Patterns of Beverages Consumed and Risk of Incident Kidney Disease

Casey M. Rebholz,[1] Bessie A. Young,[2,3] Ronit Katz,[3] Katherine L. Tucker,[4] Teresa C. Carithers,[5] Arnita F. Norwood,[6] and Adolfo Correa[6]

Abstract

Background and objectives Selected beverages, such as sugar-sweetened beverages, have been reported to influence kidney disease risk, although previous studies have been inconsistent. Further research is necessary to comprehensively evaluate all types of beverages in association with CKD risk to better inform dietary guidelines.

Design, setting, participants, & measurements We conducted a prospective analysis in the Jackson Heart Study, a cohort of black men and women in Jackson, Mississippi. Beverage intake was assessed using a food frequency questionnaire administered at baseline (2000–2004). Incident CKD was defined as onset of eGFR<60 ml/min per 1.73 m^2 and ≥30% eGFR decline at follow-up (2009–13) relative to baseline among those with baseline eGFR ≥60 ml/min per 1.73 m^2. Logistic regression was used to estimate the association between the consumption of each individual beverage, beverage patterns, and incident CKD. Beverage patterns were empirically derived using principal components analysis, in which components were created on the basis of the linear combinations of beverages consumed.

Results Among 3003 participants, 185 (6%) developed incident CKD over a median follow-up of 8 years. At baseline, mean age was 54 (SD 12) years, 64% were women, and mean eGFR was 98 (SD 18) ml/min per 1.73 m^2. After adjusting for total energy intake, age, sex, education, body mass index, smoking, physical activity, hypertension, diabetes, HDL cholesterol, LDL cholesterol, history of cardiovascular disease, and baseline eGFR, a principal components analysis–derived beverage pattern consisting of higher consumption of soda, sweetened fruit drinks, and water was associated with significantly greater odds of incident CKD (odds ratio tertile 3 versus 1 =1.61; 95% confidence interval, 1.07 to 2.41).

Conclusions Higher consumption of sugar-sweetened beverages was associated with an elevated risk of subsequent CKD in this community-based cohort of black Americans.

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[1]Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland; [2]Division of Nephrology, Veterans Affairs Puget Sound Health Care Center, Seattle, Washington; [3]Kidney Research Institute, University of Washington, Seattle, Washington; [4]Department of Biomedical and Nutritional Sciences, University of Massachusetts Lowell, Lowell, Massachusetts; [5]Department of Nutrition and Hospitality Management, University of Mississippi, Oxford, Mississippi; and [6]Department of Medicine, University of Mississippi Medical Center, Jackson, Mississippi

Correspondence:
Dr. Casey M. Rebholz, Welch Center for Prevention, Epidemiology, and Clinical Research, Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, 2024 East Monument Street, Suite 2-500, Baltimore, MD 21287. Email: crebhol1@jhu.edu

Introduction

The Dietary Guidelines for Americans and the American Heart Association recommend limiting dietary intake of added sugars, in part, by avoiding sugar-sweetened beverages because of their known association with poor health outcomes, including weight gain, type 2 diabetes, hypertension, and cardiovascular disease (1–5). A variety of other beverages, including alcohol, coffee, and fruit juice, have also been related to these health outcomes. Health promotion messages often focus on restricting selected unhealthy beverage choices, but there is a lack of comprehensive information about the health implications of the wide range of beverage options.

Preliminary research has shown that sugar-sweetened (regular) soda as well as artificially sweetened soda could influence kidney disease risk, although the evidence is not consistent (6–9). In the Atherosclerosis Risk in Communities study, we observed an independent dose-response relationship between higher diet soda consumption and risk of incident ESKD (10). Further research is necessary to comprehensively assess all types of beverages in association with kidney disease risk. Identifying kidney protective beverages could inform United States dietary guidelines as well as clinical guidelines for kidney disease prevention.

Although blacks experience a disproportionate burden of kidney disease compared with non-Hispanic whites in the United States, blacks are under-represented in the scientific literature (11,12). To address these gaps, the overall objective of our study was to examine participant characteristics associated with patterns of beverage consumption and to assess the association between beverage consumption patterns and subsequent risk of kidney disease among black adults in the general population.

Materials and Methods

Study Design and Study Population

The Jackson Heart Study (JHS) is a prospective, community-based cohort study of 5306 black adult men and women residing in three counties (Hinds, Madison, and Rankin) in Jackson, Mississippi (13). Adults were recruited to participate in a baseline examination in 2000–2004 (study visit 1) (14). Participants subsequently returned for follow-up examinations in 2005–2008 (study visit 2) and 2009–2013 (study visit 3). The design and methods of the JHS have been previously described in detail (15). For the purpose of this study, we excluded study participants with incomplete or invalid dietary data, defined as more than five items missing on the food frequency questionnaire or implausible total energy intake (<600 or >4800 kcal) (n=272); missing derived dietary data (n=237); missing serum creatinine at baseline (n=73) or at follow-up (n=1326); reduced kidney function at baseline (eGFR<60 ml/min per 1.73 m^2) (n=138); or those with missing data on covariates, including smoking status (n=28), body mass index (BMI; n=1), physical activity (n=7), diabetes status (n=3), HDL cholesterol (n=201), LDL cholesterol (n=16), and education (n=1). The analytic sample size was 3003. The Institutional Review Boards at University of Mississippi Medical Center and Johns Hopkins University (IRB00005609) reviewed and approved the study protocol. Participants provided written documentation of informed consent. Procedures were followed in accordance with the Declaration of Helsinki.

