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Ethnic, Geographic and Dietary Differences in Arsenic Exposure in the Multi-Ethnic Study of Atherosclerosis (MESA)

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Abstract

Differences in residential location as well as race/ethnicity and dietary habits may result in differences in inorganic arsenic (iAs) exposure. We investigated the association of exposure to iAs with race/ethnicity, geography and dietary intake in a random sample of 310 White, Black, Hispanic and Chinese adults in the Multi-Ethnic Study of Atherosclerosis from 6 US cities with inorganic and methylated arsenic (Σ As) measured in urine. Dietary intake was assessed by food frequency questionnaire. Chinese and Hispanic race/ethnicity was associated with 82% (95% CI: 46%, 126%) and 37% (95% CI: 10%, 70%) higher urine arsenic concentrations, respectively, compared to White participants. No differences were observed for Black participants compared to Whites. Urine arsenic concentrations were higher for participants in Los Angeles, Chicago and New York compared to other sites. Participants that ate rice 2 times/week had 31% higher urine

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arsenic compared to those that rarely/never consumed rice. Participants that drank wine 2 times/week had 23% higher urine arsenic compared to rare/never wine drinkers. Intake of poultry or non-rice grains was not associated with urinary arsenic concentrations. At the low-moderate levels typical of the US population, exposure to iAs differed by race/ethnicity, geographic location and frequency of rice and wine intake.

INTRODUCTION

Inorganic arsenic (iAs) is widespread in the environment. At low-moderate levels (<100 µg/L in drinking water), increasing evidence supports the role of arsenic in cardiovascular disease, diabetes and some cancers.^{1–8} For the general population, the main sources of iAs are through contaminated groundwater (especially in some geographical areas), certain foods and ambient air.^{9–14} While exposure to and the consequences of chronic exposure to iAs in drinking water have been well-documented, there is increasing concern about dietary sources as iAs is found in some foods, including rice, grains, poultry, fruit juice and wine.^{9,11,14–21} In the National Health and Nutrition Examination Survey (NHANES), a representative sample of the US population, rice consumption was associated with increased urinary arsenic concentrations in children²² and adults.^{23,24} The presence of arsenic in food is generally due to naturally occurring soil contamination, past use of pesticides or contaminated irrigation water.^{25,26} In poultry, however, the presence of arsenic can be due to the use of arsenical drugs as feed additives, such as roxarsone which was used in chicken production in the US until its ban in 2013²⁷ and nitarsone, which was used in turkey production at least through the end of 2015.²⁸

In urine, arsenic concentrations reflect ongoing exposures and are well correlated with arsenic intake from drinking water and dietary sources.^{29–31} After exposure, iAs and its methylated metabolites (methylarsonate [MMA] and dimethylarsinate [DMA]) are widely distributed within the body and excreted mostly through the kidneys.³² Most of the US population utilizing public water systems consumes water with arsenic levels below the US Environmental Protection Agency's (EPA) maximum contaminant level (MCL, 10 µg/L or 10 ppb). In a study of public water systems in 25 US states, nearly 97% of the population served by these water systems were estimated to have consumed water containing arsenic at levels below 10 ppb.³³ Despite this, few studies have evaluated arsenic exposure in US populations exposed to arsenic levels in drinking water below the US EPA's MCL. In these populations, foods are likely the major source of arsenic exposure.³⁴ Existent studies^{9,20–22,34,35}, moreover, have not evaluated geographical, race/ethnicity and dietary differences combined, including assessing the role of diet and geography in explaining racial/ethnic differences in arsenic exposure. Our objective was to evaluate the impact of race/ethnicity, geography and diet on iAs exposure in general US populations exposed to low arsenic levels in drinking water. To achieve this goal, we used data from the Multi-Ethnic Study of Atherosclerosis (MESA), a population based study of adults 45 to 84 years of age from Forsyth County (Winston-Salem), NC; New York, NY; Baltimore, MD; St. Paul, MN; Chicago, IL and Los Angeles, CA between 2000 and 2002 with detailed demographic and dietary information and urine available for arsenic speciation.³⁶

METHODS

Study population

MESA was originally designed as multi-center and multi-ethnic longitudinal study to investigate subclinical cardiovascular disease, enrolling a total of 6,814 non-Hispanic White (“White”), non-Hispanic Black (“Black”), Hispanic and Chinese participants who were free of cardiovascular disease between 2000 and 2002. White participants were recruited from all six study sites, Black participants were recruited from all sites except St. Paul, Hispanic participants were only recruited in Los Angeles, New York and St. Paul, and Chinese participants were only recruited in Chicago and Los Angeles. Institutional review board approval was granted at each study site, and all participants provided written informed consent. For the current study, conducted to evaluate exposure to arsenic and other metals in a sub-sample³⁷, we used a random-sampling strategy, stratified by race/ethnicity and city to select 310 participants from the MESA baseline exam for urine arsenic analysis with the goal of obtaining a predefined number of participants by race/ethnicity (90 White, 75 Black, 75 Hispanic, and 70 Chinese) and by city (30 from Winston-Salem, 55 from New York, 30 from Baltimore, 40 from St. Paul, 65 from Chicago and 90 from Los Angeles).

