Relative bioavailability of calcium-rich dietary sources in the elderly[1–4]

Ligia Martini and Richard J Wood

ABSTRACT

Background: The recent increase in the dietary calcium recommendation from 800 to 1200 mg/d for persons aged >51 y has made it important to identify alternative high-calcium dietary sources that the elderly can use in meeting their calcium requirement.

Objective: We determined the bioavailability of calcium from 3 different sources: orange juice fortified with calcium-citrate malate, skim milk, and a calcium carbonate supplement.

Design: Twelve subjects [9 women and 3 men with a mean (±SEM) age of 70 ± 3 and 76 ± 6 y, respectively] consumed low-calcium (300 mg/d) and high-calcium (1300 mg/d) diets for three 1-wk periods each during a 6-wk crossover study. The acute biochemical response to calcium from each of the 3 sources was assessed during a 4-h period after the initial breakfast meal of the high-calcium diet.

Results: Postprandial suppression of serum parathyroid hormone did not differ significantly between the test meals containing calcium-fortified orange juice, the calcium carbonate supplement, and milk. This finding suggests that the calcium bioavailability from the 3 sources was equivalent. During the 1-wk high-calcium diet periods, fasting serum calcium increased by 3% ($P < 0.0001$), serum 1,25-dihydroxyvitamin D decreased by 20% ($P < 0.0001$), and a biomarker of bone resorption (serum *N*-telopeptide collagen cross-links) decreased by 14% ($P < 0.02$) compared with the low-calcium diet period. However, no differences among the supplemental calcium sources were found in these calcium-responsive measures or fasting serum parathyroid hormone concentration.

Conclusions: In elderly subjects, the calcium bioavailability of the 3 high-calcium dietary sources tested was equivalent, during both the acute postprandial and longer-term periods. *Am J Clin Nutr* 2002;76:1345–50.

KEY WORDS Calcium-fortified foods, calcium supplements, calcium-citrate malate, calcium carbonate, PTH suppression test, bone resorption, elderly, calcium bioavailability, parathyroid hormone

INTRODUCTION

A large body of research suggests that a high calcium intake throughout life is necessary to attain peak bone mass and to minimize bone loss during aging (1). In 1997, the Food and Nutrition Board of the Institute of Medicine (1) recommended a daily intake of 1200 mg (30 mmol) dietary calcium to reduce the risk of osteoporosis in all men and women >51 y of age.

However, it is difficult for many people to consume diets supplying the recommended calcium intakes of 25–32.5 mmol/d (1000–1300 mg/d) (1), which are needed to achieve optimal bone density and protect against bone loss. In the United States, national dietary survey data (2–4) have consistently shown that calcium intakes for most groups are far below recommended amounts throughout most of childhood and adulthood, especially in the elderly, a group at high risk of developing osteoporosis. In addition, aging can cause physiologic changes, such as hypochlorhydria (5), intestinal resistance to vitamin D (6, 7), or estrogen deficiency in postmenopausal women (8), all of which may alter intestinal calcium absorption.

Given that the usual intakes of dietary calcium in the United States are far below recommended amounts for most elderly persons, increased use of traditional calcium-rich foods (such as milk and other dairy products) to increase dietary calcium intakes appears imperative.

However, there may be personal, physiologic, and cultural reasons why some elderly persons find it difficult to increase dairy product consumption. For example, it is generally well known that lactose malabsorption is a common problem in nonwhite populations, affecting most blacks and Asians (9), but it is less appreciated that the prevalence of lactose intolerance is quite high among white elderly persons. Therefore, for many elderly persons in the United States and elsewhere, calcium-rich dietary sources that are alternatives to increased milk consumption, such as calcium-fortified nondairy products and calcium supplements, will be relied on increasingly to meet current dietary calcium recommendations. The bioavailability of calcium from different sources can vary significantly (10–12). Little information is available about the relative bioavailability of calcium from different commercial products, especially for elderly people, who may have compromised ability to absorb different forms of calcium (7, 13, 14).