Assessment of Beverage Consumption

Dietary intake of foods and beverages was assessed at baseline using a modified version of the Lower Mississippi Delta Nutrition Intervention Research Initiative Food Frequency Questionnaire, which was originally developed for residents of the lower Mississippi Delta region of the United States and subsequently validated for use in the JHS (16–18). The 158-item food frequency questionnaire was administered by trained and certified research staff at baseline (study visit 1, 2000–2004). Derived dietary intake (total energy intake) was calculated on the basis of responses on the food frequency questionnaire using the University of Minnesota Nutrition Data System for Research database.

We investigated patterns of beverage consumption and individual types of beverages. Beverage consumption was energy-adjusted using the residual method, which uses the residual from regression models with dietary intake (beverage) as the dependent variable and energy intake as the independent variable (19,20). Principal components analysis (PCA) was used to empirically characterize patterns of beverage consumption. PCA identifies underlying components (patterns) of beverage consumption on the basis of linear combinations of the types of beverages consumed, with the first component explaining the largest amount of between-person variation, the second component explaining the second largest amount of variation, the third component explaining the third largest amount of variation, and the fourth component explaining the fourth largest amount of variation (21–23). Factor loadings are the correlation of each individual beverage with each component (beverage pattern). A continuous score for each component (*i.e.*, each beverage pattern) was constructed for each participant by multiplying weights (factor loadings) for each beverage by their reported frequency of consumption. These scores were used in the analysis representing level of adherence to each beverage pattern. Types of beverages included soda, diet soda, fruit-flavored drinks (*e.g.*, Hi-C, lemonade, Sunny Delight, Snapple, Kool-Aid, Tang), 100% fruit juice (100% orange juice or grapefruit juice; other 100% fruit juices including apple juice), vegetable juice, milk, coffee, tea, alcohol (beer, wine, liquor), and water. In this study population, the majority of the coffee and tea consumed was sweetened with sugar, although some used an artificial sweetener, particularly with tea. The association between beverage consumption and incident CKD was expressed according to tertiles with the lowest tertile as the reference group and continuously (per one additional serving per week for individual beverages and per one unit higher in the beverage pattern scores).

Ascertainment of Kidney Disease

The primary outcome was incident CKD, defined as eGFR<60 ml/min per 1.73 m^2 at visit 3 accompanied by ≥30% eGFR decline at visit 3, relative to baseline among those with baseline eGFR ≥60 ml/min per 1.73 m^2.

Serum creatinine concentration was measured at baseline and visit 3 using a multipoint enzymatic spectrophotometric assay with an automated analyzer (Vitros Analyzer; Ortho-Clinical Diagnostics, Raritan, NJ). For this outcome definition, calibrated and standardized serum creatinine values were used to estimate eGFR with the 2009 CKD Epidemiology Collaboration equation (24,25).

Measurement of Covariates

At the baseline study visit, a structured questionnaire was administered by trained study personnel to collect information on demographics (age, sex), socioeconomic status (education level), lifestyle factors (self-reported current cigarette smoking, physical activity), health history, and medication use. Physical activity was assessed as an index of leisure-time activity including the duration of walking, biking, and television watching (26).

Body weight and height were measured by certified technicians and nurses. BMI was calculated as weight (kilograms) divided by height (meters) squared. A standardized random zero sphygmomanometer was used to measure BP twice when study participants were seated. The mean of the two BP measurements was used in the analysis.

Blood specimens were collected from study participants at baseline and shipped to the University of Minnesota Central Laboratory for laboratory analysis (27). Glucose was measured on a chemistry autoanalyzer (Vitros Analyzer), and glycated hemoglobin was measured using HPLC. After precipitation of non-HDL cholesterol with magnesium dextran, the concentration of HDL cholesterol was measured.

Hypertension was defined as systolic BP ≥140 mm Hg, diastolic BP ≥90 mm Hg, or use of antihypertensive medication. Diabetes was defined as fasting glucose ≥126 mg/dl, hemoglobin A1c ≥6.5%, self-report of a diabetes diagnosis, or medication use for diabetes. History of cardiovascular disease was defined as self-reported history of coronary heart disease, abnormalities detected on an electrocardiogram, self-reported history of carotid angioplasty, or self-reported diagnosis of a stroke.

