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Carbonated Beverages and Chronic Kidney Disease

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Abstract

Background—Carbonated beverage consumption has been linked with diabetes, hypertension, and kidney stones, all risk factors for chronic kidney disease. Cola beverages, in particular, contain phosphoric acid and have been associated with urinary changes that promote kidney stones.

Methods—We examined the relationship between carbonated beverages (including cola) and chronic kidney disease, using data from 465 patients with newly diagnosed chronic kidney disease and 467 community controls recruited in North Carolina between 1980 and 1982.

Results—Drinking 2 or more colas per day was associated with increased risk of chronic kidney disease (adjusted odds ratio = 2.3; 95% confidence interval = 1.4–3.7). Results were the same for regular colas (2.1; 1.3–3.4) and artificially sweetened colas (2.1; 0.7–2.5). Noncola carbonated beverages were not associated with chronic kidney disease (0.94; 0.4–2.2).

Conclusions—These preliminary results suggest that cola consumption may increase the risk of chronic kidney disease.

The prevalence of chronic kidney disease more than doubled from 1988 to 2001, affecting more than 20 million adults in the United States.^{1,2} Since the incidence of kidney disease and associated health care costs are expected to continue to rise,^{3,4} identifying modifiable risk factors is a public health priority.^{5,6}

Well-documented risk factors for kidney disease include diabetes, hypertension, and family history.^{7,8} Kidney stones may also increase risk,⁹ and soft-drink consumption has been associated with diabetes,¹⁰ hypertension,¹¹ and kidney stones.^{12–14} Cola beverages, which are acidified with phosphoric acid, have been associated with urinary changes known to promote kidney stones.^{13,15,16} Current recommendations for patients with kidney disease or kidney stones include avoiding cola beverages,^{17,18} but there is limited evidence to support this recommendation.

In this paper, we examine the association between carbonated beverage intake and chronic kidney disease.

METHODS

We used data from a case-control study of North Carolina patients with chronic kidney disease and community controls.¹⁹ Trained personnel identified potential cases by reviewing

medical charts from 4 North Carolina hospitals. Cases were included if they were newly diagnosed with kidney disease between September 1980 and August 1982 and had a sustained elevation in serum creatinine (2 or more measurements $\geq 130 \mu\text{mol/L}$). Patients younger than 30 years or who resided outside of North Carolina were excluded. Patients with preexisting kidney disease, polycystic kidney disease, systemic lupus erythematosus, multiple myeloma, amyloidosis, or hereditary nephritis were also excluded.

Of 709 patients found eligible, 607 (86%) could be contacted and 554 (78%) agreed to participate. We excluded individuals who were missing data for carbonated beverages ($n = 25$) or potential confounders ($n = 64$), leaving 465 cases for analysis. Patients were classified into kidney disease subtypes using available clinical and laboratory information.

Community controls were selected using random digit dialing for those younger than 65 years of age, and Medicare recipient listing for those 65 years of age or older. Controls were frequency-matched to cases by age (within 5 years), sex, race, and proximity to a study hospital. Of 717 potential controls, 608 (85%) could be contacted and 520 (73%) agreed to participate. We excluded individuals with history of kidney disease ($n = 4$), and those with missing data on beverage consumption ($n = 18$) or on potential confounders ($n = 31$), leaving 467 for analysis.

Information was collected through telephone interviews. Subjects or proxy respondents were asked about beverage consumption over their adult life. The question was "Please tell me about your drinking habits before 1980. Since you were 18-years-old did you ever drink (list of individual beverages) and, if so, how many times per day, week, month, or year did you usually drink the beverage?" Questions were asked separately about regular cola, artificially sweetened cola, noncola caffeinated beverages, and noncola artificially sweetened beverages. Average intake of water, coffee, and tea was also recorded. At the time of the interviews (1982–1983) noncaffeinated teas and colas were uncommon, and thus tea and cola were assumed to contain caffeine. Total caffeine from noncola sources was calculated by assigning an average caffeine content of 80 mg per cup of coffee, 40 mg per cup of tea, and 40 mg per glass of other caffeinated beverages.

