Associations of Sugar and Artificially Sweetened Soda with Albuminuria and Kidney Function Decline in Women

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Summary

Background and objectives Sugar-sweetened soda is reported to be associated with increased risk for diabetes and albuminuria, but there are currently limited data on how sugar or artificially sweetened soda may be related to kidney function decline.

Design, setting, participants, & measurements This study identified 3318 women participating in the Nurses' Health Study with data on soda intake and albuminuria; of these, 3256 also had data on estimated GFR (eGFR) change between 1989 and 2000. Cumulative average beverage intake was derived from the 1984, 1986, 1990, 1994, and 1998 food frequency questionnaires. Serving categories included <1/mo (referent), 1 to 4/mo, 2 to 6/wk, 1 to 1.9/d, and ≥2/d. Microalbuminuria (MA) was considered a urinary albumin-to-creatinine ratio of 25 to 355 μg/mg. For kidney function change, the primary outcome was a ≥30% decline in eGFR over 11 years; rapid eGFR decline defined as ≥3 ml/min per 1.73 m^2 per year was also examined.

Results Consumption of ≥2 servings per day of artificially sweetened (diet) soda was independently associated with eGFR decline ≥30% (OR 2.02, 95% CI 1.36 to 3.01) and ≥3 ml/min per 1.73 m^2 per year (OR 2.20, 95% CI 1.36 to 3.55). No increased risk for eGFR decline was observed for <2 servings per day of diet soda. No associations were noted between diet soda and MA or sugar soda and MA or eGFR decline.

Conclusions Consumption of ≥2 servings per day of artificially sweetened soda is associated with a 2-fold increased odds for kidney function decline in women.

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Introduction

Higher consumption of sugar-sweetened beverages, including carbonated soft drinks, has been linked to increased risk for obesity (1) and type 2 diabetes (2,3), two factors that are independent predictors of progressive kidney function decline (4). In 2008, a cross-sectional analysis of over 9000 participants in the National Health and Nutrition Examination Survey (NHANES) 1999 to 2004 reported a significant association between microalbuminuria (MA), widely considered an early marker of early kidney disease, and consumption of ≥2 sugar soft drinks per day (adjusted odds ratio [OR] 1.40, 95% confidence interval [CI] 1.13 to 1.74) (5). No association between diet soda and albuminuria was noted. Although MA is a strong predictor of subsequent kidney function decline, no data on change in estimated GFR (eGFR) change were available in that cross-sectional study.

In the study presented here, we investigated the association between sugar soda and artificially sweetened soda with albuminuria and kidney function decline in over 3000 female participants of the Nurses' Health Study (NHS). Specifically, we studied associations between soda consumption and MA like the NHANES study while also expanding the scope to examine associations of sugar and diet soda consumption with eGFR decline. Epidemiology studies of carbonated beverages usually examine sugar-sweetened and artificially sweetened sodas separately; these include previous peer-reviewed publications of the NHS and coronary heart disease risk (6), NHS II and incident diabetes (3) or gestational diabetes (7), as well as the soda and albuminuria analysis in NHANES III (5). Therefore, we *a priori* designed the study to look at sugar-sweetened and diet sodas separately to be consistent with the previously published literature.

Materials and Methods

Participants

The NHS was initiated in 1976 with the enrollment of 121,700 U.S. nurses aged 30 to 55 years. This cohort is followed through biennial mailed questionnaires related to lifestyle factors and health outcomes. Between 1989 and 1990, 32,826 participants provided blood samples that were shipped overnight and stored at −130°C (8). In the year 2000, 18,720 of these participants submitted a second blood sample and spot urine specimens under the same handling and storage conditions. Participants who did and did not

return blood samples were similar in terms of demographics and lifestyle characteristics.

Participants for the study presented here were from a substudy of analgesic use and renal function (9) or a substudy of type 2 diabetes and kidney function who had plasma creatinine measured from 1989 and 2000 collections and urinary albumin-to-creatinine ratio (ACR) measured from the 2000 collection. The women in the analgesic study ($n = 3876$) had returned supplemental questionnaires regarding lifetime analgesic use. Among those who returned the analgesic questionnaire, 2712 women had plasma creatinine and urinary ACR data; we also included 730 women from the diabetes substudy. From this combined group ($n = 3442$), we included those with cumulative average sweetened beverage intake and urine ACR data in the year 2000 ($n = 3318$). The majority ($n = 3256$) also had plasma creatinine measured in samples collected in 1989 and 2000.