Urine arsenic assessment

MESA participants provided spot urine samples as part of the baseline MESA examination (2000- 2002) and samples were stored at -70°C or lower. In 2013, 1 mL of urine from the study participants was shipped on dry ice to the Trace Element Laboratory at the University of Graz (Graz, Austria). Arsenic species were measured in urine using anion-exchange high performance liquid chromatography (HPLC; Agilent 1100, Agilent Technologies, Waldbronn, Germany) coupled with inductively coupled plasma mass spectrometry (ICP-MS; Agilent 7700× ICPMS) following an established protocol.³⁸ Arsenite was oxidized to arsenate with hydrogen peroxide to assess overall iAs. The interassay coefficients of variation for arsenate, MMA, DMA, and arsenobetaine (together with other unretained arsenic species) were 6.0%, 6.5%, 5.9%, and 6.5%, respectively.³⁸ The limits of detection were 0.1 $\mu\text{g As/L}$ for arsenate, MMA, DMA and arsenobetaine. The percentage of participants with urinary concentrations below the limit of detection was 45.8%, 14.2%, 0%, and 3.9% for arsenate, MMA, DMA and arsenobetaine, respectively. For participants with urine arsenic levels below the limit of detection, a level equal to the limit of detection divided by the square root of two was assigned.

In populations with frequent seafood intake, such as MESA, the assessment of exposure to iAs is difficult because seafood arsenicals (arsenobetaine, arsenosugars and arsenolipids), considered to have low-toxicity, contribute to DMA and possibly MMA. To address this limitation, we calibrated urinary concentrations of iAs, DMA and MMA using a residual-based method described in detail elsewhere.³⁷ In summary, non-seafood related urinary concentrations of iAs, DMA and MMA were calibrated by regressing their original concentrations by arsenobetaine, a biomarker of recent seafood intake, and extracting model residuals. The mean levels of the corresponding arsenic species among participants with low arsenobetaine (urinary arsenobetaine $< 1 \mu\text{g/L}$) were then added to the residuals. The resulting calibrated estimates represent concentrations of inorganic and methylated arsenic

species from exposure to iAs not explained by recent seafood intake. The sum of iAs and its methylated metabolites (Σ As) was estimated as the sum of calibrated concentrations of iAs, MMA and DMA and used as our biomarker of arsenic exposure not derived from seafood.

Demographic, dietary and other variables

Demographics (age, sex, race/ethnicity), highest educational attainment and dietary intake were assessed by questionnaires at the MESA baseline examination. Participant race/ethnicity was assessed by self-report and categorized as non-Hispanic White, non-Hispanic Black, Hispanic and Chinese.

Information on the usual intake of rice, non-rice grains (e.g., pasta, bread, and cereal), poultry and alcoholic beverages during the past year was assessed using a 120-item food frequency questionnaire (FFQ, Supplemental Table 1).^{39–41} The FFQ was modified to include Chinese and Hispanic foods to accommodate the MESA study population. Frequency of food intake (rice, non-rice grains and poultry) was collected in 9 categories: “rare or never”, “1 time/month”, “2–3 times/month”, “1 time/week”, “2 times/week”, “3–4 times/week”, “5–6 times/week”, “1 time/day” and “2 times/day”. Frequency of wine intake was collected in 9 categories: “rare or never”, “1–3 times/month”, “1 time/week”, “2–4 times/week”, “5–6 times/week”, “1 time/day”, “2–3 times/day”, “4–5 times/day” and “6 times/day”. For this analysis, intake of white and brown rice and poultry were categorized as rare/never (<1 time per month), 2–4 times per month and 2 times per week; intake of non-rice grains was categorized as rare/never (<1 time/week), 2–6 times/week and 1 time/day; and intake of wine was categorized as rare/never (<1 time per month), 1–4 times per month and 2 times per week.

Body mass index was calculated by dividing measured weight in kilograms by measured height in meters squared. Urine creatinine, used to adjust for urine dilution in spot urine samples in statistical models, was determined using a Jaffe rate reaction measured with the Vitros 950IRC instrument.

Statistical analysis

We estimated multivariable adjusted ratios of the geometric means (GM) of urinary concentrations of iAs, DMA, MMA and Σ As across categories of race/ethnicity, study site, and frequency of intake of rice, non-rice grains, poultry and wine using linear regression models on log-transformed arsenic variables. The geometric mean ratio (95% confidence interval) was obtained by exponentiating the model coefficient. All models adjusted for urinary creatinine (log-transformed), sex, age (continuous), education (less than high school; high school; some college/technical school; college/graduate degree) and body mass index (continuous)⁴² (“Model 1”). Models for city and frequency of food/wine intake were further adjusted for race/ethnicity (“Model 2”). Models for frequency of wine, poultry and non-rice grains intake were further adjusted for frequency of rice intake (rare/never any rice; any rice 2–4 times per month; any rice 2 times per week) (“Model 3”). Models for race/ethnicity were further adjusted for city (“Model 2”) and frequency of rice intake (“Model 3”). For frequency of rice intake, analyses were conducted for any rice intake (white rice, brown rice, and mixed rice dishes [e.g. fried rice, arroz con pollo]) combined and for white rice among

participants that rarely/never consumed brown rice. Analyses for brown rice could not be conducted due to the limited sample size. Statistical analyses were performed using R statistical software (version 3.0.2). Sensitivity analyses further adjusting for smoking status showed similar findings (data not shown). To evaluate potential interactions between race/ethnicity and city, we estimated multivariable adjusted GMs of urinary arsenic concentrations by city stratified by race/ethnicity and multivariable adjusted ratios of the GMs by race/ethnicity restricted to participants in Los Angeles (only study site that recruited all race/ethnicities).

