Carbonated Beverage Consumption and Bone Mineral Density among Older Women: The Rancho Bernardo Study

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ABSTRACT

Objectives. The association between carbonated beverage consumption and bone mineral density was examined in a community-based cohort of older White women.

Methods. One thousand women 44 to 98 years of age had bone mineral density measured at four sites and provided medical and behavioral histories, including type and quantity of carbonated beverages consumed.

Results. Bone mineral density levels were not associated with intake of any type of carbonated beverage after adjustment for age, obesity, calcium intake, exercise, and current use of tobacco and alcohol, thiazides, estrogen, or thyroid hormone.

Conclusions. Modest intake of carbonated beverages does not appear to have adverse effects on bone mineral density in older women. (*Am J Public Health.* 1997;87:276–279)

Introduction

The per capita consumption of soft drinks in the United States has been increasing for the last 30 years, from 51.7 L (13.6 gal) per year in 1960 to 167.2 L (44.0 gal) in 1990.[1,2] Currently, US consumption of soft drinks surpasses all other beverages, including milk, beer, coffee, and water.[1–4] Therefore, any health risk of soft drinks would have broad public health implications.

Only two studies have directly investigated the relationship between soft drink consumption and osteoporosis. Both found that current consumption of nonalcoholic carbonated beverages, especially colas, was significantly associated with increased fracture risk.[5,6] These authors postulated that the phosphorus (phosphoric acid) content of cola drinks may limit calcium absorption and contribute to bone loss. However, bone density was not measured.

Two other characteristics of carbonated beverages could also affect bone. First, caffeinated carbonated beverages may contribute to bone loss as a result of their caffeine content, which has been associated with reduced bone mineral density[7] and increased fracture risk.[8] Second, high carbonated beverage intake may replace milk consumption and indirectly influence bone mineral density.[9,10] Because milk is a primary source of calcium,[11] this switch from milk to carbonated beverages may lower dietary calcium intake and lead to lower bone mineral density.[12,13]

Considering the factors just discussed, we examined the relation of lifetime and current intake of carbonated beverages to bone mineral density in a community-based cohort of older White women.

Methods

Between February 1988 and February 1992, 1000 women 44 to 98 years of age residing in a middle- to upper-middle-class southern California community, Rancho Bernardo, participated in a study of osteoporosis. Subjects were White and ambulatory, and all but three were postmenopausal. All of the women provided written informed consent.

Participants completed standardized self-administered questionnaires about their medical history and lifestyle, including information about cigarette smoking, alcohol consumption, medication use, exercise, and lifetime consumption of carbonated beverages. They were asked whether there was ever a period in their life when they drank any soft drink every day or almost every day for a year or longer; if so, they were asked to designate the type of carbonated beverages they drank and the number of years they consumed those drinks. Ever drinkers were defined as those who had ever consumed any carbonated beverage daily for a year or longer. Usual daily intake of carbonated beverages, calcium, and phosphorus within the past year was ascertained with the Harvard-Willett Semiquantitative Diet Assessment questionnaire.[14] Current drinkers were defined as those who drank at least one serving (one glass, bottle, or can) per day. All others were classed as nondrinkers/occasional drinkers. Beverages were classified as "any," "diet," "with phosphorus," and "with caffeine." All phosphorus-containing carbonated beverages were cola beverages with phosphoric acid. Other beverages with phosphorus were too infrequently consumed for separate analysis.

Bone mineral density, defined as total bone mineral content divided by area (g/cm^2), was measured at the ultradistal radius and midshaft radius of the nondominant arm by using single photon absorptiometry (Lunar model SP2B; Lunar Corp, Madison, Wis) and at the lumbar spine and hip with dual energy x-ray absorptiom-

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etry (Hologic QDR model 1000; Hologic Inc, Waltham, Mass). Bone mineral density of the ultradistal radius was defined as the lowest mean density of 4 contiguous lines of 10 lines scanned; density of the midshaft radius was defined as the mean of 4 scanned lines; density of the hip was defined as the mean of the greater trochanter, femoral neck, and intertrochanter; and density of the spine was defined as the mean of lumbar vertebrae L1 through L4. Height and weight were measured with participants wearing light clothing and not wearing shoes; body mass index was calculated as weight in kilograms divided by height in meters squared.

The Statistical Analysis System[15] was used in conducting statistical analyses. Multiply adjusted means were assessed through analysis of covariance, and differences in age-adjusted proportions were computed by using the Mantel–Haenszel method. No adjustments were made for multiple comparisons. Statistical significance was determined with 95% confidence intervals or two-tailed *P* values, where appropriate.

