



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

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## 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 h 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  $0.89 \mu\text{g/L}$  (95% confidence interval (CI): 0.78, 1.02) (seafood consumers) and  $0.31 \mu\text{g/L}$  (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

## Introduction

The World Health Organization 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

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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. In addition, 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 2-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](http://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 with 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 Supplementary Fig. 1). We excluded participants who did not have complete THg or MeHg data ( $N = 1919$ ), did not complete the 24 h 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  $\geq 20$  years old and non-Hispanic (NH) 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 h 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  $-30^{\circ}\text{C}$  or lower. Mercury concentrations were determined in separate analytic runs using inductively coupled plasma mass spectrometry (LOD =  $0.16\text{ }\mu\text{g/L}$  [THg],  $0.12\text{ }\mu\text{g/L}$  [MeHg], and  $0.05\text{ }\mu\text{g/g}$  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  $\text{LOD}/\sqrt{2}$  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 h 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 h recall component, participants are asked to report all foods eaten within the past 24 h. 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 USDA What We Eat In America (WWEIA) Survey classifies individual foods into ~150 distinct categories based

on nutrient and overall consumption patterns within the United States. See [www.ars.usda.gov/ba/bhnrc/fsrg](http://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 Supplementary 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 (1–19.9 years/20–39.9 years/40–59.9 years/ $\geq 60$  years) but was included as a continuous variable in regression models. Race/ethnicity was categorized as 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  $5.8\text{ }\mu\text{g/L}$  [47]. Studies have demonstrated that cord blood Hg is, on average, 1.7 times higher than maternal blood Hg concentrations [48]; using this ratio,  $5.8\text{ }\mu\text{g/L}$  in cord blood would be equivalent to  $3.4\text{ }\mu\text{g/L}$  in maternal blood. Therefore, we created variables to indicate whether whole blood THg or MeHg were  $>5.8$  or  $>3.4\text{ }\mu\text{g/L}$ .

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, and UHg) within the population groups (the entire population, seafood consumers within past 30 days, and 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 h 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.

## 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) vs. 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.

**Table 1** Demographic characteristics stratified by seafood consumption.

Variable	Seafood consumers <sup>a</sup>	Non-seafood consumers <sup>b</sup>	<i>p</i> value <sup>c</sup>
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 h.

NH non-Hispanic.

<sup>a</sup>Sample  $N = 5427$ .

<sup>b</sup>Sample  $N = 1770$ .

<sup>c</sup> $p < 0.05$  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 <sup>a</sup>
Whole blood total mercury			
<i>N</i> <sup>b</sup>	5427	1770	
GM (95% CI), µg/L <sup>c</sup>	0.89 (0.78, 1.02)	0.31 (0.28, 0.34)	<0.001
Percent (95% CI) > 5.8 µg/L <sup>c</sup>	3.84 (2.33, 6.28)	0.11 (0.01, 0.88)	<0.001
Percent (95% CI) > 3.4 µg/L <sup>c</sup>	9.40 (6.40, 13.62)	0.61 (0.23, 1.61)	<0.001
Whole blood methylmercury			
<i>N</i> <sup>b</sup>	5427	1770	
GM (95% CI), µg/L <sup>c</sup>	0.67 (0.57, 0.80)	0.17 (0.16, 0.19)	<0.001
Percent (95% CI) > 5.8 µg/L <sup>c</sup>	3.73 (2.22, 6.19)	0.11 (0.01, 0.88)	<0.001
Percent (95% CI) > 3.4 µg/L <sup>c</sup>	9.35 (6.43, 13.41)	0.71 (0.30, 1.70)	<0.001
Percent methylmercury/total mercury <sup>d</sup>	80.8 (77.0, 84.6)	63.1 (58.7, 67.5)	<0.001
Urinary total mercury			
<i>N</i> <sup>b</sup>	1612	521	
GM (95% CI), µg/g creatinine <sup>c</sup>	4.07 (3.66, 4.52)	2.59 (2.17, 3.08)	<0.001

GM geometric mean, 95% CI 95% confidence interval.

<sup>a</sup>*p* value based on Wald test from unadjusted regression model.

<sup>b</sup>Unweighted sample *N*.

<sup>c</sup>Population-weighted estimate.

<sup>d</sup>Whole blood methylmercury and total mercury; sample *N* = 5427 in seafood consumers and *N* = 1770 in non-seafood consumers.

Geometric mean mercury concentrations, stratified by seafood consumption, are presented in Table 2. Mercury concentrations were significantly higher among seafood consumers vs. 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) vs. 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 (Supplementary Table 2).

Geometric mean mercury concentrations for seafood consumers who reported eating specific foods within the past 24 h are presented in Supplementary 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 h, 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 Supplementary 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. Supplementary Table 5 presents the difference in all of these regression models' *R*<sup>2</sup> between the full model *R*<sup>2</sup> minus the *R*<sup>2</sup> 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 h was associated with higher MeHg (Table 4). Consumption of fish or seafood as well as beans, nuts, or soy in the past 24 h 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 h 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 h was significantly associated with higher MeHg (Table 4). Consumption of mixed rice dishes within the past 24 h was associated with higher UHg (Table 5).