RESULTS

Racial/ethnic differences in arsenic exposure

Among the study participants, 29% were White, 24% were Black, 24% were Hispanic, and 23% were Chinese. In bivariate analyses, urine arsenic concentrations were higher in Chinese participants compared with other races/ethnicities (Table 1, Supplemental Table 2). After adjustment for sex, age, education, body mass index and urine creatinine, the GM ratios (95% CI) comparing Chinese and Hispanic participants with White participants were, respectively, 1.71 (1.28, 2.29) and 1.35 (0.99, 1.83) for iAs; 2.01 (1.53, 2.65) and 1.33 (0.99, 1.77) for MMA; 1.88 (1.54, 2.30) and 1.42 (1.15, 1.75) for DMA; and 1.91 (1.57, 2.32) and 1.41 (1.15, 1.73) for Σ As (Table 2, Model 1). These findings were attenuated after adjustment for city (Table 2, Model 2) and frequency of rice intake (Table 2, Model 3). Overall, no differences were observed in urine arsenic for Black participants compared to Whites (Table 2); although Black participants in Los Angeles had higher concentrations of MMA compared to Whites in Los Angeles (GM ratio: 1.60, 95% CI: 1.00, 2.57) (Supplemental Table 3). Among participants in Los Angeles (N= 90) urine arsenic concentrations were higher for Chinese and Hispanic participants compared to Whites, although these findings were not statistically significant for Hispanic participants (Supplemental Table 3).

Geographic differences in arsenic exposure

About 10% of study participants were from Winston-Salem, 18% from New York, 10% from Baltimore, 13% from St. Paul, 21% from Chicago and 29% from Los Angeles (Table 1). In bivariable analyses, urine arsenic concentrations were highest in participants in Los Angeles compared with other cities (Table 1, Supplemental Table 4). After adjustment for sex, age, education, body mass index and urine creatinine, the GM ratios (95% CI) for participants in Los Angeles compared to participants in Winston-Salem were 1.67 (1.13, 2.45) for iAs, 1.56 (1.07, 2.26) for MMA, 1.56 (1.17, 2.08) for DMA and 1.57 (1.19, 2.07) for Σ As (Table 2, Model 1). Urinary concentrations for Σ As were also significantly higher in participants in New York (GM 1.36 [95% CI 1.01, 1.83]) and Chicago (GM ratios 1.43 [1.08, 1.90]) compared to participants in Winston-Salem. Geographical differences were markedly attenuated and no longer statistically significant after further adjustment for race/ethnicity (Table 2, Model 2) and remained similar after further adjustment for rice intake (Table 2, Model 3). In an analysis stratified by race/ethnicity, urine concentrations for Σ As were higher for Black participants in Los Angeles and for Hispanic participants in Los Angeles and New York compared to their counterparts in other cities (Figure 1). Urinary

concentrations for Σ As did not differ by city among White and Chinese participants (Figure 1).

Dietary differences in arsenic exposure

Rice intake—13.9% of participants reported rarely or never eating rice compared to 33.9% that ate rice 2–4 times per month and 52.3% that ate rice 2 times per week (Table 1). After adjustment for age, sex, education, body mass index and urine creatinine, the GM ratios (95%CI) for rice intake 2 times per week compared to rare/never rice intake were and 1.37 (1.01, 1.87) for iAs, 1.61 (1.20, 2.17) for MMA, 1.71 (1.37, 2.13) for DMA, and 1.70 (1.37, 2.10) for Σ As (Table 3, Model 1). Among participants that mainly consume white rice (i.e., rarely/never consumed brown rice; 74.5% of study population), more frequent white rice intake was associated with higher urinary arsenic concentrations (GM ratios [95%CI] for 2 times per week of white rice intake compared to rare/never intake were 1.25 [0.94, 1.67] for iAs, 1.48 [1.12, 1.95] for MMA, 1.63 [1.32, 2.00] for DMA, and 1.60 [1.31, 1.95] for Σ As) (data not shown). In an analysis of the association between rice and arsenic stratified by race/ethnicity, urine concentrations for Σ As were markedly higher among Hispanic and Chinese participants in the category with more frequent rice intake compared to White and Black participants in the same category (Figure 2).

Wine intake—55.8% of participants reported rarely or never drinking wine compared to 29.7% that consumed wine 1–4 times per month and 14.5% that consumed wine 2 times per week (Table 1). After full adjustment, more frequent wine intake was associated with higher urine arsenic concentrations, although these findings were not statistically significant (GM ratios [95%CI] for 2 times per week compared to rare/never intake were 1.12 [0.81, 1.53] for iAs, 1.29 [0.96, 1.75] for MMA, 1.17 [0.94, 1.45] for DMA and 1.20 [0.97, 1.48] for Σ As) (Table 3, Model 3).

Poultry intake—21.7% of participants reported rarely or never eating poultry compared to 39.2% that ate poultry 2–4 times per month and 39.2% that ate poultry 2 or more times per week (Table 1). After adjustment for sex, age, body mass index, urine creatinine, race/ethnicity and rice intake, the frequency of poultry intake was not associated with urine arsenic concentrations (Table 4, Model 3).