[1] From the Mineral Bioavailability Laboratory, Jean Mayer US Department of Agriculture Human Nutrition Research Center on Aging at Tufts University, Boston.

[2] The contents of this publication do not necessarily reflect the views or policies of the US Department of Agriculture, nor does mention of trade names, commercial products, or organizations imply endorsement by the US government.

[3] Supported by the US Department of Agriculture, Agricultural Research Service cooperative agreement 1950-51520-006-00D. LM was supported in part by a postdoctoral fellowship grant from Fundacao de Amparo a Pesquisa do Estado de Sao Paulo (FAPESP), Brazil, under agreement no. 98/6151-9.

[4] Address reprint requests to RJ Wood, Mineral Bioavailability Laboratory, USDA Human Nutrition Research Center on Aging at Tufts University, 711 Washington Street, Boston, MA 02111. E-mail: rwood@hnrc.tufts.edu.

Received September 10, 2001.

Accepted for publication January 10, 2002.

Thus, it is important in elderly persons to document the adequacy of calcium bioavailability from sources that are intended to provide a substantial portion of the dietary calcium requirement. A direct measure of calcium absorption from various foods can be achieved by labeling them with radioactive or stable isotopes of calcium (12, 15, 16). However, these studies can be extremely costly, and in the case of stable isotopes, they require highly sophisticated analytic capability. In addition, there are difficulties in using isotopic labeling approaches when testing commercial products, such as dietary supplements or calcium-fortified foods (17). Therefore, indirect methods that reflect relative calcium bioavailability (absorption) but do not depend on the use of calcium isotopes may be useful for comparative purposes, especially in whole foods or proprietary preparations. These indirect methods involve evaluating calcium-responsive biochemical endpoints relevant to bone and mineral metabolism, such as serum calcium or parathyroid hormone (PTH) (18, 19) or measures of urinary bone resorption markers (20). Although indirect estimates of the amount of absorbed calcium, these measures of relative calcium bioavailability do focus on relevant biochemical endpoints and biomarkers of important physiologic processes in the skeleton. Moreover, these methods can be applied easily to the study of reference populations, such as the elderly.

The aim of this study, conducted in elderly men and women, was to compare the relative bioavailability of calcium from 3 dietary sources: a commercial calcium-fortified beverage (orange juice), a calcium carbonate dietary supplement, and milk. We compared relative calcium bioavailability on the basis of both a commonly used acute postprandial PTH suppression test (10, 18, 19, 21–23) and measures of bone and calcium metabolism obtained during longer periods (7 d) of high calcium consumption within a 6-wk crossover study conducted in a metabolic unit.

SUBJECTS AND METHODS

Subjects

Twelve elderly volunteer subjects (3 men and 9 women) participated in the metabolic study. All the women were postmenopausal and none were receiving estrogen replacement therapy. All subjects were in good health as determined by a medical history, physical examination, and clinical laboratory analyses completed before enrollment. Potential subjects were excluded from participation in this study if they presented at screening with significant cardiovascular, hepatic, gastrointestinal, or renal disease or diabetes, or if they were smokers or had a history of lactose malabsorption or milk intolerance. A 3-d dietary record was obtained before the study began to estimate the subjects' usual calcium intakes. The study was approved by the Tufts University Human Investigational Research Committee and all subjects provided informed, written consent.

Study design

A 6-wk crossover trial was conducted at the Human Study Unit of the Jean Mayer USDA Human Nutrition Research Center on Aging at Tufts University in Boston. Throughout the study, all subjects resided exclusively in the Human Study Unit. For the entire 6-wk study, subjects consumed a standardized low-calcium basal diet providing 300 mg (7.5 mmol) Ca/d. During 3 wk of the study, calcium intake was increased by 1000 mg (25 mmol) daily. Each of these three 1-wk high-calcium treatment periods was preceded by a 1-wk low-calcium diet period during which the diet was exactly the same, except that the supplemental calcium sources were excluded.