Table 1. Factor loadings for individual beverages within each component/beverage pattern

Individual Beverages	Component/Beverage Pattern[a,b]			
	1	2	3	4
Citrus juice	−0.0071	**0.5788**	−0.0386	0.0031
Other fruit juice	−0.0308	**0.6189**	0.0128	0.0975
Vegetable juice	0.0875	**0.2918**	−0.0209	−0.0891
Whole milk	−0.0476	0.1413	**−0.3093**	−0.0711
Reduced-fat milk	−0.1709	−0.0872	**0.3913**	−0.0693
Low-fat milk	−0.0044	0.1200	0.1949	−0.0539
Soda	−0.0840	**−0.2640**	**−0.4282**	**0.3854**
Sweetened fruit drinks	−0.0125	0.1342	−0.0314	**0.5754**
Artificially sweetened beverages	0.0390	−0.1109	**0.5150**	0.1179
Artificially sweetened tea	−0.0137	0.0421	**0.4164**	0.1295
Tea	−0.0396	−0.0754	−0.1817	−0.1231
Coffee	0.0386	**−0.2058**	0.1046	0.0619
Water	0.0300	0.0141	0.0706	**0.6635**
Beer	**0.4907**	−0.0528	−0.1590	−0.0021
Liquor	**0.6314**	−0.0253	0.0086	−0.0005
Wine	**0.5546**	0.0377	0.1142	0.0114

[a]Factor loadings for each individual beverage in each beverage pattern represent the correlation of each individual beverage with each overall component (beverage pattern). Bolding denotes factor loadings >0.2 and <−0.2.
[b]Component 1 is the alcohol beverage pattern. Component 2 is the fruit and vegetable juice beverage pattern. Component 3 is the diet (reduced-fat and artificially sweetened) beverage pattern. Component 4 is the sugar-sweetened beverage pattern.

Statistical Analyses

We reported baseline characteristics using descriptive statistics for the overall study population and according to tertile of sugar-sweetened beverage pattern. Prospective analyses were conducted to assess the association between beverage patterns and incident CKD, as well as individual types of beverages consumed and incident CKD. Logistic regression was used to estimate odds ratios (ORs) and 95% confidence intervals (95% CIs), given the interval censoring for the assessment of incident CKD status at study visit 3. We also calculated adjusted risk differences and corresponding 95% CIs. Covariates included in the multivariable regression models were demographics (age, sex), socioeconomic status (education), anthropometrics (BMI), health behaviors (smoking, physical activity), comorbid conditions (hypertension, diabetes, cardiovascular disease), health status indicators (LDL cholesterol, HDL cholesterol, eGFR), and total energy intake. We adjusted for total energy intake to account for extraneous variation introduced by the food frequency questionnaire (19). In a separate model, we additionally adjusted for a healthy dietary pattern and a Southern dietary pattern that were derived using PCA to determine whether the beverages were associated with incident CKD independent of the overall dietary patterns. We examined potential effect modification using tests of interaction and stratification by baseline kidney function (eGFR <90 versus ≥90 ml/min per 1.73 m^2), age category (<50 versus ≥50 years of age), sex, overweight/obese status (BMI <25, 25–<30, ≥30 kg/m^2), diabetes status, and hypertension status. Stata statistical software version 14.2 was used for the analysis (StataCorp LLC, College Station, TX).

Results

Four distinct components or beverage patterns were derived in this study population (Table 1). For component 1, the highest factor loadings were observed for liquor (0.63), wine (0.55), and beer (0.49), meaning that these beverages were the most strongly positively correlated with the first component, which was labeled as the alcohol beverage pattern. For component 2, there were high positive factor loadings for citrus juice (0.58), other fruit juice (0.62), and vegetable juice (0.29), *i.e.*, the fruit and vegetable juice beverage pattern. There were high positive factor loadings for artificially sweetened beverages (0.52), artificially sweetened tea (0.42), and reduced-fat milk (0.39), and negative factor loadings for regular soda (−0.43) and whole milk (−0.31) for component 3, *i.e.*, the reduced-fat and artificially sweetened beverage pattern. The last component consisted of sweetened fruit drinks (0.58), soda (0.39), and water (0.66), *i.e.*, the sugar-sweetened beverage pattern.

At baseline, mean (SD) age was 54 (12) years, 64% were women, and mean eGFR was 98 (SD 18) ml/min per 1.73 m^2 in the analytic study population of 3003 participants (Table 2). Those who had higher scores (representing greater adherence) to the sugar-sweetened beverage pattern were more likely to be women and had lower levels of education. Those who followed a sugar-sweetened beverage pattern were also more likely to have hypertension and higher levels of HDL cholesterol. Participants were generally similar with respect to BMI, smoking status, physical activity, diabetes, history of cardiovascular disease, and eGFR across tertiles of the sugar-sweetened beverage pattern.