Non-rice grains intake—61.9% of participants reported intake of non-rice grains 2–6 times per week compared to 31.6% 1 time per day and 6.5% rarely/never eating non-rice grains (Table 1). Frequent intake of non-rice grains was not associated with urinary arsenic concentrations after adjustment for sex, age, body mass index, urine creatinine, race/ethnicity and rice intake (Table 4, Model 3).

DISCUSSION

In this study, urinary arsenic concentrations differed mostly by race/ethnicity and rice intake. Urinary arsenic concentrations were higher among Hispanic and Chinese participants and lower in White and Black participants and these associations remained after adjustment for city and rice intake. More frequent rice intake was associated with a small increase in urinary arsenic concentrations, with some attenuation in the association after adjustment for

race/ethnicity. Geographical differences in urinary arsenic concentrations seemed mostly related to the differential race/ethnic distribution by city and their differences in rice intake.

Our findings of higher arsenic levels in Hispanics are consistent with findings from the National Health and Nutrition Examination Survey (NHANES) for Mexican-Americans.^{34,43,44} Using data from 2,557 participants aged 6 years in NHANES 2003–2004, urine arsenic levels were higher in Mexican-Americans than in White and Black participants (geometric means for Mexican-American, White and Black participants were 8.6, 7.5 and 8.3 µg/g creatinine, respectively, for total arsenic and 4.4, 3.4 and 3.1 µg/g creatinine, respectively, for DMA).⁴³ While differences in arsenic exposure levels in Chinese American populations have not been specifically reported in the US, urinary arsenic concentrations were higher than Whites for the group of other race/ethnicity, which includes Chinese American, in NHANES.⁴⁵ In a study of 63 White, Asian and Somali Black-African adults from Leicester, United Kingdom, mean total concentrations of arsenic in urine were higher in Asian participants (24.5 µg/g creatinine) compared to Whites (20.9 µg/g creatinine) and Somali Black-Africans (7.2 µg/g creatinine), although this study did not account for seafood intake and the Asian participants included individuals with ancestral links to India, Pakistan, and Bangladesh.⁴⁴

Urine arsenic concentrations were higher in participants in Los Angeles, New York and Chicago. However, after adjustment for race and rice intake these differences were attenuated. Few studies^{46–48} have compared differences in arsenic exposure in groundwater across geographic areas. In the Strong Heart Study, a population where ground water is a major source of drinking water, median urine concentrations for ΣAs were higher in participants in Arizona (12.5 µg/g), intermediate in participants in North and South Dakota (9.1 µg/g), and lower in participants in Oklahoma (4.4 µg/g), consistent with data from the US Geological Survey.⁴⁹ Although arsenic concentrations in groundwater in the United States are generally higher in the Western US, parts of the Midwest and in the Northeast^{46–49}, in our study we expect that most participants were connected to community water systems and water systems in the six cities in our study were in compliance with the US Environmental Protection Agency's safe drinking water arsenic MCL, and we found little variability by race/ethnicity by city. Information on the source of drinking water for study participants or the arsenic levels in participants' drinking water were unavailable. The geographic differences in urinary arsenic concentrations observed in our study could also be explained by differences in ambient air, which is also source of arsenic exposure.²⁵

Dietary sources are likely to be the main sources of arsenic exposure in populations with low arsenic levels in drinking water.^{19,43,50} Consistent with other studies^{9,22,23,35,51–54}, in our study, urinary arsenic concentrations were higher among participants with more frequent intake of rice compared to participants that rarely or never consumed rice. The attenuation of the association between urinary arsenic and rice intake after adjustment for race/ethnicity is also consistent with previous studies that found significant variations in rice consumption and urinary concentrations of arsenic in different racial groups.^{23,51} Due to the limited categories used to assess food intake in the FFQ, our ability to quantify the frequency of rice intake may differ by race/ethnicity, particularly for racial/ethnic groups with frequent rice intake for which there may be variability in intake within the highest rice intake category

used in this study; therefore, findings after adjustment for race/ethnicity may reflect differences in measurement error in rice intake assessed using the FFQ. These findings could also reflect cultural differences in how rice and rice-containing dishes are prepared which could potentially alter arsenic concentrations in these dishes, however information on food preparation was unavailable, and we are unable to confirm this hypothesis.

Arsenic in wine can occur as a consequence of soil contamination, insecticide or herbicide use in grape production, or during wine production.^{17,18,55} We found higher arsenic levels associated with frequent intake of wine, although these findings were not statistically significant. This finding is consistent with previous studies that found higher concentrations of urinary^{56–58} and toenail¹⁷ arsenic associated with alcohol intake. In a study of 852 adults aged 25–74 years in New Hampshire, consumption of wine, especially white wine, was positively associated with toenail arsenic concentrations.¹⁷ Using data from 252 adults aged 40 years in Lanyang Basin, Taiwan, participants that consumed alcohol >1 day/week had higher urinary concentrations of iAs (mean: 120.0 µg/l vs 93.7µg/l) and ΣAs (mean: 193.3µg/l vs 176.8µg/l) compared to participants that did not.⁵⁶ However, these differences were not statistically significant, and type of alcohol (e.g., wine, beer, liquor) was not examined. In our study, frequent intake of beer was not associated with urinary arsenic concentrations (data not shown).