Assessment of Sugar and Artificially Sweetened Soda Intake

The semiquantitative food frequency questionnaires (FFQs) were designed to assess average food intake of more than 130 foods and beverages over the preceding year (10). A standard portion size and nine possible frequency-of-consumption responses, ranging from "never or less than once per month" to "6 or more times per day" were given for each food item. Total energy and nutrient intake were calculated from the reported frequency of consumption of each specified unit of food or beverage and from published data on the nutrient content of the specified portions (10). Previous validation studies in the NHS cohort comparing FFQ data to multiple weeks of food records revealed good correlations for sweetened beverages. For example, correlations between diet records and FFQs were 0.84 for colas and 0.36 for noncola carbonated soft drinks (11). Although this correlation coefficient of 0.84 for carbonated colas did not distinguish between sugar-sweetened and diet beverages in the NHS cohort, the correlation for diet soft drinks is 0.74 (with an identical $r = 0.84$ for sugar-sweetened colas) in the Health Professionals Follow-Up study (12), which used the same FFQ.

Participants were asked to report the number of servings ("one glass, bottle or can") consumed on average over the past year for regular carbonated beverages (Coke, Pepsi, other cola, or other carbonated beverages with sugar) and low-calorie sugar-free carbonated beverages with or without caffeine.

Measurement of Urinary ACR

Urinary assays were performed on spot collections submitted in the year 2000; urinary creatinine concentration was measured by a modified Jaffe method (coefficient of variation [CV] 1.6%). Urinary albumin was measured using solid-phase fluorescence immunoassay using the Hitachi 911 analyzer and Roche diagnostics reagents (Indianapolis, IN) with a lower limit of detection of 0.1 mg/L (CV 8.0%). A urinary ACR of 25 to 355 μg/mg was used to define MA; this sex-specific cutpoint for women has been reported to approximate a urinary albumin excretion rate of 30 to 300 mg/24 h (13), which is traditionally considered clinically relevant MA. In this study, 205 women (6.1%) met the criterion for MA. There were 36 women with macroalbuminuria (ACR >355 μg/mg) who were excluded from the albuminuria analyses.

Measurement of Kidney Function Decline

Plasma creatinine was analyzed using a modified kinetic Jaffe reaction (CV 10%). In 2007, repeat measurement of 20 NHS plasma samples collected in 1989 (with a range of 0.6 to 1.4 mg/dl) and initially measured in the year 2000 revealed a mean recalibration coefficient (new value/original value) of 0.97 and confirmed that plasma creatinine is stable for many years under our storage conditions.

Glomerular filtration was estimated by the four-variable Modification of Diet in Renal Disease equation in which eGFR (ml/min per 1.73 m^2) = $186 \times [\textit{plasma creatinine}\ (\text{mg/dl})]^{-1.154} \times [\textit{age}]^{-0.203} \times [0.472\ \textit{if female}] \times [1.21\ \textit{if black}]$ (14). An eGFR decline of ≥30% between 1989 and 2000 was considered to be a clinically significant change and has been used in previous analyses of renal function decline in NHS participants (15). We also examined "rapid" eGFR decline defined as ≥3 ml/min per 1.73 m^2 per year, which has been previously used as a cutoff that reflects 3 times more rapid decline than expected by normal aging (16). eGFR by the Chronic Kidney Disease–Epidemiology Collaboration (CKD-EPI) equation (17) was also analyzed.

Assessment of Covariates

Race and height were initially reported on the 1992 questionnaire. Other self-reported clinical and lifestyle variables including weight, hypertension (HTN), smoking status, physical activity, cardiovascular disease (angina, myocardial infarction, coronary artery bypass surgery, percutaneous coronary revascularization, or stroke), and blood pressure (BP) medication use were reported on the biennial questionnaires. Questionnaire data collected closest to the year when kidney function was measured (the 1988 questionnaire for eGFR decline and the 2000 questionnaire for urinary ACR) were used (Figure 1). In addition, we obtained self-reported BP from the 1990 questionnaire.

Systolic BP was reported in nine categories (<105, 105 to 114, 115 to 124, 125 to 134, 135 to 144, 145 to 154, 155 to 164, 165 to 174, and >175 mmHg), and diastolic BP was reported in seven categories (<65, 65 to 74, 75 to 84, 85 to 89, 90 to 94, 95 to 104, and >105 mmHg). A participant's BP was defined as the middle systolic and middle diastolic value of the reported category. Many of these variables have been previously validated through direct medical record review (18,19).

