallinga D, et al. Mercury from chlor-alkali plants: measured concentrations in food product sugar. *Environ Health*. 2009 1 26;8:2. [PubMed: 19171026]
43. Gagné D, Lauzière J, Blanchet R, Vézina C, Vaissière E, Ayotte P, et al. Consumption of tomato products is associated with lower blood mercury levels in Inuit preschool children. *Food Chem Toxicol*. 2013 1;51:404–10. [PubMed: 23127601]
44. Awata H, Linder S, Mitchell LE, Delclos GL. Association of Dietary Intake and Biomarker Levels of Arsenic, Cadmium, Lead, and Mercury among Asian Populations in the United States: NHANES 2011–2012. *Environ Health Perspect*. 2017;125(3):314–23. [PubMed: 27586241]
45. Wells EM, Herbstman JB, Lin YH, Hibbeln JR, Halden RU, Witter FR, et al. Methyl mercury, but not inorganic mercury, associated with higher blood pressure during pregnancy. *Environ Res*. 2017 4;154:247–52. [PubMed: 28110211]
46. CDC. NHANES 2011–2012 Laboratory Methods [Internet]. 2013 [cited 2019 Jun 5]. Available from: <https://wwwn.cdc.gov/nchs/nhanes/ContinuousNhanes/LabMethods.aspx?BeginYear=2011>
47. US EPA. Chemical Assessment Summary: Methylmercury (MeHg); CASRN 22967-92-6 [Internet]. Washington, D.C.: US Environmental Protection Agency, Integrated Risk Assessment System (IRIS); 2001 [cited 2019 Jun 26]. Available from: https://cfpub.epa.gov/ncea/iris/iris_documents/documents/subst/0073_summary.pdf
48. Stern AH, Smith AE. An assessment of the cord blood:maternal blood methylmercury ratio: implications for risk assessment. *Environ Health Perspect*. 2003 9;111(12):1465–70. [PubMed: 12948885]
49. Birch RJ, Bigler J, Rogers JW, Zhuang Y, Clickner RP. Trends in blood mercury concentrations and fish consumption among U.S. women of reproductive age, NHANES, 1999–2010. *Environ Res*. 2014 8;133:431–8. [PubMed: 24602558]
50. Mahaffey KR, Clickner RP, Bodurow CC. Blood organic mercury and dietary mercury intake: National Health and Nutrition Examination Survey, 1999 and 2000. *Environ Health Perspect*. 2004 4;112(5):562–70. [PubMed: 15064162]
51. Mortensen ME, Caudill SP, Caldwell KL, Ward CD, Jones RL. Total and methyl mercury in whole blood measured for the first time in the U.S. population: NHANES 2011–2012. *Environ Res*. 2014 10;134:257–64. [PubMed: 25173092]
52. Liu Y, Buchanan S, Anderson HA, Xiao Z, Persky V, Turyk ME. Association of methylmercury intake from seafood consumption and blood mercury level among the Asian and Non-Asian populations in the United States. *Environ Res*. 2018;160:212–22. [PubMed: 29020643]
53. Buchanan S, Targos L, Nagy KL, Kearney KE, Turyk M. Fish Consumption and Hair Mercury Among Asians in Chicago. *J Occup Environ Med*. 2015 12;57(12):1325–30. [PubMed: 26641830]
54. Lin S, Herdt-Losavio ML, Chen M, Luo M, Tang J, Hwang S-A. Fish consumption patterns, knowledge and potential exposure to mercury by race. *Int J Environ Health Res*. 2014 8;24(4):291–303. [PubMed: 23865562]
55. McKelvey W, Jeffery N, Clark N, Kass D, Parsons PJ. Population-Based Inorganic Mercury Biomonitoring and the Identification of Skin Care Products as a Source of Exposure in New York City. *Environ Health Perspect*. 2011 2;119(2):203–9. [PubMed: 20923743]