**Table 3**  $\beta$  (95% confidence interval) for adjusted linear models predicting whole blood total mercury (THg).<sup>a</sup>

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

<sup>a</sup>The natural logarithm of whole blood total mercury ( $\mu\text{g/L}$ ) is the dependent variable.

<sup>b</sup>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.

<sup>c</sup>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.

<sup>d</sup>Wald test  $p < 0.05$ .

<sup>e</sup>Wald test  $p < 0.10$ .

**Table 4**  $\beta$  (95% confidence interval) for adjusted linear models predicting whole blood methylmercury (MeHg).<sup>a</sup>

Variable	Seafood consumers (N = 5427) <sup>b</sup>	Non-seafood consumers (N = 1770) <sup>c</sup>
Age, per 10 years	0.10 (0.05, 0.15) <sup>d</sup>	0.02 (0.002, 0.04) <sup>d</sup>
Male (vs. female)	0.37 (0.03, 0.70) <sup>a</sup>	0.04 (−0.04, 0.12)
Education		
Child or missing data	−0.02 (−0.29, 0.24)	−0.08 (−0.13, −0.02) <sup>d</sup>
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) <sup>d</sup>	−0.03 (−0.12, 0.05)
4-year college degree	0.94 (0.41, 1.48) <sup>d</sup>	−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) <sup>d</sup>
Hispanic	0.02 (−0.39, 0.42)	0.10 (−0.03, 0.23)
Non-Hispanic Asian	1.71 (0.99, 2.43) <sup>d</sup>	0.21 (0.03, 0.39) <sup>d</sup>
Other or multiracial	−0.20 (−0.81, 0.41)	−0.06 (−0.12, 0.01) <sup>e</sup>
Fish, shellfish, or mixed seafood (vs. not)	1.23 (0.50, 1.96) <sup>d</sup>	—
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) <sup>d</sup>	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) <sup>d</sup>	0.11 (−0.03, 0.26)
Red or leafy vegetables or oil (vs. not)	0.43 (0.08, 0.78) <sup>d</sup>	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) <sup>d</sup>	0.84 (0.06, 1.62) <sup>d</sup>
Liquor (vs. not)	0.20 (−0.32, 0.71)	0.36 (−0.10, 0.82)

<sup>a</sup>The natural logarithm of whole blood methylmercury ( $\mu\text{g/L}$ ) is the dependent variable.

<sup>b</sup>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.

<sup>c</sup>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.

<sup>d</sup>Wald test  $p < 0.05$ .

<sup>e</sup>Wald test  $p < 0.10$ .

## 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 with 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 h; 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 h, and UHg with consumption of mixed rice dishes in the past 24 h.

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

**Table 5**  $\beta$  (95% confidence interval) for adjusted linear models predicting urinary total mercury (UHg).<sup>a</sup>

Variable	Seafood consumers (N = 1612) <sup>b</sup>	Non-seafood consumers (N = 521) <sup>c</sup>
Age, per 10 years	0.11 (0.06, 0.16) <sup>d</sup>	0.04 (−0.05, 0.12)
Male (vs. female)	−0.23 (−0.40, −0.06) <sup>d</sup>	−0.24 (−0.51, 0.03) <sup>e</sup>
Education		
Child or missing data	0.31 (0.13, 0.50) <sup>d</sup>	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) <sup>d</sup>	0.38 (−0.09, 0.85)
4-year college degree	0.34 (0.16, 0.53) <sup>d</sup>	0.56 (0.13, 0.99) <sup>d</sup>
Race/ethnicity		
Non-Hispanic White	Referent	Referent
Non-Hispanic Black	−0.25 (−0.42, −0.08) <sup>d</sup>	−0.09 (−0.39, 0.21)
Hispanic	0.12 (−0.05, 0.30)	0.21 (0.04, 0.39) <sup>d</sup>
Non-Hispanic Asian	0.36 (0.14, 0.59) <sup>d</sup>	−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) <sup>d</sup>	—
Beans, nuts or soy (vs. not)	0.19 (0.07, 0.32) <sup>d</sup>	−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) <sup>d</sup>
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)

<sup>a</sup>The natural logarithm of urinary total mercury ( $\mu\text{g/g}$  creatinine) is the dependent variable.

<sup>b</sup>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.

<sup>c</sup>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.

<sup>d</sup>Wald test  $p < 0.05$ .

<sup>e</sup>Wald test  $p < 0.10$ .

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 nondietary 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]. In addition, 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], and 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 h had, on average, higher THg concentrations and those who reported drinking wine in the past 24 h 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. In addition, 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 et al. 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 h 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 h 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 h, 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 vs. typical food consumption patterns with mercury, so any misclassification would likely be nondifferential, resulting in a bias toward the null. At the same time, the prior 24 h 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 Supplementary Table 1). In addition, 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 h (e.g., wine and liquor among non-seafood consumers with UHg, see Supplementary 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 nonzero 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 h dietary recall allowed sufficient power to investigate over 30 different food categories. In addition, 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.

## 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>.

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## Compliance with ethical standards

**Conflict of interest** The authors declare that they have no conflict of interest.

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