During the 3 high-calcium treatment periods, the increase in calcium intake was achieved by providing 500 mg additional Ca at breakfast (0800–0900) and at dinner (1700–1800). The source of the additional calcium was skim milk, a calcium carbonate dietary supplement (Os-Cal; Smith-Kline-Beecham, Pittsburgh), or calcium-fortified orange juice (fortified with calcium-citrate malate; Tropicana, Bradenton, FL). The calcium contents of the milk and calcium-fortified orange juice were determined by direct-current plasma emission spectroscopy. To provide 500 mg Ca, it was necessary to administer slightly different volumes of orange juice and milk. The volumes were 0.317 L (≈11 oz) orange juice and 0.430 L (≈15 oz) skim milk. To maintain a constant volume of liquid intake during the different periods of the study, 0.430 L distilled water was added to the breakfast and dinner meals during the 3 weeks of the low-calcium diet and during the 1-wk calcium carbonate supplement treatment. Some additional distilled water was also provided during the orange juice period to equalize the liquid volume between treatments.

Assessment of relative calcium bioavailability

Acute postprandial effects of calcium supplementation

Relative calcium bioavailability from the 3 supplemental calcium sources (each of which provided an oral dose of 500 mg Ca) was evaluated indirectly by monitoring the relative suppression of serum PTH (PTH suppression test) and measuring postprandial changes in serum and urinary calcium and urinary collagen type I *N*-telopeptide cross-links (NTX) during a 4-h period after consumption of the test breakfast. NTX is a biomarker of bone resorption activity (24, 25). Serum and urinary phosphorous concentrations were also measured.

The PTH suppression test, similar to that described by Guillemant and Guillemant (18), was conducted on 3 separate occasions during the study, at the first meal (breakfast) after initiation of each of the 3 high-calcium treatment periods in weeks 2, 4, and 6. During each preceding week (weeks 1, 3, and 5), all subjects consumed only the low-calcium basal diet to elevate serum PTH and maximize the sensitivity of the acute test to the PTH-suppressing ability of absorbed calcium. The standard test meals during each PTH suppression test were identical and supplied 90 mg Ca. Each test meal consisted of 40 g wheat bread, 12 g jelly, 7 g butter, and 1 serving of vegetable scramble [115 g imitation eggs (Egg Beaters; Beatrice Foods, Downers Grove, IL), 30 g mushrooms, 30 g green peppers, 7 g soybean oil, 0.4 g salt, and 0.1 g black pepper]. This low-calcium test meal was supplemented with 500 mg Ca as skim milk, calcium-fortified orange juice, or a single calcium carbonate dietary supplement tablet, as appropriate. The order of treatments for all subjects was randomized, and each subject received all 3 treatments. The volume of liquid consumed at each of the test meals was kept constant by adding an appropriate amount of distilled water when necessary. No attempt was made to equalize the intake of other nutrients supplied by the added milk or orange juice. The nutrient contents of the test meals used in the 3 PTH suppression tests are shown in **Table 1**.

Longer-term effects of calcium supplementation

The acute postprandial effects of a single oral calcium load on various biochemical markers could differ from the longer-term response to increases in dietary calcium after repeated administration of the supplemental calcium. Therefore, we also estimated the relative calcium bioavailability from the 3 calcium sources, assessing the effects on bone and mineral metabolism by measuring

TABLE 1
Nutrient content of calcium-supplemented test meals used for the parathyroid hormone suppression tests

	Test meal[1] + milk[2]	Test meal + orange juice[3]	Test meal + calcium carbonate[4]
Energy (kcal)	462	445	312
Fat (g)	15.2	14.8	14.4
Carbohydrate (g)	53.0	63.7	32.5
Protein (g)	29.6	16.7	14.9
Calcium (mg)	590	590	590
Phosphorus (mg)	536	145	101