Among 3003 participants, 185 (6%) developed incident CKD over a median follow-up of 8 years. The alcohol beverage pattern, the fruit and vegetable juice beverage pattern, and the reduced-fat and artificially sweetened beverage pattern were not significantly associated with incident CKD in unadjusted models or models adjusted for total energy intake, age, sex, education, BMI, smoking status, physical activity index, hypertension, diabetes, HDL

Table 2. Baseline characteristics according to tertile of sugar-sweetened beverage pattern

Characteristic	Tertile 1, *n*=1001	Tertile 2, *n*=1001	Tertile 3, *n*=1001	Overall, *n*=3003
Age, yr	53 (12)	55 (11)	54 (12)	54 (12)
Men, *n* (%)	414 (41)	342 (34)	330 (33)	1086 (36)
Education level, *n* (%)				
<High school	133 (13)	139 (14)	176 (18)	448 (15)
High school/GED	162 (16)	176 (18)	197 (20)	535 (18)
>High school	706 (71)	686 (69)	628 (63)	2020 (67)
BMI, kg/m^2	31.7 (7.3)	31.5 (6.4)	32.2 (7.7)	31.8 (7.1)
Smoking, *n* (%)	105 (11)	101 (10)	115 (12)	321 (11)
Physical activity index	2.1 (0.8)	2.1 (0.8)	2.1 (0.8)	2.1 (0.8)
Hypertension, *n* (%)	448 (45)	534 (53)	559 (56)	1541 (51)
Diabetes, *n* (%)	142 (14)	168 (17)	166 (17)	476 (16)
HDL cholesterol, mg/dl	51 (14)	52 (14)	53 (15)	52 (14)
LDL cholesterol, mg/dl	126 (36)	129 (36)	125 (35)	127 (36)
History of cardiovascular disease, *n* (%)	66 (7)	66 (7)	75 (8)	207 (7)
eGFR, ml/min per 1.73 m^2	99 (18)	97 (17)	98 (18)	98 (18)

Data presented are mean (standard deviation) for continuous variables and n (%) for categorical variables. GED, general equivalency degree; BMI, body mass index.

cholesterol, LDL cholesterol, history of cardiovascular disease, and baseline eGFR (Table 3). The sugar-sweetened beverage pattern was associated with greater odds of CKD in both unadjusted (OR tertile 3 versus 1 =1.80; 95% CI, 1.24 to 2.62) and adjusted models (adjusted OR tertile 3 versus 1 =1.61; 95% CI, 1.07 to 2.41). The association between the sugar-sweetened beverage pattern and incident CKD was also statistically significant when analyzed continuously (model 2 adjusted OR, 1.19; 95% CI, 1.05 to 1.35). There was a small but statistically significant difference in risk of incident CKD of 2.5% for those in the highest versus lowest tertile of the sugar-sweetened beverage pattern (model 2 adjusted risk difference, 0.03; 95% CI, 0.00 to 0.05) (Table 4). After additionally adjusting for a healthy dietary pattern and a Southern dietary pattern, the results were attenuated but remained statistically significant in the continuous analysis (model 3 adjusted OR, 1.18; 95% CI, 1.00 to 1.39), but not when analyzed according to tertiles (model 3 OR tertile 3 versus 1, 1.37; 95% CI, 0.86 to 2.16) (Table 3).

There was no statistical evidence of interaction by kidney function (eGFR 60 to <90 versus ≥90 ml/min per 1.73 m^2; *P*-interaction =0.39), age (<50 versus ≥50 years; *P*-interaction =0.12), sex (*P*-interaction =0.39), BMI (<25 versus 25 to <30 versus ≥30 kg/m^2; *P*-interaction =0.89), diabetes status (*P*-interaction =0.25), or hypertension status (*P*-interaction =0.89) (Supplemental Table 1). Higher adherence to the sugar-sweetened beverages was associated with greater odds of CKD among participants <50 years of age (adjusted OR tertile 3 versus 1, 5.11; 95% CI, 1.36 to 19.3; adjusted OR per one unit higher, 1.39; 95% CI, 1.11 to 1.74), but the association was not statistically significant among older participants (age ≥50 years).

For individual types of beverages, higher intake of soda (continuous adjusted OR, 1.09; 95% CI, 1.00 to 1.18) was associated with greater odds of CKD after adjusting for total energy intake, age, sex, education, BMI, smoking status, physical activity index, hypertension, diabetes, HDL cholesterol, LDL cholesterol, history of cardiovascular disease, and baseline eGFR (Supplemental Table 2). When analyzed by tertiles, higher intake of tea and beer were also associated with greater odds of CKD. No other individual types of beverages were associated with CKD.