We used unconditional logistic regression to estimate the association between carbonated beverages and chronic kidney disease. We identified potential confounders based on their known association with beverage consumption and kidney disease. We retained in the final model covariates that resulted in at least a 10% change in the coefficient. We examined the following variables as potential confounders: body mass index (BMI), education, income, smoking, analgesic use, alcohol use, caffeine from noncola sources, fluid intake from noncola sources, history of diabetes, hypertension, kidney stones, and proxy respondent status. We also explored effect modification by history of diabetes, hypertension, or kidney stones through stratified models and by modeling interaction terms. Additionally, we performed separate analyses for specific diagnostic subgroups.

RESULTS

Compared with controls, cases more frequently reported a lower income and educational level, had a higher mean BMI, and reported more frequent use of analgesic medications, as well as a history of diabetes, hypertension, and kidney stones (Table 1). Proxy respondents were more frequent for cases than controls (54% vs. 10%). Consumption of cola was higher among cases, as was fluid intake from noncola sources. Caffeine intake from noncola sources was similar between cases and controls.

Consumption of 2 or more glasses of cola per day was associated with a 2-fold risk of kidney disease compared with the reference category, after adjusting for age, race, sex,

proximity to study hospital, BMI, income, proxy respondent status, education, analgesic use, and diabetes (Table 2). Estimates for regular colas or artificially sweetened colas were similar. Increasing quantities of noncola carbonated beverages, caffeine from noncola sources, and intake of fluids from sources other than cola were not associated with kidney disease risk. Results were similar after excluding proxy respondents (Table 2) and for those without diabetes, hypertension, or history of kidney stones (Table 3). We assessed robustness of the finding by assigning high cola consumption to controls with missing data and then by assigning no cola consumption to cases with missing data. Neither assumption materially changed the results (data not shown). Table 4 presents the adjusted odds ratios for specific diagnostic subgroups of chronic kidney disease in relation to all colas combined.

DISCUSSION

Our analysis suggests that consumption of 2 or more cola beverages per day was associated with an increased risk of chronic kidney disease. We saw no increase in risk associated with consumption of noncola carbonated beverages, or with noncola caffeinated beverages.

Although we know of no previous study that assesses a link between cola beverages and kidney disease, there is evidence supporting the plausibility of our findings. One difference between cola and noncola carbonated beverages is that, while noncola beverages have been predominantly acidified using citric acid, cola beverages are generally acidified using phosphoric acid. Phosphorus may have an effect on the risk of kidney disease. Case reports have linked sodium phosphate bowel cleansing preparations to acute and sometimes irreversible renal failure,^{20,21} characterized by calcium phosphate deposits in the distal tubules and collection ducts or nephrocalcinosis.^{22–24} Although the level of phosphorus from colas is far lower than that from bowel preparations, long-term cola intake may lead to kidney damage, especially in the presence of underlying renal dysfunction.

Laboratory studies have shown that high phosphorus diets can cause nephrocalcinosis in rats.^{25,26} Diets high in phosphate may increase plasma phosphorus levels, with phosphate in colas perhaps being more bioavailable.^{27,28}

In a randomized trial among men with kidney stones, recurrence of kidney stones was higher among those who continued to drink soft drinks containing phosphoric acid, compared with those who drank beverages acidified by citric acid.¹³ Cola consumption can result in physiologic changes in urine composition that are conducive to oxalate kidney stone formation.^{15,16,29,30} We saw no association with cola among individuals with history of kidney stones, although the sample size for this subanalysis was small.

The odds ratios for a cola association were higher among those with a history of diabetes, although the test for interaction was not statistically significant. A recent study has shown a positive association between cola beverages and incidence of hypertension.¹¹ In our analysis we saw no evidence of effect modification by hypertension, nor did hypertension confound our results.