We mailed a supplementary questionnaire in 1999 to collect detailed information on the current use of each of the three analgesic medication classes (aspirin,

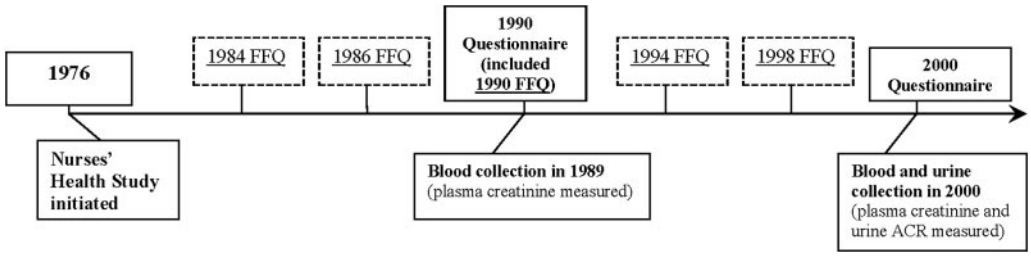

Figure 1. | Time line of questionnaire and biological sample data collection in NHS for these analyses. Questionnaires are administered every 2 years beginning in 1976 but only questionnaire data for nondietary covariates from years used for this study (1990 and 2000) are shown. FFQs asking about diet over the previous 12 months were administered in 1984, 1986, 1990, 1994, and 1998.

nonsteroidal anti-inflammatory drugs [NSAIDS], and acetaminophen), including frequency in days per months, tablets per day, tablet dosage, brand, and indication for current use. The questionnaire also asked about total consumption in two periods—the past 10 years and before 1990. The total number of tablets taken in those two periods was collected in 11 categories: none, 1 to 100, 101 to 500, 501 to 1000, 1001 to 1500, 1501 to 3000, 3001 to 5000, 5001 to 10,000, 10,001 to 15,000, 15,001 to 20,000, and ≥20,001. We used the combined total from the two periods by adding the midpoints of the categories. We converted number of tablets to lifetime intake (in grams) by multiplying the total number of tablets for the midpoint of each category by the most common dosage of each analgesic (aspirin and acetaminophen = 325 mg; NSAIDs = 200 mg) (15).

Participants reported new physician-diagnosed diseases, including diabetes, on the biennial questionnaires. We mailed a diabetes supplementary questionnaire to all women reporting diabetes to obtain further information about the date of diagnosis, symptoms, diagnostic tests, and treatment. We used the National Diabetes Data Group criteria to define diabetes self-reported up to the 1996 biennial questionnaire (20); the American Diabetes Association diagnostic criteria for diabetes released in 1997 were used for incident cases of diabetes reported in 1998 and after (21). Self-reported diagnosis of type 2 diabetes using the diabetes supplementary questionnaire has been established as 98% accurate in a separate validation study through medical record review (22). Because the onset of abnormalities in glucose handling can precede the diagnosis of type 2 diabetes by 10 years or more (23,24), we considered a participant who was diagnosed with diabetes up through the year 2000 as having diabetes.

Because we have previously reported significant associations between higher red meat intake and MA, as well as associations between dietary sodium, β-carotene, vitamin E, and lowfat dairy and eGFR decline (25), we performed additional analyses adjusting for these factors. Because higher sugar or artificially sweetened soda intake may be a marker of a generally unhealthy diet, we also considered models that included adjustment for overall diet quality as assessed by the Alternate Healthy Eating Index (AHEI). Briefly, the AHEI scoring (26) was based on intake levels of nine components (27): fruits, vegetables, the ratio of white (seafood and poultry) to red meat, trans fat, the ratio of polyunsaturated to saturated fat, cereal fiber, nuts and soy, moderate alcohol consumption (0.5 to 1.5 servings/d), and long-term multivitamin use (<5 or ≥5 years). These components were chosen on the basis of their association with disease and mortality risk in observational and experimental studies.

Statistical Analyses

Wilcoxon rank sum and χ^2 tests were used to compare differences between groups. For analyses of MA, cumulative average beverage intake for each participant was calculated from FFQs returned in 1984, 1986, 1990, 1994, and 1998 as described previously (6,28). For analyses of eGFR decline, cumulative average intakes of beverages in 1984, 1986, and 1990 were used (Figure 1).