56. Cui W, Liu G, Bezerra M, Lagos DA, Li Y, Cai Y. Occurrence of Methylmercury in Rice-Based Infant Cereals and Estimation of Daily Dietary Intake of Methylmercury for Infants. *J Agric Food Chem*. 2017 11 8;65(44):9569–78. [PubMed: 29067797]
57. Feng X, Li P, Qiu G, Wang S, Li G, Shang L, et al. Human exposure to methylmercury through rice intake in mercury mining areas, Guizhou province, China. *Environ Sci Technol*. 2008 1 1;42(1):326–32. [PubMed: 18350916]
58. Rothenberg SE, Yin R, Hurley JP, Krabbenhoft DP, Ismawati Y, Hong C, et al. Stable Mercury Isotopes in Polished Rice (*Oryza sativa* L.) and Hair from Rice Consumers. *Environ Sci Technol*. 2017 6 6;51(11):6480–8. [PubMed: 28482656]
59. Tong Y-D, Ou L-B, Chen L, Wang H-H, Chen C, Wang X-J, et al. Modeled methylmercury exposure and risk from rice consumption for vulnerable populations in a traditional fish-eating area in China. *Environ Toxicol Chem*. 2015 5;34(5):1161–8. [PubMed: 25639888]
60. Antoniadis V, Shaheen SM, Boersch J, Frohne T, Du Laing G, Rinklebe J. Bioavailability and risk assessment of potentially toxic elements in garden edible vegetables and soils around a highly contaminated former mining area in Germany. *J Environ Manage*. 2017 1 15;186(Pt 2):192–200. [PubMed: 27117508]
61. Bempah CK, Ewusi A. Heavy metals contamination and human health risk assessment around Obuasi gold mine in Ghana. *Environ Monit Assess*. 2016 5;188(5):261. [PubMed: 27037696]
62. Kootbodien T, Mathee A, Naicker N, Moodley N. Heavy metal contamination in a school vegetable garden in Johannesburg. *S Afr Med J*. 2012 3 7;102(4):226–7. [PubMed: 22464503]
63. Riaz A, Khan S, Muhammad S, Liu C, Shah MT, Tariq M. Mercury contamination in selected foodstuffs and potential health risk assessment along the artisanal gold mining, Gilgit-Baltistan, Pakistan. *Environ Geochem Health*. 2018 4;40(2):625–35. [PubMed: 28695305]
64. Yu H, Li J, Luan Y. Meta-analysis of soil mercury accumulation by vegetables. *Sci Rep*. 2018 19;8(1):1261. [PubMed: 29352200]
65. De Temmerman L, Waegeneers N, Claeys N, Roekens E. Comparison of concentrations of mercury in ambient air to its accumulation by leafy vegetables: an important step in terrestrial food chain analysis. *Environ Pollut*. 2009 4;157(4):1337–41. [PubMed: 19118931]
66. Jiskra M, Sonke JE, Obrist D, Bieser J, Ebinghaus R, Myhre CL, et al. A vegetation control on seasonal variations in global atmospheric mercury concentrations. *Nature Geosci*. 2018 4;11(4):244–50.
67. Göthberg A, Greger M, Bengtsson B-E. Accumulation of heavy metals in water spinach (*Ipomoea aquatica*) cultivated in the Bangkok region, Thailand. *Environmental Toxicology and Chemistry*. 2002;21(9):1934–9. [PubMed: 12206434]
68. Islam GMR, Khan FE, Hoque MM, Jolly YN. Consumption of unsafe food in the adjacent area of Hazaribag tannery campus and Buriganga River embankments of Bangladesh: heavy metal contamination. *Environ Monit Assess*. 2014 11;186(11):7233–44. [PubMed: 25030244]
69. Bache CA, Gutenmann WH, St. John LE, Sweet RD, Hatfield HH, Lisk DJ. Mercury and methylmercury content of agricultural crops grown on soils treated with various mercury compounds. *Journal of Agricultural and Food Chemistry*. 1973 7;21(4):607–13. [PubMed: 4718930]
70. Benbrahim M, Denaix L, Thomas A-L, Balet J, Carnus J-M. Metal concentrations in edible mushrooms following municipal sludge application on forest land. *Environ Pollut*. 2006 12;144(3):847–54. [PubMed: 16616804]
71. Cappon CJ. Uptake and speciation of mercury and selenium in vegetable crops grown on compost-treated soil. *Water Air Soil Pollut*. 1987 8 1;34(4):353–61.
72. Sloan JJ, Dowdy RH, Balogh SJ, Nater E. Distribution of mercury in soil and its concentration in runoff from a biosolids-amended agricultural watershed. *J Environ Qual*. 2001 12;30(6):2173–9. [PubMed: 11790029]
73. Li Z, Wang Q, Luo Y. Exposure of the urban population to mercury in Changchun city, Northeast China. *Environ Geochem Health*. 2006 4;28(1–2):61–6. [PubMed: 16528593]
74. Wai K-M, Dai J, Yu PKN, Zhou X, Wong CMS. Public health risk of mercury in China through consumption of vegetables, a modelling study. *Environ Res*. 2017;159:152–7. [PubMed: 28800473]