We cannot rule out the possibility that the observed effect may have been due to caffeine, but we did not see an increasing risk with increasing consumption of caffeine from noncola sources.

This study had several limitations. We were not able to examine dietary differences between cases and controls or between cola and noncola drinkers. Cola drinkers may also differ from noncola drinkers in other ways that could lead to residual confounding. Among controls, cola drinkers were younger, less likely to have a history of diabetes, and more likely to have

a history of kidney stones compared with noncola drinkers. We controlled for age and diabetes, and history of kidney stones was not a confounder.

The high proportion of proxy respondents among cases is a further limitation. However, the estimated effect of cola was stronger after excluding proxy respondents, suggesting that their inclusion attenuated the estimated risk.

Beverage consumption was self-reported and thus subject to potential bias. However, since a link between carbonated beverage intake and risk of kidney disease was unknown at the time of the study, it is unlikely that cases would have differentially reported their beverage consumption. In this analysis, only colas were consistently associated with chronic kidney disease, which makes reporting bias an unlikely explanation for our findings.

Participants reported average beverage intake over their adult life. While these reports may reflect more recent consumption, there is no reason to believe that those who currently drank colas had not done so in the past. Colas have been available since the late 1800s and were first popularized in the southern United States.

Cola consumption is common and chronic kidney disease is a substantial public health burden. Our preliminary result of an association between cola consumption and risk of chronic kidney disease deserves to be explored in more detailed studies.

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TABLE 1

Selected Characteristics of Patients With Chronic Kidney Disease and Community Controls in the North Carolina Health Study, 1980–1982 (n = 932)

Characteristic	Cases (n = 465)		Controls (n = 467)	
	No.	Distribution*	No.	Distribution*
Age (yrs) [†]	465	63 ± 12.2	467	61 ± 12.2
White race	260	56	255	55
Male sex	260	56	262	56
Annual income				
<\$10,000	266	57	194	42
\$10,000	199	43	273	58
Education (yrs)				
<8	174	38	115	24
8–11	136	29	129	28
12+	155	33	223	48
Smoking status [†]				
Nonsmoker	213	46	185	40
Ex-smoker	101	22	116	25
Current smoker	145	32	160	35
BMI (kg/m ²) [†]	465	27.1 ± 6.4	467	25.3 ± 4.3
<18.5	10	2	10	2
18.5–24.9	180	39	233	50
25–29.9	169	36	160	34
30+	106	23	64	14
History of diabetes	136	29	28	6
History of hypertension	282	61	143	31
History of kidney stones	78	17	28	6
Proxy respondents	251	54	45	10
Proximity to study hospital				
Near	244	52	244	52
Far	221	48	223	48
Analgesic use				
Never	249	54	327	70
Weekly	135	29	113	24
Daily	81	17	27	6
Daily cola consumption (glasses/d) [§]	465	1.0 (0.2–2.0)	467	0.4 (0.1–1.0)
Caffeine from noncola sources (mg/d) [§]	465	120 (52–201)	467	120 (74–200)
Fluid intake from other sources (glasses/d) [§]	438	5.0 (3.3–7.1)	441	4.6 (3.1–6.1)

* Percent, unless otherwise indicated.

[†] Mean ± SD.

[‡]Total number varies due to missing values (n = 920).

[§]Median (interquartile range).