A cumulative average approach was chosen because it generally reflects long-term diet and also likely reduces measurement error from intraindividual variation over time (28). Logistic regression was used to examine associations between categories of beverage intake and presence of MA in 2000 or eGFR decline ≥30% decline or rapid eGFR decline of ≥3 ml/min per 1.73 m^2 per year between 1989 and 2000. Because only 3% reported ≥1 serving per day of sugar-sweetened soda, ≥1 serving per day was used as the highest category for sugar soda.

Multivariable models were adjusted for age, HTN, body mass index (BMI), cigarette smoking, physical activity, cardiovascular disease, and diabetes. For the outcome of MA, angiotensin-converting enzyme inhibitor (ACEI) or angiotensin-type 2 receptor blocker (ARB) medication use and eGFR in 2000 were also included. For eGFR decline models, covariate data from the 1988 questionnaire were used. All analyses were performed with SAS software, version 9.1 (SAS Institute, Inc., Cary, NC). The Partners' Healthcare Brigham and Women's Hospital Human Research Committee Institutional Review Board approved this study.

Results

Characteristics of these NHS women are summarized in Table 1. Women who reported ≥1 serving per day of sugar soda compared with <1 serving per month were younger (62 *versus* 68 years), more like to have smoked (60% *versus* 55%), have higher caloric intake (median 2025 *versus* 1643 kcal/d), and lower activity levels (median 6.7 *versus* 12.4 METS/wk); no other significant differences (including lifetime intake of analgesic medications) were noted. Similarly, compared with women with <1 serving per week of diet soda, those with ≥2 servings per week were younger (65 *versus* 69 years), had more HTN (66% *versus* 44%), were more likely to have ever smoked (61% *versus* 39%), had lower alcohol intake (median 0 *versus* 0.9 g/d) and activity level (8.3 *versus* 11.5 METS/wk). However, women with ≥2 servings per day of diet soda had higher ACEI medication use (18% *versus* 12%), higher median ACR (3.9 to 3.4 μg/mg), and higher lifetime intake of all three analgesic medications; no other significant differences were noted.

Table 1. Participants in the NHS (n = 3318)

Characteristics	NHS
Age (years)	67
Caucasian (%)	97
HTN (%)	54
Diabetes (%)	23
Cardiovascular disease (%)	6
Current smoker (%)	6
Ever smoker (%)	48
Alcohol intake (g/day)	0.9
Calorie intake (kcal/day)	1708
Activity level (METS/wk)	11.5
BMI (kg/m^2)	26.3
ACEI or ARB medication use (%)	15
Plasma creatinine in 1989 (mg/dl)	0.75
Plasma creatinine in 2000 (mg/dl)	0.81
eGFR in 1989 (ml/min per 1.73 m^2)	84
eGFR in 2000 (ml/min per 1.73 m^2)	76
Urinary ACR (μg/mg)	3.4
Sugar soda intake servings (%)	
<1 per month	58
1 to 4 per month	22
2 to 6 per week	17
≥1 per day	3
Artificially sweetened soda servings (%)	
<1 per month	27
1 to 4 per month	15
2 to 6 per week	36
1 to 1.9 per day	14
≥2 per day	8

Results expressed as median or %. Values are given from the year 2000 except where noted.

MA

In our study population, 205 women met the criteria for MA; results that also included the 36 women with macroalbuminuria were not significantly different (n = 241). No significant association between sugar soda intake and albuminuria was noted (Table 2). Although there was a significant age and energy-adjusted association between ≥2 servings a day of artificially sweetened soda and MA, this was no longer significant in the multivariable-adjusted model (Table 2); diabetes and BMI were the strongest confounders of the association between artificially sweetened sodas and MA. Results were not meaningfully different when controlled for red meat intake or for diet quality by AHEI.

eGFR Decline

There were 381 (11.5%) women with eGFR decline ≥30% over the 11 years of follow-up; this reflected in these women a median increase in plasma creatinine of 0.33 mg/dl (with medians of 0.69 mg/dl in 1989 and 1.04 mg/dl for the group). Between 1990 and 1998, there appeared to be little change in the quantity of soda intake in our study population as reported by the food questionnaires. For example, median change was zero for sugar and diet soda between 1990 and 1998 with a mean change of −0.1 servings per month for sugar soda and −1.5 servings per month for artificially sweetened soda. No significant associations were noted for sugar soda intake and eGFR decline ≥30%. Among women who consumed ≥2 servings per day of artificially sweetened soda, the adjusted odds for eGFR decline were 2-fold higher (Table 3).

