Carbonated beverages and urinary calcium excretion[1–3]

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ABSTRACT

Background: Intake of carbonated beverages has been associated with increased fracture risk in observational studies. The usual explanation given is that one or more of the beverage constituents increase urinary calcium.

Objective: We assessed the short-term effects on urinary calcium excretion of carbonated beverages of various compositions.

Design: An incomplete random block design was used to study 20–40-y-old women who customarily consumed ≥680 mL carbonated beverages daily. Four carbonated beverages were tested: 2 with caffeine and 2 without. Two contained phosphoric acid as the acidulant and 2 contained citric acid. The study included one neutral control (water) and one positive control (skim or chocolate milk). Serving size was 567 mL for the carbonated beverages and water and 340 mL for the milks. Beverages were consumed with a light breakfast after an overnight fast; no other foods were ingested until urine collection was complete. pH, titratable and total acidity, sodium, creatinine, and calcium were measured in 2-h (morning) fasting and 5-h postbeverage urine specimens.

Results: Relative to water, urinary calcium rose significantly only with the milks and the 2 caffeine-containing beverages. The excess calciuria was ≈0.25 mmol, about the same as previously reported for caffeine alone. Phosphoric acid without caffeine produced no excess calciuria; nor did it augment the calciuria of caffeine.

Conclusions: The excess calciuria associated with consumption of carbonated beverages is confined to caffeinated beverages. Acidulant type has no acute effect. Because the caffeine effect is known to be compensated for by reduced calciuria later in the day, we conclude that the net effect of carbonated beverage constituents on calcium economy is negligible. The skeletal effects of carbonated beverage consumption are likely due primarily to milk displacement. *Am J Clin Nutr* 2001;74:343–7.

KEY WORDS Carbonated beverages, colas, caffeine, phosphorus, phosphoric acid, urinary calcium, acid loading, citric acid, fracture risk

INTRODUCTION

In several observational studies, intake of carbonated beverages was associated with reduced bone mass or increased fracture risk, both later in life (1) and in children and adolescents (2–4). In most reports, colas were more strongly associated than were other carbonated beverages. Several investigators suggested that the factor or factors responsible for this association may be the increase in phosphorus intake or the net acid load of those beverages that use phosphoric acid as the acidulant or the caffeine of those beverages that are caffeinated. More recently, fructose, found in beverages that use natural sweeteners, was implicated as a possible cause of reduced calcium balance (5). For most of these factors, the effect is usually attributed to increased urinary calcium loss. Individually, phosphorus and caffeine were shown to have little or no net effect (6–11), but concern remains about the acid load (12). The combination of all 3 factors, as would be found in many colas, has not been directly tested.

The issue is especially important today because calcium intakes in North America fall far short of current recommendations. Per capita carbonated beverage consumption has risen dramatically, and carbonated sodas are now the preferred beverage of 20–40 y-old women (13). Interference in the calcium economy of persons with already low calcium intakes would only aggravate any calcium shortfall. This issue was studied in an experimental design only once before, and then only for a single cola (9). Accordingly, and because of the wide interest among nutritionists and dietitians in the possible effects of carbonated beverages, we undertook the present study. We investigated the acute effect on urinary calcium loss of intake of carbonated beverages of various compositions by adult women who were habitual users of such beverages.

SUBJECTS AND METHODS

Protocol

Four different carbonated beverages were tested, water was used as a negative control, and 2 milks (white and chocolate) were used as positive controls, bringing the total of tested beverages to 7. The beverages, their composition, and pertinent characteristics are shown in **Table 1**. Two of the beverages (Coke and Coke-Free; The Coca-Cola Company, Atlanta) included phosphoric acid as the acidulant and 2 (Mountain Dew; The Pepsi Cola Company, Purchase, NY; and Sprite; The Coca-Cola

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TABLE 1
Beverage composition and characteristics[1]