Discussion

In this community-based United States cohort of 3003 black men and women with preserved kidney function at baseline (eGFR≥60 ml/min per 1.73 m^2), we found that consuming a sugar-sweetened beverage pattern was associated with greater odds of developing CKD. This association remained in the linear model even after adjusting for total dietary patterns, although the relationship was attenuated. Importantly, the dietary patterns themselves included beverages, so overadjustment is likely. We detected three other prevalent beverage patterns including alcoholic beverages, fruit and vegetable juices, and diet (reduced-fat and artificially sweetened) beverages, but none of these were associated with kidney disease.

There has been a plethora of research demonstrating the adverse cardiometabolic consequences of consuming sugar-sweetened beverages, including sweetened fruit drinks and regular (not artificially sweetened) soda (4). Accordingly, dietary guidelines have made recommendations for selected types of beverages, such as soda, as well as other individual types of beverages, such as coffee and alcohol (3,28). However, less attention has been focused on beverage patterns, despite the emphasis of the dietary guidelines on patterns of dietary intake reflecting human behavior. To the best of our knowledge, this study is the first to empirically derive patterns of beverage consumption in relation to CKD.

Previous epidemiologic and basic science research has established the link between sugar-sweetened beverages and health outcomes as well as the biologic mechanism underlying this relationship. Sugar-sweetened beverages contain added sugars and associated energy that, when consumed regularly over an extended period of time, can lead to positive energy balance, resulting in weight gain and the development of obesity (29). Obesity, in turn, is a risk factor for diabetes, cardiovascular disease, and CKD (30–32). Higher intake of sugar-sweetened beverages has

Table 3. Odds ratios (95% confidence intervals) for beverage patterns and incident CKD

Beverage Pattern	Model[a]	Categorical Analysis			Continuous Analysis
		Tertile 1	Tertile 2	Tertile 3	
Alcohol	1	1 [Ref]	1.29 (0.90 to 1.85)	1.04 (0.71 to 1.51)	0.96 (0.83 to 1.11)
	2	1 [Ref]	1.20 (0.68 to 1.74)	1.09 (0.68 to 1.74)	1.01 (0.86 to 1.18)
	3	1 [Ref]	1.22 (0.76 to 1.97)	1.38 (0.83 to 2.30)	1.12 (0.95 to 1.35)
Fruit and vegetable juice	1	1 [Ref]	1.36 (0.93 to 1.97)	1.31 (0.90 to 1.92)	1.01 (0.89 to 1.15)
	2	1 [Ref]	1.10 (0.73 to 1.65)	1.05 (0.70 to 1.58)	0.97 (0.83 to 1.13)
	3	1 [Ref]	1.26 (0.79 to 2.00)	1.19 (0.75 to 1.90)	1.01 (0.84 to 1.20)
Diet beverages	1	1 [Ref]	0.82 (0.57 to 1.19)	0.97 (0.67 to 1.38)	1.04 (0.92 to 1.18)
	2	1 [Ref]	0.82 (0.55 to 1.22)	0.74 (0.50 to 1.09)	0.95 (0.82 to 1.10)
	3	1 [Ref]	0.89 (0.57 to 1.39)	0.80 (0.51 to 1.25)	0.95 (0.79 to 1.14)
Sugar-sweetened beverage	1	1 [Ref]	1.30 (0.88 to 1.93)	**1.80 (1.24 to 2.62)**	**1.20 (1.07 to 1.34)**
	2	1 [Ref]	1.18 (0.77 to 1.82)	**1.61 (1.07 to 2.41)**	**1.19 (1.05 to 1.35)**
	3	1 [Ref]	1.19 (0.75 to 1.91)	1.37 (0.86 to 2.16)	**1.18 (1.00 to 1.39)**

Bolding denotes statistically significant results. Ref, reference value.
[a]Model 1 was unadjusted. Model 2 was adjusted for total energy intake, age, sex, education, body mass index, smoking status, physical activity index, hypertension, diabetes, HDL cholesterol, LDL cholesterol, history of cardiovascular disease, and baseline eGFR. Model 3 was adjusted for all of the covariates in model 2 plus scores for a healthy dietary pattern and a Southern dietary pattern.

also been shown to be directly associated with the incidence and prevalence of kidney disease (6,7). For kidney disease specifically, fructose, which is found at high concentrations in sugar-sweetened beverages, can increase serum concentrations of urate and lead to the development of kidney disease through renin production, vascular disease, and interstitial fibrosis (33–35). Fruit juices have some similar characteristics to sugar-sweetened beverages in terms of their sugar content (albeit natural rather than added), energy content, and associated health outcomes. However, in this study, citrus juice and other fruit juices were not significantly associated with risk of kidney disease. The high amount of vitamin C relative to fructose in 100% fruit juice may mitigate the serum urate raising effect (34). In addition, 100% fruit juice is a rich source of both vitamin C and potassium, which have consistently been shown to reduce BP, thereby perhaps counteracting the negative health outcomes associated with other nutritional aspects of fruit juice (36,37).