[1] All test meals consisted of bread, jelly, butter, imitation eggs, mushrooms, green peppers, soybean oil, salt, and black pepper and contained 90 mg Ca and 101 mg P. Supplemental calcium (500 mg) from various sources was added to each test meal.
[2] 0.430 L skim milk.
[3] 0.317 L calcium-fortified orange juice (Tropicana, Bradenton, FL).
[4] One 500-mg Ca supplement (OsCal; Smith-Kline-Beecham, Pittsburgh).

various calcium-responsive biochemical indexes after 1 wk of calcium supplementation. The effects of calcium supplementation on mineral and bone metabolism were assessed by measuring serum calcium, phosphorous, intact $PTH_{1–84}$, 25-hydroxyvitamin D, 1,25-dihydroxyvitamin D, and NTX and urinary calcium, phosphorous, and NTX corrected for creatinine excretion.

Blood and urine measurements

At the end of each study period, blood samples were drawn in the morning after an overnight fast and then hourly for 4 h postprandially during the PTH suppression test. The serum was separated and frozen at −80 °C until assayed. Except for during the PTH suppression test, urine samples were collected for 24-h periods. During the last 3 d of each study period, the daily urine samples were combined into a 3-d pool and a representative aliquot was retained for subsequent analysis. During the PTH suppression test, urine was collected for three 2-h periods: −2 h to 0 h, 0 h to 2 h, and 2 h to 4 h. Urinary calcium was measured by using direct-current argon plasma emission spectroscopy. Serum calcium and phosphorus and urinary creatinine were measured with standard automated colorimetric assays. Intact serum PTH was measured with an immunoradiometric assay (Nichols Institute Diagnostics, San Juan Capistrano, CA); the intraassay CV was 4%. Serum 1,25-dihydroxyvitamin D_3 and 25-hydroxyvitamin D were measured with a specific radioimmunoassay kit (DiaSorin, Stillwater, NM) after extraction of the serum with acetonitrile; the intraassay CV was 8%. Serum and urinary NTX were measured with an enzyme-linked competitive inhibition immunosorbent assay kit (Ostex International, Seattle); the CV was 8%. Vitamin D deficiency was defined as a serum 25-hydroxyvitamin D concentration < 10 ng/mL (< 25 nmol/L) (26).

Statistical analysis

Unless otherwise noted, the data are expressed as means ± SEMs. For the purpose of statistical analysis, the area-under-the-curve (AUC) procedure was used to summarize serial postprandial measures. The main effects of the different calcium sources during the crossover study were evaluated with the general linear model analysis of variance with separate hypothesis testing of any sequence effect (SYSTAT version 9 for WINDOWS; SPSS Inc, Chicago). If a significant effect of calcium source was evident (F test < 0.05), then the individual differences between calcium sources were compared by using the general linear model Tukey pairwise comparisons procedure. Statistical analysis for the longer-term calcium study was performed on the basis of analysis of covariance of the differences in measures between the preceding low-calcium diet period and the various high-calcium periods of the study, with the low-calcium baseline value included as the covariate.

RESULTS

Subject characteristics

The mean (± SEM) age of the women was 70 ± 3 y and of the men was 76 ± 6 y. The mean body weight of the women was 62 ± 3 kg and of the men was 69 ± 3 kg. On the basis of a 3-d dietary record obtained before the start of the study, the reported mean calcium intake was 752 ± 106 mg/d for women and 1022 ± 240 mg/d for men. None of the subjects had a serum 25-hydroxyvitamin D concentration < 25 nmol/L, which would indicate vitamin D deficiency.