Beverage	Acidulant	Phosphorus	Calcium	Caffeine	Sugars	Na[2]	pH	Net acid[3]
		mmol	*mmol*	*mg*	*g*	*mEq*		*mEq H⁺*
Caffeinated cola	Phosphoric acid	2.7	0	77.5	67.5	4.2	2.54	4.94
Caffeine-free cola	Phosphoric acid	2.5	0	0	62.5	2.2	2.94	4.53
Caffeinated noncola	Citric acid	0.04	0	91.7	76.7	1.5	3.27	0
Caffeine-free noncola	Citric acid	0	0	0	65.0	2.1	3.37	0
White milk, nonfat	NA	9.3	11.2	0	16.0	7.5	—	—
Chocolate milk, lite[4]	NA	10.7	11.6	0	16.2	8.7	—	—

[1] Content per 567-mL (20-oz) serving of carbonated beverages and per 340-mL (12-oz) serving of milk. NA, not applicable.
[2] Determined by analysis.
[3] Total H from H_3PO_4 would be 1.67 × the values shown, but H_3PO_4 is only 60% dissociated at pH 7.4.
[4] Lite = low fat and no added sugar.

Company) used citric acid. Two provided caffeine (Coke and Mountain Dew) and 2 did not (Coke-Free and Sprite). The brands studied were chosen because their contents of the putatively harmful agents were toward the high end of their categories.

An incomplete random block design was set up so that 5 beverages were tested on each woman: water, both of the cola beverages, one of the milks, and one of the 2 citric acid–containing beverages. Within each subject the beverage sequence was randomized by using the random number function of EXCEL (Microsoft, Redmond, WA).

The sequence of tests in each woman was arranged on a 1-wk cycle, so that the entire suite of 5 tests was completed, in most subjects, within one calendar month. The subjects reported to the research unit fasting in the morning, after having voided at home, noting the time. They stayed in the unit, fasting, until a 2-h second specimen was collected. They were then fed a breakfast consisting of the test beverage and 2 pieces of specially baked, low-calcium, Italian style white bread, toasted, with butter. The milk serving was 340 mL (12 oz) and the carbonated beverage servings were 567 mL (20 oz). The difference in fluid volume between the milks and the other beverages was compensated for by having the subjects ingest an additional 227 mL deionized water. The differences in carbohydrate intake between the beverages were compensated for by providing the subjects with the requisite number of jelly beans (consisting mainly of sucrose and corn syrup) to equalize total sugar intake for all test meals (1.256 MJ total). All urine was collected during the next 5-h period, during which time subjects consumed an additional 567 mL deionized water; no further food was consumed until the 5-h urine collection had been completed.

Women were recruited who habitually consumed at least two 340-mL (12-oz) cans of carbonated beverages daily. The total sample size was 32. Two women dropped out of the study early and their data are not included in this report. All subjects were instructed to maintain their usual calcium intakes throughout the study and to refrain from high-sodium foods for 2 d before each test. The mean (±SD) age of the subjects was 31.4 ± 5.6 y and their mean body mass index (in kg/m^2) was 25.2 ± 3.75. The subjects' habitual intake of carbonated beverages, by history, ranged from two to seven 340-mL (12-oz) servings/d ($\bar{x}$: 3.16). Twenty-seven of the 30 subjects habitually consumed predominantly colas, and 3 consumed predominantly the citric acid–containing beverages. Median calcium intake, as determined by a food-frequency questionnaire modified from the Block food-frequency questionnaire (14), was 19.6 mmol/d (interquartile range: 15.8–30.4 mmol/d).

Analytic methods

Urine samples were analyzed for calcium, creatinine, sodium, titratable acidity, and total acidity. Colas were analyzed for acidity, sodium, and phosphorus. Calcium was determined by atomic absorption and sodium by flame emission spectrophotometry (Perkin Elmer AAnalyst, Norwalk, CT), creatinine by an autoanalyzer method based on the Jaffe reaction (15), and both titratable and total acidity by the method of Chan (16). (This method equates total acidity to titratable acidity plus ammonium ion, less carbonic acid.) Phosphorus was analyzed by an autoanalyzer method based on Fiske and Subba Row (17). pH was measured by using a Fisher Accumet pH meter (model 915; Fisher Scientific, St Louis). The caffeine content of the beverages was attained from The Food Processor database (ESHA, Salem, OR).