We found that younger participants, *i.e.*, those <50 years of age, with higher levels of adherence to the sugar-sweetened beverage pattern tended to have an elevated risk of CKD, although there was no evidence of statistical interaction (*P*-interaction =0.12). Younger persons may be particularly vulnerable to the harmful consequences of consuming sugar-sweetened beverages. Public health interventions to reduce sugar-sweetened beverage intake may be most effective if targeted toward younger adults.

Our finding that water had a positive factor loading on the sugar-sweetened beverage pattern that was associated with greater odds of CKD was unexpected. We hypothesized that there would be an inverse association between water consumption and risk of kidney disease. There are some studies demonstrating that higher water intake is associated with slower CKD progression (38,39). For example, in a community-based cohort study among individuals in Canada, higher urine volume as a proxy for fluid intake relative to lower urine volume was associated with slower kidney function decline over a 6-year period (40). However, in the CKD Water Intake Trial, which was a randomized, clinical trial conducted among participants with stage 3 CKD, increasing water intake did not slow kidney function decline over 1 year (41). Although water is recommended for various health benefits, there is limited evidence in support of such a recommendation for the general population and there are somewhat inconsistent results for water intake and CKD (42,43).

A possible reason for this surprising finding for water is that study participants in the JHS may have reported their consumption of a wide variety of types of water, including flavored and sweetened water, which may be consumed frequently by the general population in warm climates like Mississippi. There are a number of different types of water which have a "health halo" (*e.g.*, vitamin water), which are advertised as being healthy but have not been proven to have health benefits (44,45). Unfortunately, we did not collect information about specific brands or types of bottled water in the JHS. Future studies should consider collecting additional information on types of water, either by adding more detailed questions to a food frequency questionnaire or by using less-structured approaches to dietary assessment, *e.g.*, diet records and dietary recalls.

In our study, we observed that associations were stronger for the sugar-sweetened beverage pattern than for the individual beverages represented within that beverage pattern. In a cross-sectional analysis of National Health and Nutrition Examination Survey (NHANES) data, low versus high intake of plain water was associated with greater odds of having CKD, but this association was not evident for other beverages (46). These results from NHANES and our study findings underscore the importance of taking a holistic approach to characterize the overall pattern of beverage consumption as we did in this study. In addition, individuals do not typically consume a single type of beverage. As such, pattern analysis is more appropriate for capturing this diet behavior of consuming a

Table 4. Risk differences (95% confidence intervals) for beverage patterns and incident CKD

Beverage Pattern	Model[a]	Categorical Analysis			Continuous Analysis
		Tertile 1	Tertile 2	Tertile 3	
Alcohol	1	1 [Ref]	0.0150 (−0.0064 to 0.0363)	0.0020 (−0.0183 to 0.0223)	−0.0025 (−0.0106 to 0.0055)
	2	1 [Ref]	0.0096 (−0.0124 to 0.0316)	0.0042 (−0.0191 to 0.0275)	0.0004 (−0.0078 to 0.0086)
	3	1 [Ref]	0.0097 (−0.0133 to 0.0328)	0.0165 (−0.0093 to 0.0423)	0.0065 (−0.0034 to 0.0163)
Fruit and vegetable juice	1	1 [Ref]	0.0170 (−0.0037 to 0.0377)	0.0150 (−0.0056 to 0.0355)	0.0008 (−0.0066 to 0.0082)
	2	1 [Ref]	0.0047 (−0.0162 to 0.0256)	0.0026 (−0.0179 to 0.0231)	−0.0015 (−0.0092 to 0.0063)
	3	1 [Ref]	0.0114 (−0.0117 to 0.0345)	0.0086 (−0.0140 to 0.0313)	0.0004 (−0.0088 to 0.0095)
Diet beverages	1	1 [Ref]	−0.0110 (−0.0319 to 0.0099)	−0.0020 (−0.0236 to 0.0196)	0.0024 (−0.0051 to 0.0099)
	2	1 [Ref]	−0.0107 (−0.0323 to 0.0109)	−0.0158 (−0.0365 to 0.0048)	−0.0027 (−0.0099 to 0.0046)
	3	1 [Ref]	−0.0061 (−0.0299 to 0.0176)	−0.0117 (−0.0350 to 0.0116)	−0.0027 (−0.0117 to 0.0063)
Sugar-sweetened beverage	1	1 [Ref]	0.0130 (−0.0065 to 0.0325)	0.0340 (0.0127 to 0.0552)[b]	0.0111 (0.0039 to 0.0182)[b]
	2	1 [Ref]	0.0077 (−0.0121 to 0.0276)	0.0246 (0.0041 to 0.0450)[b]	0.0095 (0.0024 to 0.0166)[b]
	3	1 [Ref]	0.0085 (−0.0139 to 0.0309)	0.0158 (−0.0072 to 0.0387)	0.0090 (−0.0002 to 0.0183)

Ref, reference value.
[a]Model 1 was unadjusted. Model 2 was adjusted for total energy intake, age, sex, education, body mass index, smoking status, physical activity index, hypertension, diabetes, HDL cholesterol, LDL cholesterol, history of cardiovascular disease, and baseline eGFR. Model 3 was adjusted for all of the covariates in model 2 plus scores for a healthy dietary pattern and a Southern dietary pattern.
[b]Denotes statistically significant results.

few different types of beverages, analogous to dietary patterns in which a wide variety of nutrients and foods are eaten in combination (23,47).