All analyses were additionally adjusted for nutrients, red meat, and AHEI diet quality without meaningful change in results. Additional adjustment for multiple individual nutrients known to be associated with eGFR decline in this cohort, including sodium, β-carotene, vitamin E, and lowfat dairy, also resulted in persistent significant association between ≥2 servings per day of diet soda and eGFR decline (OR 1.97, 95% CI 1.32 to 2.96). Including weight change between 1988 and 1990 in the model did not meaningfully alter the results (OR 1.99, 95% CI 1.33 to 2.97). We also specifically examined the influence of obesity (defined as BMI ≥ 30 kg/m^2) by including an interaction term in the multivariable model; BMI did not significantly modify the association between diet soda and eGFR decline (interaction P = 0.33).

Also, when eGFR decline ≥3 ml/min per 1.73 m^2 per year was used as the primary outcome, we observed similar associations with ≥2 servings per day of artificially sweetened soda (OR 2.20, 95% CI 1.36 to 3.55). These results were also not influenced by adjustment for other nutrients or diet quality. Using the CKD-EPI equation to estimate GFR resulted in consistent but slightly attenuated results. The OR was 1.48 (95% CI 1.00 to 2.21) for the association between ≥2 servings of artificially sweetened soda and ≥30% eGFR decline.

We also examined associations for all sugar-sweet-

Table 2. OR and 95% CI for cumulative averaged soda intake and presence of MA (ACR 25 to 355 μg/mg)

Soda Intake	Number of Cases/Total	Age and Energy-Adjusted OR [95% CI]	Multivariable-Adjusted[a] OR [95% CI]
Sugar soda (servings)			
<1 per month	131/1915	1.0 (referent)	1.0 (referent)
1 to 4 per month	31/725	0.63 [0.42, 0.94]	0.69 [0.45, 1.04]
2 to 6 per week	40/558	1.11 [0.77, 1.02]	1.21 [0.82, 1.78]
≥1 per day	3/84	0.89 [0.27, 2.89]	0.79 [0.23, 2.68]
Artificially sweetened soda (servings)			
<1 per month	48/867	1.0 (referent)	1.0 (referent)
1 to 4 per month	26/481	0.88 [0.54, 1.45]	0.72 [0.43, 1.20]
2 to 6 per week	67/1182	0.89 [0.61, 1.31]	0.53 [0.35, 0.81]
1 to 1.9 per day	40/479	1.70 [1.09, 2.65]	0.89 [0.56, 1.44]
≥2 per day	24/273	2.48 [1.46, 4.21]	0.92 [0.52, 1.65]

[a]Adjusted for age, caloric intake, HTN, BMI, diabetes, cigarette smoking, activity (METS/wk), cardiovascular disease, eGFR, and ACEI/ARB medication use.

Table 3. OR and 95% CI for cumulative averaged soda intake and eGFR decline ≥30% between 1989 and 2000

Soda Intake	Number of Cases/Total	Age and Energy-Adjusted OR [95% CI]	Multivariable-Adjusted[a] OR [95% CI]
Sugar soda (servings)			
<1 per month	239/1902	1.0 (referent)	1.0 (referent)
1 to 4 per month	61/720	0.67 [0.50, 0.90]	0.68 [0.50, 0.92]
2 to 6 per week	58/551	0.90 [0.66, 1.22]	0.90 [0.65, 1.23]
≥1 per day	14/83	1.70 [0.93, 3.11]	1.56 [0.84, 2.91]
Artificially sweetened soda (servings)			
<1 per month	88/867	1.0 (referent)	1.0 (referent)
1 to 4 per month	44/481	0.88 [0.60, 1.28]	0.82 [0.55, 1.20]
2 to 6 per week	136/1187	1.19 [0.89, 1.58]	1.02 [0.76, 1.37]
1 to 1.9 per day	48/459	1.14 [0.78, 1.66]	0.90 [0.61, 1.33]
≥2 per day[b]	56/262	2.71 [1.86, 3.94]	2.02 [1.36, 3.01]

Including lifetime consumption (grams) of acetaminophen, aspirin, or NSAIDs in the multivariable-adjusted models did not meaningfully change the associations between ≥2 servings of diet soda and faster eGFR decline.

[a]Adjusted for age, caloric intake, HTN, BMI, diabetes, cigarette smoking, activity (METS/wk), and cardiovascular disease.

[b]Using systolic or diastolic BP measurements as a covariate instead of HTN (yes/no) in the multivariable models also did not meaningfully change the associations between ≥2 servings of diet soda and faster eGFR decline.

ened beverages, which included noncarbonated sugar drinks such as fruit punch, lemonade, fruit drinks, and sugared ice tea, and we did not note any significant associations with MA or eGFR decline for any category, including ≥2 servings per day.