75. Sponder M, Fritzer-Szekeres M, Marculescu R, Mittlböck M, Uhl M, Köhler-Vallant B, et al. Blood and urine levels of heavy metal pollutants in female and male patients with coronary artery disease. *Vasc Health Risk Manag*. 2014;10:311–7. [PubMed: 24868163]
76. Gundacker C, Komarnicki G, Zödl B, Forster C, Schuster E, Wittmann K. Whole blood mercury and selenium concentrations in a selected Austrian population: does gender matter? *Sci Total Environ*. 2006 12 15;372(1):76–86. [PubMed: 16963109]
77. Chung H-K, Park JY, Cho Y, Shin M-J. Contribution of dietary patterns to blood heavy metal concentrations in Korean adults: findings from the Fifth Korea National Health and Nutrition Examination Survey 2010. *Food Chem Toxicol*. 2013 12;62:645–52. [PubMed: 24120902]
78. Dressler VL, Santos CMM, Antes FG, Bentlin FRS, Pozebon D, Flores EMM. Total Mercury, Inorganic Mercury and Methyl Mercury Determination in Red Wine. *Food Anal Methods*. 2012 6 1;5(3):505–11.
79. Frías S, Díaz C, Conde JE, Pérez Trujillo JP. Selenium and mercury concentrations in sweet and dry bottled wines from the Canary Islands, Spain. *Food Addit Contam*. 2003 3;20(3):237–40. [PubMed: 12623647]
80. Santos S, Lapa N, Alves A, Morais J, Mendes B. Analytical methods and validation for determining trace elements in red wines. *Journal of Environmental Science and Health, Part B*. 2013 5 1;48(5):364–75.
81. Semla M, Schwarcz P, Mezey J, Binkowski ŁJ, Błaszczyk M, Formicki G, et al. Biogenic and Risk Elements in Wines from the Slovak Market with the Estimation of Consumer Exposure. *Biol Trace Elem Res*. 2018 7;184(1):33–41. [PubMed: 28988282]
82. Ahluwalia N, Dwyer J, Terry A, Moshfegh A, Johnson C. Update on NHANES Dietary Data: Focus on Collection, Release, Analytical Considerations, and Uses to Inform Public Policy. *Adv Nutr*. 2016 Jan;7(1):121–34.
83. Árvay J, Tomáš J, Hauptvogel M, Massányi P, Harangozó , Tóth T, et al. Human exposure to heavy metals and possible public health risks via consumption of wild edible mushrooms from Slovak Paradise National Park, Slovakia. *J Environ Sci Health B*. 2015;50(11):833–43. [PubMed: 26357894]

Table 1:

Demographic characteristics stratified by seafood consumption

Variable	Seafood consumers ^a	Non-seafood consumers ^b	p-value ^c
Age			
1–19 years	18.2 (16.3, 20.4)	37.2 (35.2, 39.3)	<0.001
20–39 years	27.3 (23.5, 31.5)	29.7 (24.6, 35.2)	
40–59 years	31.9 (29.0, 35.0)	22.1 (18.5, 26.3)	
60 years	22.6 (20.0, 25.4)	11.0 (8.4, 14.3)	
Sex			
Male	51.3 (49.8, 52.7)	49.5 (46.9, 52.2)	0.221
Female	48.7 (47.3, 50.2)	50.5 (47.8, 53.1)	
Race/ethnicity			
NH White	64.4 (56.2, 71.8)	66.4 (55.3, 75.9)	0.102
NH Black	12.6 (8.4, 18.6)	9.5 (5.5, 16.1)	
Hispanic	15.5 (10.7, 21.9)	17.5 (11.5, 25.6)	
NH Asian	4.7 (3.4, 6.4)	3.2 (2.1, 4.8)	
Multiracial/other	2.9 (2.0, 4.1)	3.4 (2.2, 5.4)	
Education			
<20 years old/missing	18.2 (16.3, 20.4)	37.2 (35.2, 39.3)	<0.001
< High school	11.8 (9.3, 14.9)	13.6 (10.4, 17.6)	
High school	16.0 (13.4, 19.0)	14.9 (12.6, 17.6)	
Some college	26.5 (23.7, 29.5)	19.6 (17.0, 22.4)	
College degree	27.5 (23.0, 32.4)	14.6 (11.2, 19.0)	

Values are population-weighted percent and 95% confidence intervals; seafood consumption is self-reported within the past 30 days or 24 hours.
 NH = Non-Hispanic.

^a Sample N=5427;

^b Sample N=1770;

^c p<0.05 for differences by seafood consumption using Pearson's Chi-square test.

Table 2.

Mercury concentration, stratified by seafood consumption

Variable	Seafood consumers	Non-seafood consumers	<i>p</i> -value ^a
Whole blood total mercury			
N ^b	5427	1770	
GM (95% CI), µg/L ^c	0.89 (0.78, 1.02)	0.31 (0.28, 0.34)	<0.001
Percent (95% CI) >5.8 µg/L ^c	3.84 (2.33, 6.28)	0.11 (0.01, 0.88)	<0.001
Percent (95% CI) >3.4 µg/L ^c	9.40 (6.40, 13.62)	0.61 (0.23, 1.61)	<0.001
Whole blood methylmercury			
N ^b	5427	1770	
GM (95% CI), µg/L ^c	0.67 (0.57, 0.80)	0.17 (0.16, 0.19)	<0.001
Percent (95% CI) >5.8 µg/L ^c	3.73 (2.22, 6.19)	0.11 (0.01, 0.88)	<0.001
Percent (95% CI) >3.4 µg/L ^c	9.35 (6.43, 13.41)	0.71 (0.30, 1.70)	<0.001
Percent methylmercury/total mercury ^d	80.8 (77.0, 84.6)	63.1 (58.7, 67.5)	<0.001
Urinary total mercury			
N ^b	1612	521	
GM (95% CI), µg/g creatinine ^c	4.07 (3.66, 4.52)	2.59 (2.17, 3.08)	<0.001

GM = geometric mean; 95% CI = 95% confidence interval.

^a *p*-value based on Wald test from unadjusted regression model.^b Unweighted sample N.^c Population-weighted estimate.^d Whole blood methylmercury and total mercury; sample N=5427 in seafood consumers and N=1770 in nonseafood consumers.

Table 3.

β (95% confidence interval) for adjusted linear models predicting whole blood total mercury (THg)^a