The use of arsenic-based drugs (roxarsone and nitarsone) in poultry production may contribute to dietary exposure to arsenic before the banning of these drugs by the FDA in 2013 and 2015, respectively.^{27,28,59–61} Urinary arsenic concentrations were not associated with intake of poultry or non-rice grains in this study. In a study of 10,868 participants aged 6 years in NHANES 2003–2010, increased urinary arsenic species were associated with increased consumption of grain products (DMA, MMA) and meat/poultry (DMA).³⁵ Using data from 3,329 participants aged 6 years in NHANES 2003–2010 with undetectable arsenobetaine concentrations, poultry intake was positively associated with concentrations of total arsenic and DMA in urine (GM ratios were 1.12 [95% CI: 1.04, 1.22] for total arsenic and 1.13 [95% CI: 1.06, 1.20] for DMA comparing the highest quartile of poultry consumption to non-consumers).⁶²

Intake of fruit juices was assessed in the food frequency questionnaire using questions on the intake of “orange juice and grapefruit juice” and “any other fruit juices (apple, grape, punch, kool-aid, guava juice, etc.)”, because of these categories we were unable to separate intake of juices that may contain arsenic (i.e., apple, pear and grape juice) from other fruit juices and were therefore unable to evaluate the impact of juice intake on urinary arsenic concentrations in this study population.

Strengths and limitations

The use of a well-characterized multi-ethnic cohort designed to recruit participants from non-White groups, including Black, Hispanic and Chinese participants allowed us to evaluate differences in arsenic exposure in previously unexplored racial groups with varying geographic and dietary patterns. The high quality laboratory methods to measure arsenic species in urine allowed us to assess exposure to inorganic and methylated arsenic species not derived from seafood in a general population exposed to low arsenic levels in drinking

water. Urinary arsenic biomarkers are ideal measures to make ethnic or geographical comparisons in arsenic exposure as they integrate multiple sources including water, food and air.

A few limitations should be taken into account. First, although a random-stratified strategy was used to select participants in this study, MESA participants in the overall cohort were enrolled at each study site with the intent of having specific distributions across strata defined by race/ethnicity, gender, and age group, and not by random sample; because of this sampling strategy, MESA participants might not be representative of populations in each city. Also, data for some races/ethnicities were unavailable for some study cities, and we were unable to evaluate associations comparing all races/ethnicities in all cities. Third, the information on food intake was based on self-report using a FFQ. Self-reported food intake might not reflect actual arsenic concentrations in these foods. The use of a FFQ with defined categories for intake might limit our ability to precisely quantify rice intake and dietary measurement error in estimating rice intake can be different across race/ethnic groups. Urine arsenic was assessed in spot urine samples and we accounted for differences in urine dilution by adjusting for urine creatinine. Lastly, arsenic species measured in this study included arsenate, MMA and DMA; data was unavailable for other arsenic species including roxarsone and nitarsone, which are responsible for arsenic in poultry.^{27,28,59–61}

Conclusions

Using data from a population with 4 different race/ethnic groups from 6 different geographical areas of the United States and a wide range of food intake, we found differences in urinary arsenic concentrations by race/ethnicity and intake of rice, while the geographical differences were mostly explained by the differential distribution of race/ethnicity and rice intake across cities. While chronic exposure to moderate arsenic levels in drinking water has been associated with the development of cancer^{63,64}, cardiovascular disease^{4,65}, and diabetes^{5,66,67}, little is known about the health effects of chronic exposure to low arsenic levels through food, in particular for racial/ethnic groups that are frequent consumers of rice such as Chinese Americans and Hispanics. Research is needed to assess whether the observed differences in arsenic exposure by rice intake and across racial/ethnic groups may explain differences in risk for arsenic-related disease across US populations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Figure 1.

Adjusted geometric means of urinary concentrations for the sum of inorganic arsenic + methylated species (Σ As) by city stratified by race/ethnicity. Points represent the adjusted geometric mean of urinary concentrations for Σ As. Horizontal lines represent 95% confidence intervals (CIs). Model 1 was adjusted for urine creatinine, sex, age, education and body mass index. Model 2 was further adjusted for frequency of rice intake.

Figure 2.

Adjusted geometric means of urinary concentrations for the sum of inorganic arsenic + methylated species (Σ As) by frequency of rice intake stratified by race/ethnicity. Points represent the adjusted geometric mean of urinary concentrations for Σ As. Horizontal lines represent 95% confidence intervals (CIs). Model 1 was adjusted for urine creatinine, sex, age, education and body mass index. Model 2 was further adjusted for city.