Discussion

Our results did not confirm the previously reported association between sugar soda and albuminuria, but we report a novel finding that ≥2 servings per day of artificially sweetened soda was associated with faster kidney function decline. No association between lower levels of artificially sweetened soda intake and eGFR decline was seen, implying a threshold effect rather than one that increases linearly (Table 3). We performed additional adjustments for other nutrients and foods known to be associated with eGFR decline in our study population as well as for diet quality (using AHEI) and weight change and observed a persistent direct association between ≥2 servings per day of diet soda and kidney function decline.

The observed association between diet soda and faster kidney function decline was not an *a priori* hypothesis and may be subject to incomplete adjustment for confounding despite our efforts in constructing additional models that included nutrients, foods,

and diet quality. We would also emphasize that causality cannot be established from an analysis of an observational cohort study, and that higher consumption of diet soda may be a marker of unmeasured characteristics that put women at higher risk for progressive kidney function decline.

However, if there is a causal association, we cannot determine if there is a specific type of artificial sweetener that may be associated with kidney function decline or even if it is an artificial sweetener or another ingredient in diet soda not found in sugar soda. Aspartame and saccharin were the primary artificial sweeteners used in carbonated low-calorie soft drinks in the 1980s and 1990s (29), which pertain to the years assessed by the FFQs used for the kidney function decline analyses. Saccharin-treated rats have been reported to demonstrate increased renal sodium excretion, but no GFR data were given (30). Upon extensive literature search, we did not find any published articles that investigated the effect of artificially sweetened soda or artificial sweeteners on glomerular filtration in animal models or humans, so more research is needed in this area. We also do not have information on soda brands but only general categories of sugar and artificially sweetened sodas from the FFQs.

A recent analysis of 15,745 participants of the Atherosclerosis Risk in Communities (ARIC) cohort also reported no association between higher consumption of sugar-sweetened soda (categorized as >1 glass per day, exactly 1 glass per day, <1 glass per day) with incident chronic kidney disease, which was defined as eGFR $<$ 60 ml/min per 1.73 m^2 at visit 2 and 4 in those with eGFR above this threshold at baseline (31). The ARIC study did not find an association between diet soda and incident chronic kidney disease using the same categories and definitions as for sugar-sweetened sodas, but the different definitions of soda intake and change in kidney function (in particular, the rate of eGFR decline was not specifically examined) make it difficult to directly compare these results with those of the study presented here.

Several important limitations of this investigation deserve mention. First, because only 3% of the women in our study consumed ≥1 sugar soda per day, a direct comparison to the albuminuria findings of the NHANES 1999 to 2004 results may not be possible because 17% of the NHANES population reported intake of ≥2 sugar sodas per day; however, no data on <2 servings per day of sugar or diet soda were provided in that study (5). No significant associations were seen for all sugar-sweetened beverage intake (carbonated and noncarbonated) at ≥2 servings per day, which is consistent with our null result for sugar soda. However, we cannot definitively exclude an association between sugar soda and our renal outcomes because of the low number of cases. We also did not collect data on noncarbonated artificially sweetened beverages. Moreover, our study population was comprised of mostly older Caucasian women whereas the NHANES study was comprised of a nationally representative U.S. population of adults including men and women, African Americans, and Hispanics. Therefore, our results may not necessarily be generalizable to other races or ethnicities, age groups, or men.

Additional limitations include the one-time assessment of ACR in the year 2000; therefore, the ACR analyses are cross-sectional like those in the NHANES study. The relatively few women meeting the definition of MA or macroalbuminuria (n = 241 or 7.2% of participants with albuminuria data) may have also limited our power to detect an association between carbonated beverages and albuminuria. The presence of residual confounding, particularly by unmeasured confounders, is also possible as with any observational study. However, notable strengths include data on eGFR change over 11 years in >3000 well characterized women who have eGFR and albuminuria data.

In summary, ≥2 servings per day of artificially sweetened soda was significantly associated with faster kidney function decline in older women with preserved kidney function. In light of the documented increase in soft drink consumption across all age groups between 1977 and 2001 (32), this finding generates a new hypothesis about diet soda and renal decline and has potential important public health implications if further research can establish the generalizability of this finding in men and non-whites as well as a causal relationship between artificially sweetened soda and kidney function decline.

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Disclosures

None.

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