Effects of supplemental calcium source on measures of bone and mineral metabolism

Acute postprandial effects of calcium supplementation

First, we investigated whether there were any significant differences in the effects of the different supplemental calcium sources on the measured variables during an acute 4-h postprandial period of observation (the PTH suppression test). In **Table 2**, the mean values for serum calcium, phosphorous, and PTH and urinary calcium, phosphorous, and NTX during the PTH suppression test are shown. For serum calcium concentration, there was no significant postprandial change, on the basis of calculated AUC values for the percentage change in serum calcium. The AUC for serum calcium was −0.6 (95% CI: −0.1, −1) for milk, −0.1 (95% CI: 0.6, −0.8) for orange juice, and −0.03 (95% CI: 0.42, −0.47) for the calcium carbonate supplement.

However, the acute oral load of 500 mg Ca consumed with the test meal did cause the expected postprandial suppression of serum PTH after each of the 3 different test meals. The mean AUC for the percentage change in serum PTH from baseline was −21 (−5, −38) for milk, −33 (−15, −50) for orange juice, and −33 (−18, −49) for calcium carbonate. Likewise, there was a postprandial increase in urinary calcium corrected for creatinine excretion. The mean AUC for the percentage change in urinary calcium corrected for creatinine excretion was 147 (50, 245) for milk, 136 (52, 221) for orange juice, and 102 (54, 149) for calcium carbonate. There was no significant postprandial change in urinary NTX corrected for creatinine excretion during the 4-h PTH suppression test compared with the 2-h baseline fasting urinary NTX concentration. The mean AUC for urinary NTX (corrected for creatinine excretion) was −8 (−30, 14) for milk, 10 (−14, 34) for orange juice, and 2 (−27, 31) for calcium carbonate. The calcium source had no effect on the 4-h postprandial serum concentrations of calcium or PTH or the urinary excretion of calcium or NTX.

There was a significant difference (P < 0.001, analysis of covariance) between the supplemental calcium sources in the AUC for percentage change in serum phosphorous concentration after the test meals. The AUC for serum phosphorous was significantly greater after the test meal supplemented with milk compared with the test meals supplemented with orange juice and calcium carbonate (P < 0.003 and P < 0.001, respectively; Tukey's test). The mean AUC

TABLE 2
Acute postprandial changes in serum calcium, phosphorus, and intact parathyroid hormone (PTH) and urinary calcium, phosphorus, and collagen type I *N*-telopeptide cross-links (NTX) in elderly subjects after different high-calcium test meals[1]

	Serum calcium[2]	Serum phosphorus[3,4]	Serum PTH[2,4]	Urinary calcium[2,4]	Urinary phosphorus[4,5]	Urinary NTX[2]
	mmol/L	*mmol/L*	*pmol/L*	*nmol/mmol Cr*	*nmol/mmol Cr*	*nmol BCE/nmol Cr*
Calcium carbonate tablet						
Baseline[6]	2.29 ± 0.017	1.23 ± 0.03	3.66 ± 0.32	263 ± 56	1968 ± 168	77 ± 14
1 h postprandial	2.27 ± 0.017	1.13 ± 0.02	2.90 ± 0.21			
2 h postprandial	2.32 ± 0.024	1.06 ± 0.03	2.37 ± 0.11	291 ± 73	2203 ± 200	81 ± 14
3 h postprandial	2.29 ± 0.017	1.09 ± 0.04	2.47 ± 0.21			
4 h postprandial	2.32 ± 0.022	1.13 ± 0.02	2.80 ± 0.32	605 ± 87	1478 ± 193	78 ± 17
Milk						
Baseline[6]	2.27 ± 0.019	1.23 ± 0.03	3.66 ± 0.32	254 ± 65	1971 ± 189	85 ± 12
1 h postprandial	2.22 ± 0.032	1.19 ± 0.05	3.12 ± 0.22			
2 h postprandial	2.22 ± 0.032	1.16 ± 0.04	2.90 ± 0.22	379 ± 85	2833 ± 252	75 ± 12
3 h postprandial	2.24 ± 0.022	1.23 ± 0.05	2.90 ± 0.22			
4 h postprandial	2.27 ± 0.030	1.25 ± 0.04	2.90 ± 0.22	568 ± 107	2392 ± 227	56 ± 7
Calcium-fortified orange juice						
Baseline[6]	2.29 ± 0.014	1.19 ± 0.03	3.55 ± 0.32	286 ± 59	1817 ± 227	74 ± 12
1 h postprandial	2.27 ± 0.017	1.16 ± 0.03	2.69 ± 0.21			
2 h postprandial	2.27 ± 0.024	1.03 ± 0.04	2.26 ± 0.22	385 ± 76	2529 ± 249	74 ± 10
3 h postprandial	2.29 ± 0.017	1.06 ± 0.03	2.15 ± 0.22			
4 h postprandial	2.32 ± 0.014	1.13 ± 0.03	2.58 ± 0.11	639 ± 99	1274 ± 182	63 ± 9