There are a few strengths and limitations that deserve mention. A major strength was that the study population consisted of black men and women residing in a southern region of the United States, a population that experiences a greater burden of kidney disease compared with other racial/ethnic populations and has remained understudied (11,12). It has also previously been shown that there is a distinct dietary pattern consumed in the South, which provides further justification for assessing beverage patterns in the JHS (48). That said, our study findings may not be generalizable to other racial/ethnic populations and other regions of the United States, so it would be worthwhile to investigate beverage patterns in association with health outcomes in other cohorts. Another strength is the prospective study design that allows for us to establish temporality between baseline beverage consumption and ascertainment of 185 incident CKD cases over a median follow-up of 8 years. In addition, detailed information was collected *via* examination and structured questionnaires administered by trained personnel at the study visits, which allowed us to adjust for multiple potential confounding variables, including baseline eGFR, in multivariable regression models. However, there is the possibility of residual confounding from unmeasured or imperfectly measured covariates, which could partly explain the observed associations. Beverage consumption was assessed by self-report using an interviewer-administered food frequency questionnaire which is affected by recall bias and measurement error (49). In general, food frequency questionnaires may not adequately capture sweeteners and milk that are added to such beverages as coffee and tea, although our food frequency questionnaire included separate questions about these items which were considered in our analysis. In addition, the food frequency questionnaire was administered at one time point, *i.e.*, baseline, which does not reflect any changes in beverage consumption that occurred during the follow-up period. Beverage consumption is considered to be relatively stable over time. For example, in the Atherosclerosis Risk in Communities study, the majority (69%) of participants either did not change their frequency of coffee consumption or changed by one category, *e.g.*, from one cup per day to two to three cups per day, over a 6-year period (50).

In conclusion, following a pattern of beverage consumption consisting of sweetened fruit drinks, soda, and water was associated with greater odds of developing CKD in this community-based cohort of black adults in Jackson, Mississippi. These results contribute to the growing body of literature elucidating the negative health consequences of consuming sugar-sweetened beverages. Additional research is needed to understand the healthfulness of different types of bottled water, including flavored and sweetened water. Patterns of beverage consumption may better reflect this dietary behavior and be more informative when studying their relationship with disease outcomes than individual beverages.

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Disclosures

None.

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See related Patient Voice, "Diet and Risk for Developing Kidney Disease," and editorial, "The Millennial Physician and the Obesity Epidemic: A Tale of Sugar-Sweetened Beverages," on pages 1–2 and 4–6, respectively.

Patterns of Beverages Consumed and Risk of Incident Kidney Disease: Results from the Jackson Heart Study

Casey M. Rebholz, Bessie A. Young, Ronit Katz, Katherine L. Tucker, Teresa C. Carithers, Arnita F. Norwood, Adolfo Correa

Supplemental Table 1. Adjusted[a] Odds Ratios for the Association between Sugar-Sweetened Beverage Pattern and Incident CKD According to Kidney Function, Age, Sex, Obesity Status, Diabetes Status, and Hypertension Status

	Categorical Analysis				**Continuous Analysis**		**P for interaction**
Subgroup	**Tertile 1**	**Tertile 2**	**Tertile 3**	**P-value**	**OR (95% CI)**	**P-value**	
eGFR 60-<90 mL/min/1.73 m^2	1 [Ref]	0.99 (0.57, 1.70)	1.55 (0.95, 2.55)	0.06	**1.29 (1.08, 1.53)**	**0.004**	0.39
eGFR ≥ 90 mL/min/1.73 m^2	1 [Ref]	1.83 (0.88, 3.78)	1.81 (0.89, 3.70)	0.13	1.08 (0.88, 1.33)	0.47	
Age <50 years	1 [Ref]	0.99 (0.15, 6.44)	**5.11 (1.36, 19.3)**	**0.007**	**1.39 (1.11, 1.74)**	**0.005**	0.12
Age ≥50 years	1 [Ref]	1.16 (0.74, 1.82)	1.36 (0.88, 2.10)	0.16	1.10 (0.94, 1.28)	0.22	
Men	1 [Ref]	0.93 (0.47, 1.87)	1.23 (0.66, 2.32)	0.51	1.11 (0.90, 1.35)	0.33	0.39
Women	1 [Ref]	1.36 (0.77, 2.41)	**1.90 (1.10, 3.29)**	**0.02**	**1.25 (1.06, 1.48)**	**0.009**	
BMI <25 kg/m^2	1 [Ref]	0.90 (0.19, 4.31)	1.26 (0.34, 4.68)	0.70	1.23 (0.90, 1.68)	0.19	0.89
BMI 25-<30 kg/m^2	1 [Ref]	2.10 (0.89, 4.96)	**3.13 (1.38, 7.06)**	**0.006**	1.14 (0.88, 1.48)	0.33	
BMI ≥30 kg/m^2	1 [Ref]	1.07 (0.62, 1.86)	1.30 (0.77, 2.20)	0.31	**1.20 (1.01, 1.42)**	**0.04**	
Diabetes	1 [Ref]	0.93 (0.43, 2.04)	1.96 (0.97, 3.97)	**0.03**	**1.36 (1.08, 1.70)**	**0.01**	0.25
No diabetes	1 [Ref]	1.31 (0.78, 2.21)	1.45 (0.88, 2.40)	0.15	1.13 (0.96, 1.32)	0.14	
Hypertension	1 [Ref]	0.94 (0.58, 1.54)	1.43 (0.92, 2.24)	0.08	**1.19 (1.03, 1.39)**	**0.02**	0.89
No hypertension	1 [Ref]	**3.37 (1.21, 9.40)**	2.69 (0.97, 7.49)	0.07	1.22 (0.94, 1.59)	0.13	