TABLE 1—Percentage of Older Women (n = 1000) Who Ever or Currently Drank Carbonated Beverages and Mean Years of Consumption: Rancho Bernardo, Calif., 1988 through 1992

Type of Carbonated Beverage	Ever Drinkers (Daily for ≥1 Year)		Current Drinkers	
	%	Years, Mean (SD)	1 Serving per Day, %	2 to 5 Servings per Day %
Any[a]	23.6	15.1 (14.5)	4.1	2.2
Diet	15.8	14.1 (13.2)	3.4	1.9
With phosphorus	19.7	15.8 (15.1)	3.4	1.7
With caffeine	17.5	16.5 (15.5)	1.5	0.9

[a]Includes all types of carbonated beverages.

Results

The mean age of these women was 72 years (range = 44 to 98). Table 1 shows the number and proportion of women who consumed carbonated beverages and the mean years of consumption.

Table 2 shows the age-adjusted means and proportions of potential confounders of bone mineral density stratified by drinking status of any carbonated beverage. Ever and current drinkers were younger, were heavier for their height, and smoked more than nondrinkers/occasional drinkers. Total and dietary intakes of calcium were lower, but not significantly, in ever and current drinkers than in nondrinkers/occasional drinkers.

Bone mineral density at any site was not associated with ever or current consumption of any carbonated beverage after adjustment for age, body mass index, total calcium intake, exercise, and current use of cigarettes, alcohol, thiazides, estrogen, or thyroid hormone (Table 3). A multivariate model that did not include total calcium intake yielded similar results (data not shown).

Analyses were repeated in ever drinkers stratified by mean years of carbonated beverage consumption. There were no significant differences in bone mineral density levels among ever drinkers who reported drinking any carbonated beverage for fewer than 10 years (n = 140), 10 to 20 years (n = 43), 20 to 30 years (n = 19), or more than 30 years (n = 28). Analyses by the amount of carbonated beverages current drinkers consumed also revealed no significant associations with bone mineral density. Current drinkers who drank one serving per day (n = 41) did not differ in their bone mineral density levels from those who consumed two to five servings per day (n = 22). As a means of determining whether carbonated beverage intake had an effect in women with low calcium intake, the analyses were repeated for 105 women whose total daily calcium intake was below 400 mg. In these women, bone mineral density did not differ by ever or current drinker status. Finally, analyses were performed on the 625 postmenopausal women who were not currently using estrogen replacement therapy to determine whether carbonated beverage intake had an effect in women with low estrogen levels. No significant associations were found (data not shown).

All of the adjusted and stratified analyses just described were repeated for ever or current drinkers of diet, phosphorus-containing, or caffeinated carbonated beverages. No associations were found (data not shown).

Discussion

In this community-based study of older adults, there were no significant associations between bone mineral density levels and lifetime or current consumption of carbonated beverages. Analyses by caffeinated or phosphorus-containing carbonated beverages or analyses restricted to women with low calcium intake or no estrogen replacement therapy yielded similar results.

A possible limitation of this study is that food frequency questionnaires are imperfect tools. Nevertheless, soft drink consumption is likely to be better recalled than consumption of other food items because soft drinks are packaged in standard, discrete containers. Another possible limitation of this study is that participants may not have consumed enough soft drinks to demonstrate an impact on bone mineral density. Overall consumption of carbonated beverages was low; 65% of current drinkers reported drinking only one serving per day. A single 12-oz (336-g) can of a caffeinated carbonated beverage contains a maximum of 54 mg of caffeine.[16] The same amount of brewed coffee yields up to 206 mg.[16] In proportion, the caffeine contribution of one caffeinated carbonated beverage is small.

Similarly, although investigators usually attribute a deleterious effect of carbonated beverages to phosphorus content,[5,6,16] the phosphorus contribution of carbonated beverages is minor in comparison with the overall amount present in the usual American diet. A single can of phosphorus-containing soft drink yields between 26 and 76 mg of phosphorus,[17] while the amount of phosphorus in unprocessed foods is about 1430 to 1520 mg per capita per day,[18] with an additional 500 to 1000 mg[3] added for processing. In this cohort, the average dietary phosphorus intake was 1167 mg and did not significantly differ with drinking status of phosphorus-containing carbonated beverages.