[1] $\bar{x}$ ± SEM; n = 12. Cr, creatinine; BCE, bone collagen equivalent.
[2] There was no significant effect of calcium source.
[3] Serum phosphorus area under the curve was significantly higher with milk than with calcium-fortified orange juice ($P < 0.003$, Tukey's test) and calcium carbonate ($P < 0.001$, Tukey's test).
[4] The percentage postprandial change was significantly different from zero, $P < 0.05$ (Tukey's pairwise comparisons).
[5] Area under the curve for urinary phosphorus (corrected for creatinine excretion) was significantly higher with milk than with calcium-fortified orange juice and calcium carbonate, $P < 0.002$ (Tukey's pairwise comparisons).
[6] Baseline was before the meal (t = 0 h) for blood measures and was t = –2 to 0 h for urinary measures.

for percentage change in serum phosphorous was −0.2 (0.3, −0.7) for milk, −1.2 (−0.7, −1.6) for orange juice, and −1.4 (−0.9, −1.8) for calcium carbonate. These differences in serum phosphorous probably simply reflect the higher oral phosphorous load associated with milk consumption (Table 1). This difference was also seen in the urinary phosphorous concentration corrected for creatinine excretion; the mean AUC was 36 (14, 59) for milk, 11 (−9, 32) for orange juice, and −7 (−20, 6) for calcium carbonate.

Longer-term effects of calcium supplementation

We evaluated the effects of the different calcium sources during 1-wk periods of calcium supplementation. As shown in **Table 3**, on the basis of an analysis of covariance of the difference between the low-calcium and high-calcium diet periods, consumption of an additional 1000 mg Ca/d during three 1-wk periods increased fasting serum calcium by 3% ($P < 0.0001$), decreased serum 1,25-dihydroxyvitamin D by 20% ($P < 0.0001$), and decreased serum NTX by 14% ($P < 0.02$). However, there were no significant differences in these responses among the 3 different supplemental calcium sources.

DISCUSSION

In the present study, we found that consumption of a standardized low-calcium test meal together with 500 mg supplemental Ca caused the expected postprandial suppression of serum PTH concentration in elderly subjects. The acute drop in serum PTH reflects a homeostatic adaptation of the parathyroid gland, mediated by its calcium-sensing receptor (27), in response to a transient postprandial increase in plasma calcium; this postprandial increase resulted from absorption of the calcium in the test meal.

The calcium-dependent postprandial decrease in PTH has been well described by others (18, 19) and is the basis of the PTH suppression test for measuring calcium bioavailability. Although we did not study the postprandial biochemical responses of these subjects in the absence of a high calcium load, studies by others have shown that there is little change in either serum calcium or PTH in the absence of a calcium load (10). We did not observe the expected acute postprandial rise in serum calcium in our subjects after consumption of the calcium-containing test meal. The absence of a serum calcium response to the oral calcium load was probably a result of the very small changes that occur in this measure after a calcium load (Table 2). Another possible explanation is that we measured total serum calcium, rather than ionic calcium concentrations, as some others have done (10, 23). Nevertheless, the observed postprandial decline in PTH, a 29% decrease overall, in our elderly subjects suggests that the calcium from the test meal was absorbed within the time frame of the acute 4-h postprandial study. The overall absorbability of calcium from the supplemental sources was further confirmed by the marked postprandial increase in the rate of urinary calcium excretion, a 53% increase overall.