Table 1Characteristics of participants overall and by tertiles of ΣAs in urine^a

	Overall	Urinary concentrations of ΣAs (µg/L)		
No. of participants	310	< 2.1 100	2.1 to 4.5 105	> 4.5 105
Sex, %				
Female	134 (43.2)	51 (51.0)	35 (33.3)	48 (45.7)
Male	176 (56.8)	49 (49.0)	70 (66.7)	57 (54.3)
Age, years	61.4 ± 0.5	59.4 ± 0.9	63.3 ± 0.9	61.4 ± 0.9
Race/ethnicity, %				
White	90 (29.0)	42 (42.0)	28 (26.7)	20 (19.0)
Chinese	70 (22.6)	9 (9.0)	22 (21.0)	39 (37.1)
Black	75 (24.2)	34 (34.0)	26 (24.8)	15 (14.3)
Hispanic	75 (24.2)	15 (15.0)	29 (27.6)	31 (29.5)
City, %				
Winston-Salem, NC	30 (9.7)	16 (16.0)	11 (10.5)	3 (2.9)
New York, NY	55 (17.7)	15 (15.0)	21 (20.0)	19 (18.1)
Baltimore, MD	30 (9.7)	21 (21.0)	3 (2.9)	6 (5.7)
St. Paul, MN	40 (12.9)	17 (17.0)	12 (11.4)	11 (10.5)
Chicago, IL	65 (21.0)	23 (23.0)	20 (19.0)	22 (21.0)
Los Angeles, CA	90 (29.0)	8 (8.0)	38 (36.2)	44 (41.9)
Education, %				
< high school	56 (18.1)	11 (11.0)	18 (17.1)	27 (25.7)
High school	48 (15.5)	17 (17.0)	16 (15.2)	15 (14.3)
Some college/technical	82 (26.5)	26 (26.0)	30 (28.6)	26 (24.8)
College/graduate degree	124 (40.0)	46 (46.0)	41 (39.0)	37 (35.2)
Body mass index, kg/m ²	27.4 ± 0.3	27.7 ± 0.5	28.0 ± 0.5	26.6 ± 0.5
Urine creatinine, mg/dL	122.4 ± 4.1	79.4 ± 6.4	124.5 ± 6.3	161.3 ± 6.3
Rice intake, %				
Any rice (N=310)				
Rare/never any rice	43 (13.9)	20 (20.0)	16 (15.2)	7 (6.7)
2–4 times/month any rice	105 (33.9)	41 (41.0)	34 (32.4)	30 (28.6)
2 times/week any rice	162 (52.3)	39 (39.0)	55 (52.4)	68 (64.8)
Ate mainly white rice (N=231)				
Rare/never white rice	49 (21.2)	20 (28.6)	19 (23.2)	10 (12.7)
2–4 times/month white rice	56 (24.2)	22 (31.4)	21 (25.6)	13 (16.5)
2 times/week white rice	126 (54.5)	28 (40.0)	42 (51.2)	56 (70.9)
Ate mainly brown rice (N=78)				
Rare/never brown rice	49 (62.8)	20 (66.7)	19 (63.3)	10 (55.6)
2–4 times/month brown rice	20 (25.6)	9 (30.0)	7 (23.3)	4 (22.2)
2 times/week brown rice	9 (11.5)	1 (3.3)	4 (13.3)	4 (22.2)
Non-rice grain intake, %				

	Overall	Urinary concentrations of ΣAs (µg/L)		
No. of participants	310	< 2.1 100	2.1 to 4.5 105	> 4.5 105
Rare/never	20 (6.5)	7 (7.0)	3 (2.9)	10 (9.5)
2–6 times/week	192 (61.9)	62 (62.0)	69 (65.7)	61 (58.1)
1 times/day	98 (31.6)	31 (31.0)	33 (31.4)	34 (32.4)
Poultry intake, %				
Rare/never	67 (21.7)	14 (14.0)	23 (21.9)	30 (28.8)
2–4 times/month	121 (39.2)	38 (38.0)	43 (41.0)	40 (38.5)
2 times/week	121 (39.2)	48 (48.0)	39 (37.1)	34 (32.7)
Wine intake, %				
Rare/never	173 (55.8)	56 (56.0)	54 (51.4)	63 (60.0)
1–4 times/month	92 (29.7)	32 (32.0)	34 (32.4)	26 (24.8)
2 times/week	45 (14.5)	12 (12.0)	17 (16.2)	16 (15.2)

^aValues represent n (%), except for age, body mass index and urine creatinine for which means ± SE are reported

Table 2

Ratio of geometric mean of urine arsenic concentrations (iAs, MMA, DMA and the sum of iAs + methylated species) by race/ethnicity and city