TABLE 2

Risk of Chronic Kidney Disease Associated With Carbonated Beverage Consumption, Caffeine Intake, and Noncola Fluid Intake Among all Subjects and Among Self-Respondents

	All Subjects		Self-Respondents Only	
	No. Cases/Controls	AOR* (95% CI)	No. Cases/Controls	AOR* (95% CI)
All colas combined	465/467		214/422	
Never or < 1 per wk [†]	81/128	1.0	33/118	1.0
1 per wk to < 1 per day	146/158	1.50 (0.97–2.33)	64/144	1.62 (0.96–2.72)
1 to <2 per day	108/98	1.67 (1.03–2.67)	51/85	2.13 (1.23–3.70)
2 per day	130/83	2.27 (1.40–3.68)	66/75	2.82 (1.62–5.00)
Regular colas [‡]	465/467		214/422	
Never or < 1 per wk [†]	107/141	1.0	41/130	1.0
1 per wk to < 1 per day	141/156	1.43 (0.94–2.19)	63/142	1.62 (0.98–2.67)
1 to <2 per day	106/97	1.69 (1.07–2.67)	56/84	2.16 (1.27–3.66)
2 per day	111/73	2.12 (1.30–3.43)	54/66	2.51 (1.44–4.38)
Artificially sweetened colas [§]	465/467		214/422	
Never or < 1 per wk [†]	376/416	1.0	176/378	1.0
1 per wk to <1 per day	40/29	1.30 (0.69–2.46)	16/27	1.37 (0.67–2.79)
1 to <2 per day	26/16	0.89 (0.38–2.06)	11/13	1.28 (0.51–3.21)
2 per day	23/6	2.07 (0.68–6.31)	11/4	4.21 (1.21–14.61)
Noncola carbonated beverages [¶]	449/458		206/413	
Never or < 1 per wk [†]	346/339	1.0	147/299	1.0
1 per wk to < 1 per day	56/71	0.80 (0.50–1.27)	31/68	0.73 (0.44–1.22)
1 to <2 per day	29/32	0.96 (0.50–1.84)	16/31	0.89 (0.44–1.78)
2 per day	18/16	0.94 (0.41–2.18)	12/15	0.96 (0.40–2.30)
Caffeine from noncola beverages (mg/d) [§]	465/467		214/422	
<100 [‡]	214/220	1.0	110/199	1.0
100 < 200	125/121	1.07 (0.72–1.58)	59/116	0.85 (0.55–1.32)
200 < 300	71/68	0.70 (0.43–1.15)	23/57	0.68 (0.38–1.22)

	All Subjects			Self-Respondents Only		
	No. Cases/Controls	AOR*	(95% CI)	No. Cases/Controls	AOR*	(95% CI)
300	55/58	0.68	(0.40–1.14)	22/50	0.61	(0.33–1.14)
Noncola fluid intake ^{§,}	438/441			201/402		
Never or <3 per day [‡]	72/76	1.0		33/68	1.0	
3 to <5 per day	114/151	0.79	(0.48–1.30)	48/141	0.67	(0.38–1.18)
5 to <7 per day	120/114	1.09	(0.65–1.81)	57/101	1.08	(0.61–1.93)
7 per day	132/100	1.26	(0.75–2.11)	63/92	1.20	(0.68–2.13)

AOR indicates adjusted odds ratios; CI, confidence interval.

* All models adjusted for matching factors and confounders: BMI, income, proxy respondent, education, analgesic use and diabetes; BMI and age were modeled as continuous variables, all others as categorized in Table 1. Each beverage type was modeled separately. Results are adjusted for other beverages only as indicated.

[‡]Reference category.

[‡]Models also adjusted for artificially sweetened colas.

[§]Models also adjusted for regular colas.

^{||}Models adjusted for any cola.

^{||}Number of participants reduced due to missing values for specified beverage.

Risk of Chronic Kidney Disease Associated With Increasing Levels of Carbonated Cola Consumption, Stratified by Self-reported Diabetes, Hypertension, and Kidney Stones