Nevertheless, the concern over high phosphorus intake may be applicable when calcium intake is very low. In

TABLE 2—Age-Adjusted Estimates of Potential Confounders of Bone Mineral Density in Older Women, by Ever or Current Consumption of Any Carbonated Beverage: Rancho Bernardo, Calif, 1988 through 1992

Potential Confounder	Ever		Current	
	Nondrinkers or Occasional Drinkers (n = 764)	Drinkers (Daily for ≥1 Year) (n = 236)	Nondrinkers or Occasional Drinkers (n = 937)	Drinkers (≥1 Serving per Day) (n = 63)
Age, y, mean (95% CI)	73.6 (72.9, 74.3)	68.5 (67.3, 69.7)	72.8 (72.2, 73.4)	66.3 (64.0, 68.6)
Body mass index, kg/m², mean (95% CI)	24.4 (24.1, 24.7)	25.3 (24.8, 25.8)	24.5 (24.2, 24.8)	25.9 (24.9, 26.9)
Daily mineral intake, mg				
Total calcium intake (dietary and supplement)	998.6 (956.1, 1041.1)	908.9 (832.3, 985.5)	980.4 (942.1, 1018.7)	934.3 (794.5, 1074.1)
Dietary calcium	688.7 (660.9, 716.5)	621.2 (571.1, 671.3)	674.1 (649.0, 699.2)	651.8 (560.2, 743.4)
Dietary phosphorus	1182.2 (1147.9, 1216.5)	1118.9 (1057.1, 1180.7)	1163.4 (1132.5, 1194.3)	1214.2 (1101.4, 1327.0)
Ever cigarette smoker (≥100),[a] %	46.9	58.1*	48.5	68.1*
Current cigarette smoker, %	8.9	15.3	10.6	7.9
Current alcohol use (any in past 30 days), %	74.8	74.6	74.4	81.0
Current thiazide use, %	23.4	20.3	22.6	23.8
Current estrogen replacement therapy use, %	35.5	44.1	37.0	44.4
Current thyroid hormone use, %	20.6	17.0	19.6	20.6
Current exercise (≥3 times per week), %	66.4	65.7	66.1	68.3

Note. CI = confidence interval.
[a]≥100 cigarettes smoked per lifetime.
*$P < .05$ (for age-adjusted proportions relative to nondrinkers and occasional drinkers).

TABLE 3—Adjusted Bone Mineral Densities (g/cm²) (95% CIs) in Older Women, by Ever or Current Consumption of Any Carbonated Beverage: Rancho Bernardo, Calif., 1988 through 1992

Measurement Site	Ever		Current	
	Nondrinkers or Occasional Drinkers (n = 764)	Drinkers (Daily for ≥1 Year) (n = 236)	Nondrinkers or Occasional Drinkers (n = 937)	Drinkers (≥1 Serving per Day) (n = 63)
Ultradistal radius	0.236 (0.231, 0.241)	0.241 (0.232, 0.250)	0.237 (0.233, 0.241)	0.241 (0.223, 0.259)
Midshaft radius	0.598 (0.592, 0.604)	0.602 (0.590, 0.614)	0.599 (0.593, 0.605)	0.599 (0.576, 0.622)
Total hip	0.783 (0.775, 0.791)	0.785 (0.770, 0.800)	0.785 (0.778, 0.792)	0.764 (0.735, 0.793)
Lumbar spine	0.893 (0.881, 0.905)	0.910 (0.888, 0.932)	0.899 (0.889, 0.909)	0.863 (0.822, 0.904)

Note. Means for bone mineral density were adjusted for age, body mass index, total calcium intake, exercise, and current use of cigarettes, alcohol, thiazides, estrogen, or thyroid hormone. CI = confidence interval.

animal models, high-phosphorus, low-calcium intakes have been shown to cause secondary hyperparathyroidism and increased bone resorption, leading to osteoporosis.[19] Furthermore, carbonated beverages may contribute to the high-phosphorus, low-calcium diet by reducing calcium intake.[9,10,19] In this cohort, carbonated beverage drinkers had modest but not significantly lower calcium intakes. Analyses stratified by total calcium intake or not adjusted for calcium showed no significant association between carbonated beverages and bone mineral density levels.

There may be characteristics of carbonated beverage drinkers that are beneficial to bone. For example, carbonated beverage drinkers were younger and heavier than nondrinkers, and both variables were positively associated with bone mineral density. After adjustment for age and body mass index, no association between carbonated beverages and bone mineral density was seen; however, some other factor could have been operative. For example, Wyshak et al.[5,6] suggested that very active women (e.g., elite athletes) who consume carbonated beverages may be at a greater fracture risk than more sedentary women because their lower levels of endogenous estrogen increase susceptibility. Few women in this cohort engaged in such strenuous activity. In addition, analyses restricted to postmenopausal women not using estrogen showed no association of carbonated beverages with bone mineral density. In conclusion, these data provide no evidence that moderate intake of carbonated beverages has any adverse effect on bone mineral density levels in older women. □