Variable	Seafood consumers (N=5427) ^b	Non-seafood consumers (N=1770) ^c
Age, per 10 years	0.08 (0.05, 0.10) ^d	0.05 (0.01, 0.09) ^d
Male (vs. female)	0.09 (0.02, 0.17) ^d	0.07 (−0.02, 0.16)
Education		
Child or missing data	−0.17 (−0.31, −0.04) ^d	−0.23 (−0.46, −0.01) ^d
Less than high school	referent	referent
High school	0.14 (0.005, 0.27) ^d	−0.06 (−0.33, 0.21)
Some college	0.20 (0.09, 0.31) ^d	0.11 (−0.12, 0.35)
4-year college degree	0.53 (0.39, 0.67) ^d	0.02 (−0.21, 0.24)
Race/ethnicity		
Non-Hispanic white	referent	referent
Non-Hispanic black	0.09 (−0.12, 0.30)	0.25 (0.08, 0.43) ^d
Hispanic	0.09 (−0.04, 0.21)	0.29 (0.003, 0.58) ^d
Non-Hispanic Asian	0.81 (0.64, 0.97) ^d	0.19 (−0.10, 0.48)
Other or multiracial	−0.08 (−0.32, 0.17)	0.11 (−0.15, 0.36)
Fish, shellfish or mixed seafood (vs. not)	0.47 (0.32, 0.61) ^d	--
Beans, nuts or soy (vs. not)	0.12 (0.03, 0.21) ^d	0.01 (−0.08, 0.11)
Asian foods (vs. not)	0.17 (0.06, 0.27) ^d	0.09 (−0.17, 0.35)
Soup (vs. not)	0.16 (0.07, 0.25) ^d	0.02 (−0.19, 0.23)
Mixed rice dishes (vs. not)	0.14 (0.03, 0.26) ^d	0.17 (0.01, 0.32) ^d
Rice (vs. not)	0.15 (0.06, 0.24) ^d	0.15 (−0.12, 0.42)
Red or leafy vegetables or oil (vs. not)	0.18 (0.09, 0.26) ^d	0.15 (0.06, 0.23) ^d
Beer (vs. not)	0.01 (−0.12, 0.14)	0.12 (−0.08, 0.33)
Wine (vs. not)	0.47 (0.35, 0.60) ^d	0.47 (−0.07, 1.01) ^e
Liquor (vs. not)	0.18 (0.03, 0.34) ^d	0.32 (0.003, 0.63) ^d

^a. The natural logarithm of whole blood total mercury (μg/L) is the dependent variable.

^b. Model covariates include age, sex, education, race/ethnicity, fish/shellfish/mixed seafood, beans/nuts/soy, Asian foods, soup, mixed rice dishes, rice, red vegetables/leafy vegetables/vegetable oil, beer, wine, and liquor.

^c. Model covariates include age, sex, education, race/ethnicity, beans/nuts/soy, Asian foods, soup, mixed rice dishes, rice, red vegetables/leafy vegetables/vegetable oil, beer, wine, and liquor.

^d. Wald test $p < 0.05$.

^eWald test $p < 0.10$.

Author Manuscript

Author Manuscript

Author Manuscript

Author Manuscript

Table 4.

β (95% confidence interval) for adjusted linear models predicting whole blood methylmercury (MeHg)^a

Variable	Seafood consumers (N=5427) ^b	Non-seafood consumers (N=1770) ^c
Age, per 10 years	0.10 (0.05, 0.15) ^d	0.02 (0.002, 0.04) ^d
Male (vs. female)	0.37 (0.03, 0.70) ^a	0.04 (−0.04, 0.12)
Education		
Child or missing data	−0.02 (−0.29, 0.24)	−0.08 (−0.13, −0.02) ^d
Less than high school	referent	referent
High school	0.18 (−0.08, 0.45)	−0.08 (−0.23, 0.07)
Some college	0.22 (0.01, 0.44) ^d	−0.03 (−0.12, 0.05)
4-year college degree	0.94 (0.41, 1.48) ^d	−0.09 (−0.20, 0.03)
Race/ethnicity		
Non-Hispanic white	referent	referent
Non-Hispanic black	0.03 (−0.41, 0.46)	0.11 (0.03, 0.19) ^d
Hispanic	0.02 (−0.39, 0.42)	0.10 (−0.03, 0.23)
Non-Hispanic Asian	1.71 (0.99, 2.43) ^d	0.21 (0.03, 0.39) ^d
Other or multiracial	−0.20 (−0.81, 0.41)	−0.06 (−0.12, 0.01) ^e
Fish, shellfish or mixed seafood (vs. not)	1.23 (0.50, 1.96) ^d	--
Beans, nuts or soy (vs. not)	0.07 (−0.17, 0.32)	−0.01 (−0.08, 0.06)
Asian foods (vs. not)	0.23 (−0.22, 0.68)	0.06 (−0.06, 0.18)
Soup (vs. not)	0.42 (0.10, 0.73) ^d	0.08 (−0.08, 0.24)
Mixed rice dishes (vs. not)	0.63 (−0.57, 1.84)	0.01 (−0.07, 0.08)
Rice (vs. not)	0.35 (0.05, 0.66) ^d	0.11 (−0.03, 0.26)
Red or leafy vegetables or oil (vs. not)	0.43 (0.08, 0.78) ^d	0.04 (−0.04, 0.12)
Beer (vs. not)	−0.12 (−0.54, 0.30)	0.04 (−0.18, 0.27)
Wine (vs. not)	1.00 (0.57, 1.43) ^d	0.84 (0.06, 1.62) ^d
Liquor (vs. not)	0.20 (−0.32, 0.71)	0.36 (−0.10, 0.82)

^aThe natural logarithm of whole blood methylmercury (μg/L) is the dependent variable.