	N	Model 1	Model 2	Model 3
Race/ethnicity				
iAs				
White	90	1.00 (ref)	1.00 (ref)	1.00 (ref)
Chinese	70	1.71 (1.28, 2.29)	1.56 (1.14, 2.15)	1.49 (1.03, 2.16)
Black	75	0.89 (0.68, 1.18)	0.84 (0.63, 1.11)	0.82 (0.62, 1.10)
Hispanic	75	1.35 (0.99, 1.83)	1.44 (1.04, 1.98)	1.39 (0.99, 1.96)
MMA				
White	90	1.00 (ref)	1.00 (ref)	1.00 (ref)
Chinese	70	2.01 (1.53, 2.65)	1.92 (1.42, 2.61)	1.78 (1.25, 2.55)
Black	75	0.99 (0.76, 1.28)	0.98 (0.75, 1.29)	0.96 (0.73, 1.26)
Hispanic	75	1.33 (0.99, 1.77)	1.30 (0.95, 1.77)	1.23 (0.88, 1.70)
DMA				
White	90	1.00 (ref)	1.00 (ref)	1.00 (ref)
Chinese	70	1.88 (1.54, 2.30)	1.80 (1.44, 2.26)	1.70 (1.31, 2.21)
Black	75	0.85 (0.70, 1.04)	0.85 (0.69, 1.03)	0.83 (0.68, 1.02)
Hispanic	75	1.42 (1.15, 1.75)	1.37 (1.09, 1.72)	1.30 (1.02, 1.66)
ΣAs				
White	90	1.00 (ref)	1.00 (ref)	1.00 (ref)
Chinese	70	1.91 (1.57, 2.32)	1.82 (1.46, 2.26)	1.70 (1.32, 2.19)
Black	75	0.88 (0.73, 1.06)	0.87 (0.72, 1.05)	0.85 (0.70, 1.03)
Hispanic	75	1.41 (1.15, 1.73)	1.37 (1.10, 1.70)	1.30 (1.03, 1.64)
City				
iAs				
Winston-Salem, NC	30	1.00 (ref)	1.00 (ref)	1.00 (ref)
New York, NY	55	1.01 (0.67, 1.53)	0.83 (0.54, 1.26)	0.81 (0.53, 1.24)
Baltimore, MD	30	1.03 (0.66, 1.61)	1.03 (0.66, 1.59)	1.02 (0.66, 1.57)
St. Paul, MN	40	0.89 (0.58, 1.37)	0.66 (0.42, 1.03)	0.65 (0.42, 1.03)
Chicago, IL	65	1.10 (0.74, 1.63)	0.86 (0.57, 1.29)	0.85 (0.56, 1.28)
Los Angeles, CA	90	1.67 (1.13, 2.45)	1.24 (0.83, 1.85)	1.23 (0.82, 1.84)
MMA				
Winston-Salem, NC	30	1.00 (ref)	1.00 (ref)	1.00 (ref)
New York, NY	55	0.97 (0.65, 1.45)	0.86 (0.57, 1.29)	0.83 (0.55, 1.25)
Baltimore, MD	30	0.83 (0.54, 1.28)	0.83 (0.55, 1.26)	0.81 (0.54, 1.24)
St. Paul, MN	40	1.03 (0.68, 1.55)	0.87 (0.56, 1.34)	0.86 (0.56, 1.33)
Chicago, IL	65	1.11 (0.76, 1.63)	0.81 (0.55, 1.20)	0.80 (0.54, 1.18)
Los Angeles, CA	90	1.56 (1.07, 2.26)	1.16 (0.78, 1.70)	1.14 (0.77, 1.68)

	N	Model 1	Model 2	Model 3
DMA				
Winston-Salem, NC	30	1.00 (ref)	1.00 (ref)	1.00 (ref)
New York, NY	55	1.44 (1.06, 1.95)	1.19 (0.88, 1.61)	1.16 (0.86, 1.56)
Baltimore, MD	30	0.92 (0.66, 1.28)	0.92 (0.67, 1.25)	0.90 (0.66, 1.23)
St. Paul, MN	40	1.30 (0.95, 1.78)	0.97 (0.70, 1.34)	0.97 (0.71, 1.34)
Chicago, IL	65	1.48 (1.11, 1.99)	1.10 (0.82, 1.47)	1.06 (0.80, 1.42)
Los Angeles, CA	90	1.56 (1.17, 2.08)	1.13 (0.85, 1.50)	1.10 (0.83, 1.47)
ΣAs				
Winston-Salem, NC	30	1.00 (ref)	1.00 (ref)	1.00 (ref)
New York, NY	55	1.36 (1.01, 1.83)	1.14 (0.85, 1.52)	1.10 (0.82, 1.47)
Baltimore, MD	30	0.93 (0.67, 1.28)	0.93 (0.69, 1.25)	0.91 (0.67, 1.22)
St. Paul, MN	40	1.23 (0.91, 1.67)	0.95 (0.69, 1.29)	0.94 (0.69, 1.28)
Chicago, IL	65	1.43 (1.08, 1.90)	1.05 (0.79, 1.39)	1.03 (0.78, 1.36)
Los Angeles, CA	90	1.57 (1.19, 2.07)	1.13 (0.86, 1.49)	1.12 (0.85, 1.47)

ΣAs: sum of inorganic and methylated arsenic species

Bold indicates statistical significance with a p-value <0.05

Model 1 adjusted for urine creatinine, sex, age, education and body mass index

Model 2 further adjusted for city (for race/ethnicity) or for race/ethnicity (for city)

Model 3 further adjusted for frequency of rice intake

Table 3

Ratio of geometric mean of urine arsenic concentrations by frequency of intake of rice and wine