Statistical methods

The data were analyzed in various ways, depending on the a priori hypotheses, ie, some by ANOVA, testing for treatment and order, and some by simple paired *t* tests. When the data were not normally distributed, the summary descriptive statistics were the median and interquartile range. Differences were tested against a null hypothesis of zero. In addition to the raw data, various derived variables were calculated, principally focused on estimating the excess urinary calcium (Ca_u) after beverage consumption. The increments produced by carbonated beverage ingestion were not much greater than the changes observed with water alone or the usual day-to-day variability in calciuria. (The within-subject CV for calcium content of the 2-h fasting specimens was 10.9%; that for the calcium-to-creatinine ratio was 9.1%.) For that reason and to minimize bias in estimating effects, we used 4 methods of estimating excess calciuria. The resulting data were analyzed statistically as above. The 4 methods, based on the analyte content in the 5-h urine sample after the test breakfast, except where otherwise indicated, were as follows:

$$\text{Excess } Ca_u1 = Ca_u \text{ (bev)} - Ca_u \text{ (water)} \quad (1)$$

$$\text{Excess } Ca_u2 = \text{Ca/creatinine (bev)} - \text{Ca/creatinine (water)} \quad (2)$$

$$\text{Excess } Ca_u3 = \text{Ca (5 h)} - [\text{Ca/creatinine (fasting)} \times \text{creatinine (5 h)}] \quad (3)$$

$$\text{Excess } Ca_u4 = \text{Excess } Ca_u3 \text{ (bev)} - \text{excess } Ca_u3 \text{ (water)} \quad (4)$$

where bev is beverage. The first method is the simple excess for a given beverage relative to the water meal, separated in time by

TABLE 2
Urinary sodium and calcium excretion[1]

Beverage	Fasting		5-h Postingestion		
	Na/creatinine[2]	Ca/creatinine	Na/creatinine[2]	Ca/creatinine	Ca
	mEq/mmol	*mmol/mmol*	*mEq/mmol*	*mmol/mmol*	*mmol*
Caffeinated cola (n = 30)	7.86 (5.61–13.2)	0.176 (0.130–0.232)	12.1 (8.73–17.3)	0.278 (0.235–0.391)	0.712[3] (0.522–0.959)
Caffeine-free cola (n = 30)	9.14 (5.58–12.2)	0.132 (0.078–0.223)	11.9 (8.73–14.7)	0.225 (0.150–0.306)	0.523 (0.341–0.778)
Caffeinated noncola (n = 15)	9.46 (7.00–13.7)	0.225 (0.136–0.236)	14.0 (10.2–20.6)	0.304 (0.228–0.442)	0.719[3] (0.566–0.934)
Caffeine-free noncola (n = 15)	8.75 (6.36–13.8)	0.159 (0.089–0.216)	11.7 (9.46–15.1)	0.278 (0.199–0.368)	0.549 (0.428–0.797)
Water (n = 30)	7.51 (5.55–10.3)	0.137 (0.103–0.238)	9.18 (7.76–19.1)	0.212 (0.147–0.317)	0.559 (0.261–0.714)
Milk (n = 30)	8.44 (5.62–13.71)	0.171 (0.077–0.288)	18.9 (11.9–23.57)	0.456 (0.198–0.563)	0.996 (0.458–1.422)

[1] Median (interquartile range).
[2] Na/creatinine was greater in postingestion samples than in fasting samples for all beverages except milk, $P < 0.001$.
[3] The 2 caffeine-containing beverages differed marginally from the 2 noncaffeinated beverages, $P < 0.08$.