TABLE 3

	Cases No. (%)	Controls No. (%)	AOR*	(95% CI)	Cases No. (%)	Controls No. (%)	AOR*	(95% CI)
	History of Diabetes							
	Yes (n = 164)				No (n = 768)			
	(n = 136)	(n = 28)			(n = 329)	(n = 439)		
All colas combined								
Never or <1/wk [‡]	21 (15)	13 (46)	1.0		60 (18)	115 (26)	1.0	
1/wk to <1/d	42 (31)	5 (18)	5.7	(1.2–27.0)	104 (32)	153 (35)	1.2	(0.8–1.9)
1 to <2/d	32 (24)	5 (18)	6.4	(1.4–28.7)	76 (23)	93 (21)	1.4	(0.8–2.2)
>2/d	41 (30)	5 (18)	4.1	(0.9–18.3)	89 (27)	78 (18)	2.0	(1.2–3.3)
History of Hypertension [‡]								
	Yes (n = 425)				No (n = 504)			
	(n = 292)	(n = 143)			(n = 180)	(n = 324)		
All colas combined								
Never or <1/wk [‡]	52 (18)	34 (24)	1.0		29 (16)	94 (29)	1.0	
1/wk to <1/d	90 (32)	49 (34)	1.2	(0.6–2.4)	55 (31)	109 (34)	1.6	(0.9–3.1)
1 to <2/d	65 (23)	32 (22)	1.4	(0.7–2.8)	42 (23)	66 (20)	1.9	(0.9–3.7)
2/d	75 (27)	28 (20)	1.7	(0.8–3.5)	54 (30)	55 (17)	2.7	(1.3–5.3)
History of Kidney Stones [‡]								
	Yes (n = 106)				No (n = 822)			
	(n = 78)	(n = 28)			(n = 384)	(n = 438)		
All colas combined								
Never or <1/wk [‡]	15 (19)	3 (11)	1.0		65 (17)	125 (29)	1.0	
1/wk to <1/d	26 (33)	9 (32)	0.8	(0.1–4.5)	120 (31)	149 (34)	1.6	(1.1–2.7)
1 to <2/d	23 (30)	9 (32)	0.7	(0.1–4.3)	85 (22)	89 (20)	1.8	(1.1–3.0)
2/d	14 (18)	7 (25)	0.6	(0.1–4.2)	114 (30)	75 (17)	2.7	(1.6–4.5)

* All models adjusted for matching factors and the confounders BMI, income, proxy respondent, education, analgesic use, and diabetes (except diabetic models are not adjusted for diabetes).

[†] Reference category.

[‡] Number of participants reduced due to missing values.

TABLE 4

Risk of Chronic Kidney Disease Associated With Increasing Levels of Cola Consumption Overall and by Subtype of Chronic Kidney Disease*

	No. Cases	Carbonated Cola Beverages					
		1/Week to < 1 per Day		1 to <2 per Day		2 per Day	
		AOR [†]	(95% CI)	AOR [†]	(95% CI)	AOR [†]	(95% CI)
Chronic kidney disease (all types)	465	1.5	(1.0–2.3)	1.7	(1.0–2.7)	2.3	(1.4–3.7)
Nephrosclerosis	86	2.3	(1.1–5.0)	1.5	(0.6–3.5)	1.9	(0.6–3.8)
Diabetic nephropathy	95	1.4	(0.5–3.3)	1.5	(0.6–3.8)	1.9	(0.8–4.8)
Glomerulonephritis	63	1.0	(0.4–2.2)	1.5	(0.6–3.6)	1.2	(0.5–3.0)
Interstitial nephritis	92	1.2	(0.6–2.5)	1.4	(0.6–3.2)	1.9	(0.9–4.2)
Renal insufficiency [‡]	101	1.5	(0.7–3.0)	1.0	(0.5–2.3)	2.4	(1.1–5.3)
ESRD [‡]	28	0.8	(0.2–3.0)	0.6	(0.1–2.6)	1.6	(0.5–5.9)

* Reference category = never, or less than once per week.

[†] All models adjusted for matching factors and confounders BMI, income, proxy respondent, education, analgesic use, and diabetes (except diabetic nephropathy model not adjusted for diabetes). BMI and age were modeled as continuous variables, all others as categorized in Table 1.

[‡] It was not possible to further classify 129 patients whose disease had progressed to renal insufficiency or end-stage disease (ESRD).