^bModel covariates include age, sex, education, race/ethnicity, fish/shellfish/mixed seafood, beans/nuts/soy, Asian foods, soup, mixed rice dishes, rice, red vegetables/leafy vegetables/vegetable oil, beer, wine, and liquor.

^cModel covariates include age, sex, education, race/ethnicity, beans/nuts/soy, Asian foods, soup, mixed rice dishes, rice, red vegetables/leafy vegetables/vegetable oil, beer, wine, and liquor.

^dWald test $p < 0.05$.

^eWald test $p < 0.10$.

Table 5.

β (95% confidence interval) for adjusted linear models predicting urinary total mercury (UHg)^a

Variable	Seafood consumers (N=1612) ^b	Non-seafood consumers (N=521) ^c
Age, per 10 years	0.11 (0.06, 0.16) ^d	0.04 (−0.05, 0.12)
Male (vs. female)	−0.23 (−0.40, −0.06) ^d	−0.24 (−0.51, 0.03) ^e
Education		
Child or missing data	0.31 (0.13, 0.50) ^d	0.35 (−0.08, 0.79)
Less than high school	referent	referent
High school	0.21 (−0.08, 0.51)	0.31 (−0.08, 0.70)
Some college	0.21 (0.04, 0.37) ^d	0.38 (−0.09, 0.85)
4-year college degree	0.34 (0.16, 0.53) ^d	0.56 (0.13, 0.99) ^d
Race/ethnicity		
Non-Hispanic white	referent	referent
Non-Hispanic black	−0.25 (−0.42, −0.08) ^d	−0.09 (−0.39, 0.21)
Hispanic	0.12 (−0.05, 0.30)	0.21 (0.04, 0.39) ^d
Non-Hispanic Asian	0.36 (0.14, 0.59) ^d	−0.02 (−0.48, 0.43)
Other or multiracial	0.32 (−0.10, 0.75)	0.06 (−0.28, 0.40)
Fish, shellfish or mixed seafood (vs. not)	0.24 (0.06, 0.43) ^d	--
Beans, nuts or soy (vs. not)	0.19 (0.07, 0.32) ^d	−0.02 (−0.38, 0.35)
Asian foods (vs. not)	0.13 (−0.10, 0.35)	−0.16 (−0.66, 0.34)
Soup (vs. not)	0.06 (−0.13, 0.26)	0.02 (−0.31, 0.34)
Mixed rice dishes (vs. not)	0.08 (−0.18, 0.34)	0.65 (0.02, 1.27) ^d
Rice (vs. not)	−0.02 (−0.19, 0.15)	0.20 (−0.15, 0.55)
Red or leafy vegetables or oil (vs. not)	0.08 (−0.08, 0.25)	−0.07 (−0.27, 0.12)
Beer (vs. not)	−0.07 (−0.23, 0.09)	−0.01 (−0.35, 0.34)
Wine (vs. not)	0.13 (−0.01, 0.41)	−0.54 (−1.55, 0.47)
Liquor (vs. not)	0.08 (−0.19, 0.34)	0.50 (−0.37, 1.36)

^aThe natural logarithm of urinary total mercury ($\mu\text{g/g}$ creatinine) is the dependent variable.

^bModel covariates include age, sex, education, race/ethnicity, fish/shellfish/mixed seafood, beans/nuts/soy, Asian foods, soup, mixed rice dishes, rice, red vegetables/leafy vegetables/vegetable oil, beer, wine, and liquor.

^cModel covariates include age, sex, education, race/ethnicity, beans/nuts/soy, Asian foods, soup, mixed rice dishes, rice, red vegetables/leafy vegetables/vegetable oil, beer, wine, and liquor.

^dWald test $p < 0.05$.

^eWald test $p < 0.10$.