	N	Model 1	Model 2	Model 3
Rice				
iAs				
Rare/never any rice	43	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–4 times/month any rice	105	1.06 (0.77, 1.46)	1.04 (0.76, 1.43)	1.06 (0.78, 1.45)
2 times/week any rice	162	1.37 (1.01, 1.87)	1.10 (0.79, 1.54)	1.11 (0.79, 1.55)
MMA				
Rare/never any rice	43	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–4 times/month any rice	105	1.23 (0.90, 1.67)	1.22 (0.90, 1.64)	1.24 (0.92, 1.67)
2 times/week any rice	162	1.61 (1.20, 2.17)	1.24 (0.90, 1.70)	1.28 (0.93, 1.77)
DMA				
Rare/never any rice	43	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–4 times/month any rice	105	1.32 (1.05, 1.67)	1.30 (1.04, 1.61)	1.30 (1.04, 1.61)
2 times/week any rice	162	1.71 (1.37, 2.13)	1.32 (1.05, 1.67)	1.29 (1.02, 1.63)
ΣAs				
Rare/never any rice	43	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–4 times/month any rice	105	1.30 (1.04, 1.62)	1.28 (1.03, 1.57)	1.28 (1.03, 1.58)
2 times/week any rice	162	1.70 (1.37, 2.10)	1.31 (1.05, 1.64)	1.29 (1.03, 1.62)
Wine				
iAs				
Rare/never	173	1.00 (ref)	1.00 (ref)	1.00 (ref)
1–4 times/month	92	1.07 (0.85, 1.36)	1.08 (0.86, 1.36)	1.09 (0.86, 1.37)
2 times/week	45	1.01 (0.74, 1.39)	1.11 (0.81, 1.52)	1.12 (0.81, 1.53)
MMA				
Rare/never	173	1.00 (ref)	1.00 (ref)	1.00 (ref)
1–4 times/month	92	1.12 (0.89, 1.40)	1.12 (0.90, 1.39)	1.12 (0.90, 1.40)
2 times/week	45	1.14 (0.84, 1.54)	1.31 (0.98, 1.77)	1.29 (0.96, 1.75)
DMA				
Rare/never	173	1.00 (ref)	1.00 (ref)	1.00 (ref)
1–4 times/month	92	1.07 (0.89, 1.27)	1.07 (0.92, 1.26)	1.07 (0.91, 1.26)
2 times/week	45	1.07 (0.85, 1.35)	1.20 (0.96, 1.49)	1.17 (0.94, 1.45)
ΣAs				
Rare/never	173	1.00 (ref)	1.00 (ref)	1.00 (ref)
1–4 times/month	92	1.08 (0.91, 1.28)	1.09 (0.93, 1.27)	1.09 (0.93, 1.27)
2 times/week	45	1.09 (0.87, 1.37)	1.23 (1.00, 1.52)	1.20 (0.97, 1.48)

ΣAs: sum of inorganic and methylated arsenic species

Bold indicates statistical significance with a p-value <0.05

Model 1 adjusted for urine creatinine, sex, age, education and body mass index

Model 2 further adjusted for race/ethnicity

Model 3 further adjusted for city (for rice) or for frequency of rice intake (for wine)

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Table 4

Ratio of geometric mean of urine arsenic concentrations by frequency of intake of poultry and non-rice grains

	N	Model 1	Model 2	Model 3
Poultry				
iAs				
Rare/never	67	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–4 times/month	121	0.70 (0.53, 0.91)	0.84 (0.63, 1.12)	0.83 (0.62, 1.11)
2 times/week	121	0.70 (0.53, 0.92)	0.84 (0.63, 1.13)	0.82 (0.61, 1.11)
MMA				
Rare/never	67	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–4 times/month	121	0.81 (0.62, 1.05)	1.04 (0.79, 1.37)	1.02 (0.77, 1.34)
2 times/week	121	0.68 (0.52, 0.88)	0.88 (0.66, 1.16)	0.84 (0.63, 1.12)
DMA				
Rare/never	67	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–4 times/month	121	0.74 (0.61, 0.90)	0.95 (0.77, 1.16)	0.92 (0.75, 1.12)
2 times/week	121	0.72 (0.59, 0.88)	0.92 (0.75, 1.14)	0.88 (0.72, 1.09)
ΣAs				
Rare/never	67	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–4 times/month	121	0.74 (0.61, 0.90)	0.94 (0.78, 1.15)	0.91 (0.75, 1.11)
2 times/week	121	0.70 (0.58, 0.86)	0.90 (0.74, 1.10)	0.86 (0.71, 1.05)
Non-rice grains				
iAs				
Rare/never	20	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–6 times/week	192	1.13 (0.74, 1.72)	1.06 (0.71, 1.60)	1.06 (0.70, 1.60)
1 time/day	98	1.05 (0.67, 1.63)	1.01 (0.65, 1.55)	0.99 (0.64, 1.53)
MMA				
Rare/never	20	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–6 times/week	192	1.02 (0.68, 1.53)	0.97 (0.66, 1.43)	0.97 (0.66, 1.44)
1 time/day	98	0.93 (0.61, 1.43)	0.91 (0.61, 1.37)	0.90 (0.60, 1.36)
DMA				
Rare/never	20	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–6 times/week	192	0.90 (0.66, 1.23)	0.84 (0.63, 1.12)	0.84 (0.63, 1.12)
1 time/day	98	0.90 (0.65, 1.25)	0.86 (0.63, 1.16)	0.84 (0.62, 1.13)
ΣAs				
Rare/never	20	1.00 (ref)	1.00 (ref)	1.00 (ref)
2–6 times/week	192	0.92 (0.68, 1.25)	0.86 (0.65, 1.13)	0.86 (0.66, 1.13)
1 time/day	98	0.90 (0.66, 1.24)	0.86 (0.65, 1.15)	0.85 (0.63, 1.13)

ΣAs: sum of inorganic and methylated arsenic species

Bold indicates statistical significance with a p-value <0.05

Model 1 adjusted for urine creatinine, sex, age, education and body mass index

Model 2 further adjusted for race/ethnicity

Model 3 further adjusted for frequency of rice intake

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