≥1 wk; the second is the same excess, adjusted for creatinine; the third is the excess relative to what might have been predicted for that woman from her fasting calcium-to-creatinine ratio on that same morning; and the fourth is the same as the third, except with the similarly calculated water excess subtracted from the beverage excess. Because the findings for the 2 milks did not differ significantly, and because milk was in the experiment only as a positive control (and its type was not relevant to the research question), we pooled the results from milk across the 30 subjects.

RESULTS

The measured urinary sodium and calcium excretion values, by beverage consumed, are shown in **Table 2**. Except for milk and to a lesser extent the 2 caffeine-containing beverages, there was little urinary calcium excretion with any of the beverages. The urinary sodium-to-creatinine ratio in the 5-h collections was higher than that in the fasting collection for all beverages except milk (which would be expected to elevate urinary sodium because it contains some sodium). This finding probably reflects the sodium diuresis of volume expansion. This rise in the ratio of sodium to creatinine was nonsignificantly greater for the 2 caffeine-containing beverages than for the caffeine-free products (which did not differ significantly from each other), probably reflecting the recognized mild sodium diuretic effect of caffeine. Urinary sodium (Na_u) showed the expected relation to urinary calcium (data not shown), with the slope of Ca_u on Na_u being ≈8 μmol Ca/mEq Na (0.8 mmol Ca/100 mEq Na) in the fasting samples and ≈14 μmol Ca/100 mEq Na (≈1.4 mmol Ca/100 mEq Na) in the 5-h postbeverage collections.

The within-subject excess calcium excretion values, as calculated by use of the 4 methods, are shown in **Table 3**. As expected, milk produced highly significant increases, amounting to ≈0.5 mmol by 3 of the 4 methods. This shows that the protocol and methods used could detect excess calciuria. The 2 caffeinated beverages also produced increases, significant for all 4 methods. The magnitude of the excess, however, was relatively small [ie, in the range of 0.110–0.346 mmol (4–14 mg)] over the 5-h postingestion period. The caffeine-free beverages, by contrast, produced much smaller increases that were barely significant (and not so for several of the methods). The foregoing excess calciuria estimates were significantly different from zero.

The purpose of the present study was also to compare the beverages with one another to evaluate the effects of their differing compositions. The variability in response between women was of the same general magnitude as the excess calciuria values, and by ANOVA there were no significant differences among the carbonated beverages. However, in a post hoc analysis comparing the aggregate of the 2 caffeinated beverages with the aggregate of the 2 caffeine-free beverages, the urinary calcium rise was greater for the caffeine-containing products ($P < 0.05$). Within acidulant type, the beverage effects were not significantly different from one another.

The urinary acid excretion data are shown in **Table 4**. As expected, the 2 phosphoric acid–containing colas produced the

TABLE 3
Excess urinary calcium excretion[1]

Beverage	Excess 1	Excess 2	Excess 3	Excess 4
Caffeinated cola (n = 30)	0.264 ± 0.061[2]	0.110 ± 0.024[2]	0.265 ± 0.047[2]	0.152 ± 0.043[3]
Caffeine-free cola (n = 30)	0.102 ± 0.058	0.066 ± 0.032[4]	0.128 ± 0.097	0.014 ± 0.093
Caffeinated noncola (n = 15)	0.263 ± 0.088[4]	0.125 ± 0.037[3]	0.346 ± 0.063[2]	0.254 ± 0.059[2]
Caffeine-free noncola (n = 15)	0.156 ± 0.073	0.090 ± 0.033[4]	0.279 ± 0.053[2]	0.137 ± 0.054[4]
Milk (n = 30)	0.514 ± 0.081[2]	0.238 ± 0.037[2]	0.541 ± 0.082[2]	0.427 ± 0.076[2]

[1] $\bar{x}$ ± SEM; values are in mmol or mmol/mmol, as applicable for each method. *See* text for derivation of the various excess excretion values.
[2–4] Significantly different from 0.0 (H_0): [2] $P < 0.001$, [3] $P < 0.01$, [4] $P < 0.05$.