[a] Multivariable regression models were adjusted for total energy intake, age, sex, income status, body mass index, smoking status, physical activity index, hypertension, diabetes, high-density lipoprotein cholesterol, history of cardiovascular disease, and baseline estimated glomerular filtration rate (Model 2). Bold font denotes statistically significant results.

Supplemental Table 2. Adjusted[a] Odds Ratios (95% CI) for Individual Beverages and Incident CKD

	Categorical Analysis				Continuous Analysis	
Beverage	**Tertile 1**	**Tertile 2**	**Tertile 3**	**P-value**	**OR (95% CI)**	**P-value**
Citrus juice	1 [Ref]	0.72 (0.47, 1.10)	0.84 (0.57, 1.24)	0.44	0.96 (0.84, 1.11)	0.60
Other fruit juice	1 [Ref]	1.10 (0.70, 1.74)	1.24 (0.83, 1.85)	0.29	1.05 (0.83, 1.33)	0.68
Vegetable juice	1 [Ref]	0.78 (0.50, 1.23)	0.73 (0.46, 1.14)	0.19	0.53 (0.22, 1.31)	0.17
Whole milk	1 [Ref]	1.42 (0.87, 2.30)	1.53 (0.95, 2.47)	0.11	1.16 (0.88, 1.54)	0.30
Reduced-fat milk	1 [Ref]	0.85 (0.53, 1.36)	0.80 (0.51, 1.26)	0.35	0.84 (0.60, 1.18)	0.31
Low-fat milk	1 [Ref]	0.72 (0.41, 1.26)	0.58 (0.29, 1.17)	0.14	0.72 (0.43, 1.20)	0.20
Soda	1 [Ref]	1.07 (0.69, 1.67)	1.16 (0.76, 1.75)	0.49	**1.09 (1.00, 1.18)**	**0.05**
Sweetened fruit drinks	1 [Ref]	1.27 (0.82, 1.97)	1.25 (0.82, 1.91)	0.34	1.07 (0.96, 1.19)	0.23
Artificially-sweetened beverages	1 [Ref]	0.98 (0.63, 1.54)	1.08 (0.67, 1.72)	0.73	1.07 (0.95, 1.20)	0.26
Artificially-sweetened tea	1 [Ref]	0.93 (0.59, 1.47)	0.72 (0.45, 1.15)	0.15	1.16 (0.91, 1.48)	0.23
Tea	1 [Ref]	1.48 (0.91, 2.39)	**1.65 (1.05, 2.59)**	**0.04**	0.98 (0.75, 1.29)	0.89
Coffee	1 [Ref]	0.70 (0.47, 1.06)	0.72 (0.48, 1.08)	0.14	0.92 (0.83, 1.03)	0.17
Water	1 [Ref]	1.45 (0.95, 2.23)	1.51 (0.98, 2.31)	0.07	1.02 (1.00, 1.04)	0.08
Beer	1 [Ref]	1.52 (0.92, 2.51)	**1.81 (1.04, 3.15)**	0.05	1.07 (0.83, 1.38)	0.60
Liquor	1 [Ref]	1.10 (0.66, 1.81)	1.16 (0.65, 2.05)	0.63	0.91 (0.61, 1.37)	0.66
Wine	1 [Ref]	1.27 (0.77, 2.08)	1.07 (0.63, 1.83)	0.99	1.40 (0.49, 3.99)	0.52

[a] Multivariable regression models were adjusted for total energy intake, age, sex, income status, body mass index, smoking status, physical activity index, hypertension, diabetes, high-density lipoprotein cholesterol, history of cardiovascular disease, and baseline estimated glomerular filtration rate (Model 2). Bold font denotes statistically significant results.