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References

1. *Statistical Abstract of the United States: 1986.* 106th ed. Washington, DC: US Bureau of the Census; 1986.
2. *Statistical Abstract of the United States: 1994.* 114th ed. Washington, DC: US Bureau of the Census; 1994.
3. Ensminger AH, Ensminger ME, Konlande JE, Robson JRK. *Foods & Nutrition Encyclopedia.* 2nd ed. Boca Raton, Fla: CRC Press; 1994.
4. Maxwell JC. Annual soft drink report. *Beverage Industry.* 1994;85:4–20.
5. Wyshak G, Frisch RE, Albright TE, Albright NL, Schiff I, Witschi J. Nonalcoholic carbonated beverage consumption and bone fractures among women former college athletes. *J Orthop Res.* 1989;7: 91–99.
6. Wyshak G, Frisch RE. Carbonated beverages, dietary calcium, the dietary calcium/phosphorus ratio, and bone fractures in boys and girls. *J Adolesc Health.* 1994;15: 210–215.
7. Barrett-Connor E, Chang JC, Edelstein SL. Coffee-associated osteoporosis offset by daily milk consumption. *JAMA.* 1994;271: 280–283.
8. Keil DP, Felson DT, Hannan MT, Anderson JJ, Wilson PWF. Caffeine and the risk of hip fracture: the Framingham study. *Am J Epidemiol.* 1990;132:675–684.
9. Smith S, Swain J, Brown EM, et al. A preliminary report of the short-term effect of carbonated beverage consumption on calcium metabolism in normal women. *Arch Intern Med.* 1989;149:2517–2519.
10. Massey LK, Strang MM. Soft drink consumption, phosphorus intake, and osteoporosis. *J Am Diet Assoc.* 1982;80:581–583.
11. Phillips MC, Briggs GM. Milk and its role in the American diet. *J Dairy Sci.* 1975;58: 1751–1753.
12. Heaney RP. The role of nutrition in prevention and management of osteoporosis. *Clin Obstet Gynecol.* 1987;30:833–846.
13. Einhorn TA, Levine B, Michel P. Nutrition and bone. *Orthop Clin North Am.* 1990;21: 43–50.
14. Willett WC, Sampson L, Stampfer MJ, et al. Reproducibility and validity of a semiquantitative food frequency questionnaire. *Am J Epidemiol.* 1985;122:51–65.
15. *SAS/STAT User's Guide, Version 6.03.* Cary, NC: SAS Institute Inc; 1988.
16. Mazariegos-Ramos E, Guerrero-Romero F, Rodriquez-Moran M, Lazcano-Burciaga G, Paniagua R, Amato D. Consumption of soft drinks with phosphoric acid as a risk factor for the development of hypocalcemia in children: a case-control study. *J Pediatr.* 1995;126:940–942.
17. Pennington JAT. *Bowes & Churches Food Values of Portions Commonly Used.* 16th ed. Philadelphia, Pa: JB Lippincott; 1994.
18. National Research Council. *Recommended Dietary Allowance.* 10th ed. Washington, DC: National Academy Press; 1989:174–194.
19. Calvo MS. The effects of high phosphorus intake on calcium homeostasis. In: Draper HH, ed. *Advances in Nutritional Research.* New York, NY: Plenum Press; 1994;9:183–207.

Transmission of *Mycobacterium tuberculosis* in a California State Prison, 1991

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ABSTRACT

Objectives. An investigation was conducted to determine whether ongoing transmission of *Mycobacterium tuberculosis* was occurring in a California state prison.

Method. Prison pharmacy records were used to identify cases of active tuberculosis (TB).

Results. Ten of the 18 cases of active TB treated at the facility during 1991 were diagnosed at the prison that same year (an incidence of 184 per 100 000). Three inmates were infectious for a total of 7 months while imprisoned. The prevalence of TB skin test–positivity among inmates was 30%, and the incidence of new infection attributable to incarceration was 5.9 per 100 inmates per year.

Conclusions. Transmission of *M. tuberculosis* may be occurring in the California prison system. (*Am J Public Health.* 1997;87:279–282)

Introduction

The control of tuberculosis (TB) within prisons and jails has been a long-standing concern.[1–5] Correctional institutions house large numbers of persons at high risk for tuberculosis, including persons of lower socioeconomic status, persons from inner cities where rates of *Mycobacterium tuberculosis* infection are higher, and persons co-infected with human immunodeficiency virus (HIV).[6–11] In addition, close quarters, poor ventilation, and overcrowding in these institutions facilitate transmission. *M. tuberculosis* infection has been found to be associated with increased time in or admission to a large city jail system.[12]

In fall 1991, three cases of active TB were diagnosed at a California state prison, two among inmates and one in a prison employee. A study was conducted to determine whether transmission of *M. tuberculosis* was occurring at this facility.

Methods

Because individual medical records do not remain at the prison, we identified cases of active TB at the state prison in 1991 by checking pharmacy records for inmates taking at least two anti-TB medications and checking laboratory culture results for specimens positive for *M. tuberculosis.* TB cases were defined by using the Centers for Disease Control and Prevention surveillance case definition for

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