TABLE 4
Urine acidity values[1]

Beverage	Fasting	5-h Postingestion			
	pH	pH	TA	NH_4	NAE
			mEq	*mEq*	*mEq*
Caffeinated cola (n = 30)	5.68 ± 0.39	6.01 ± 0.33	2.56 ± 2.35	0.042 ± 0.093	2.59 ± 2.40
Caffeine-free cola (n = 30)	5.83 ± 0.46	6.10 ± 0.36	1.93 ± 2.28	0.024 ± 0.047	1.95 ± 2.30
Caffeinated noncola (n = 15)	5.77 ± 0.46	6.05 ± 0.47	1.22 ± 2.76	0.013 ± 0.0097	1.24 ± 2.77
Caffeine-free noncola (n = 15)	5.89 ± 0.55	6.16 ± 0.46	0.899 ± 3.12	0.015 ± 0.025	0.91 ± 3.12
Water (n = 30)	5.73 ± 0.48	6.19 ± 0.43	0.831 ± 3.22	0.011 ± 0.011	0.84 ± 3.22
Milk (n = 30)	5.81 ± 0.56	6.29 ± 0.46	−0.127 ± 4.30	0.043 ± 0.047	−0.08 ± 4.33

[1] $\bar{x}$ ± SD. TA, titratable acidity; NAE, net acid excretion.

urines with the greatest acid excretion; even so, the total acid excretion over the 5-h postingestion period amounted to only 2.59 mEq for the caffeinated and 1.95 mEq for the caffeine-free colas, less than one-half the ingested acid load. Average net acid excretion for the milk was −0.08 ± 4. 33 mEq, reflecting the alkaline ash characteristic of dairy products when metabolized.

DISCUSSION

Most apparent in these data is the smallness of the rise in urinary calcium after the ingestion of carbonated beverages. This most likely means both that carbonated beverages as a whole have little intrinsic effect on calcium economy and that, accordingly, variations in beverage composition have little effect. However, even a small excess excretion, if cumulative and not offset by additional calcium absorption, would inevitably lead to bone loss. Hence, it seems worthwhile to try to tease out the separate contributions of the various constituents of these beverages.

Excluding milk, the largest differences (and the only significant differences between the beverages) were found for the 2 caffeine-containing beverages. The mean magnitude of the excess calciuria for the caffeine-containing beverages, most directly expressed by Ca_u excesses 1, 3, or 4, ranged from 0.15 to 0.35 mmol (6–14 mg). Massey et al (18–20), in various studies, reported excess calciuria in the range of 0.05 to 0.22 mmol (2–9 mg) per 100 mg caffeine, ie, slightly lower than the values we found. However, Massey et al did not administer a sugar load as contained in our beverages and their collection periods were substantially shorter than ours. Making allowances for these differences in protocol, it seems that our excess calciuria values are roughly comparable to theirs. If that is the case, then the added variables of acid and phosphorus seem to have produced no detectable effects in their own right. This conclusion is supported by our finding that the caffeine-free cola containing phosphoric acid did not produce a significant excess calciuria by 3 of the 4 methods (and by none of them when the analysis was corrected for multiple comparisons).

At the same time it must be noted that the calciuric effect of caffeine is biphasic. Massey et al previously showed a compensatory drop in overnight calciuria in caffeine consumers (11), and no net effect on 24-h excretion (10). Similarly, Barger-Lux et al (21) found no effect of caffeine on 24-h urinary calcium at caffeine doses 5–6 times those used in this study. Smith et al (9) showed no net effect of 1.4 L diet cola on either serum or 24-h urinary calcium. Hence, that portion of the observed excess calciuria that may be due to caffeine can probably be dismissed as being of no consequence to calcium economy.

The issue, however, is somewhat more complicated because carbonated beverages contain several other substances known to influence urinary calcium; even their volume may exert some influence. Sugars raise urinary calcium (5, 22–24), as does sodium (25). By contrast, phosphorus reduces urinary calcium (26). The effects of all 3 in this study may be approached by examining the data for the water meal. With this meal, subjects ingested the full volume and a load of sugar energetically equivalent to that of the test beverages, but no acid, phosphorus, or caffeine. The mean excess sodium excretion, calculated from the rise in the sodium-to-creatinine ratio (ie, the analog of Ca_u excess 3), was 2.4 mEq. This natriuresis would, in turn, be responsible for sweeping out ≈0.019–0.024 mmol calcium (≈1 mg). The actual excess calcium excretion was ≈0.11 mmol (4.5 mg). The difference (3.5 mg) may be attributable to the sugar. Both Lemann et al (22) and Linderman et al (23) described excess calciuria from oral glucose loads of the magnitude we used, amounting to 0.2–0.3 mmol (8–13 mg), ie, somewhat larger than what we observed with the water meal in our subjects. Hence, it seems likely that the excess calciuria we observed with water was due entirely to this combination of volume expansion (with sodium diuresis) and sugar. The effects of both factors are, like those of caffeine, known to be offset by reduced excretion later in the day (20, 21). Hence, even the relatively small effect seen in our studies would not be expected to contribute to an elevated 24-h calciuria.

By contrast, published data (26, 27) and extensive unpublished observations in our laboratory suggest that the small phosphorus load would lower Ca_u by ≈0.09 mmol (3.6 mg). This well-established hypocalciuric effect of phosphorus was shown to be associated with an increase in digestive juice loss of calcium (28), which was not measured in this study. This is actually the only net effect that, from our data, can be plausibly attributed to the colas.

Our failure to find a significant effect of the acid load was to some extent unexpected. Perhaps it should not have been. The body normally produces 50–100 mEq noncarbonic protons/d in the metabolism of ingested food (29), and the 4.5–5.0-mEq load imposed by the colas in our study is substantially less than would have been produced endogenously by even a moderate-protein breakfast. Even seven 340-mL (12-oz) cola units/d (the maximum consumed by any of our subjects) would impose an acid load of only ≈21 mEq.

Nevertheless, colas have been implicated in at least some observational studies (2, 4), possibly because, if they have a directly harmful effect, they act by means other than increasing urinary calcium loss. If so, our study would not have detected

that effect. It seems more likely, however, that the prominence of colas in the observational studies is due to their prominence in the market. As exemplified in the subjects in our study, consumption of colas is substantially greater than consumption of all other carbonated beverages combined. If carbonated beverages act mainly by displacing more nutritious beverages in the diet, there may not be sufficient power to find significant associations for the other beverages in most published observational studies, thus giving colas a spurious prominence.

Of tangential interest may be the rise in urinary calcium in our study with the milk meal. The calcium load in this meal was ≈11.25 mmol (450 mg), and the absorption fraction at this load for women of this age would be expected to be ≈0.25 (30), meaning that ≈2.8 mmol (112 mg) was absorbed. With excess urinary calcium amounting to ≈0.5 mmol, the milk meal would have resulted in the retention of ≈2.3 mmol calcium. The rise in urinary calcium with milk is thus a reflection, also, of absorption and not in any sense a negative effect; its relative smallness is likely a reflection, also, of the ability of these women with habitually low calcium intakes to store calcium (as bone) when the diet permits.

On a methodologic note, method 2 for estimating excess urinary calcium (incorporating corrections for both creatinine and the water meal) resulted in the smallest absolute variability from person to person and therefore may be the more sensitive approach for studies of this sort. However, the 4 methods each provided slightly different information, and the aggregate of ≥2 approaches may well be more useful. Certainly one may have more confidence in an effect found by all 4 methods.

The absence in this study of calciuric effects of carbonated beverages beyond those known to be offset by reduced excretion later in the day, and the fact that our subjects were habituated to carbonated beverages, leads us to conclude that the principal significance for bone health of the carbonated beverages lies not in what these beverages contain, but in what they do not: the nutrients needed for bone health. Until evidence of other mechanisms of action can be brought forth, the most parsimonious explanation for the association of carbonated beverage intake with poor bone status is milk displacement.

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