We did not find a significantly different acute response to milk, as opposed to the other supplemental calcium sources, in the elderly subjects in our study. Other investigators have reported a relatively attenuated acute postprandial suppressing effect of milk on serum PTH

TABLE 3
Serum and urinary measures in 12 elderly subjects during 1-wk periods of consumption of a basal low-calcium (300 mg/d) diet and 3 different high-calcium (1300 mg/d) diets consisting of the basal diet plus 1000 mg supplemented Ca/d from either milk, calcium-fortified orange juice, or a calcium carbonate tablet[1]

	Calcium carbonate tablet	Milk	Calcium-fortified orange juice
Serum calcium			
LCD (mmol/L)	2.30 ± 0.02[2]	2.28 ± 0.02	2.29 ± 0.02
HCD (mmol/L)	2.36 ± 0.02	2.34 ± 0.02	2.38 ± 0.02
Change (%)[3]	3	3	4
Serum phosphorus			
LCD (mmol/L)	1.23 ± 0.03	1.22 ± 0.04	1.22 ± 0.03
HCD (mmol/L)	1.25 ± 0.03	1.25 ± 0.03	1.28 ± 0.03
Change (%)	2	2	5
Serum PTH			
LCD (ng/L)	34.1 ± 2.7	31.9 ± 2.4	34.6 ± 3.6
HCD (ng/L)	33.4 ± 4.0	32.7 ± 2.8	30.7 ± 2.8
Change (%)	−2	2	−11
Serum 1,25-$(OH)_2$ vitamin D			
LCD (pmol/L)	107 ± 5	112 ± 1	112 ± 10
HCD (pmol/L)	92 ± 8	82 ± 8	89 ± 6
Change (%)[3]	−14	−27	−20
Serum 25-(OH) vitamin D			
LCD (nmol/L)	57 ± 3	58 ± 3	56 ± 5
HCD (nmol/L)	54 ± 2	54 ± 3	54 ± 4
Change (%)	−5	−7	−3
Serum NTX			
LCD (nmol BCE/L)	23.7 ± 2.1	22.0 ± 2.2	21.0 ± 1.8
HCD (nmol BCE/L)	19.4 ±2	18.0 ± 1.5	18.6 ± 1.4
Change (%)[4]	−18	−18	−11
Urinary calcium			
LCD (nmol/mmol Cr)	277 ± 48	291 ± 53	265 ± 47
HCD (nmol/mmol Cr)	414 ± 71	428 ± 71	434 ± 77
Change (%)	49	47	64
Urinary phosphorus			
LCD (nmol/mmol Cr)	2347 ± 166	2284 ± 137	2292 ± 23
HCD (nmol/mmol Cr)	1738 ± 86	3150 ± 189	1626 ± 171
Change (%)	−26	38	−29
Urinary NTX			
LCD (nmol BCE/mmol Cr)	51 ± 7	58 ± 8	56 ± 6
HCD (nmol BCE/mmol Cr)	45 ± 7	45 ± 5	43 ± 5
Change (%)	−12	−22	−23

[1] Cr, creatinine; PTH, parathyroid hormone; NTX, collagen type I *N*-telopeptide cross-links; BCE, bone collagen equivalent; LCD, low-calcium diet; HCD, high-calcium diet. There was no significant effect of the supplemental calcium source for any variable.

[2] $\bar{x}$ ± SEM.

[3] $P < 0.0001$ for analysis of covariance, with no difference among the groups.

[4] $P < 0.02$ for analysis of covariance, with no difference among the groups.

compared with some other supplemental calcium sources (10, 23). The reason for the difference between our study and others is unknown, but it may relate to the older age of our subjects, because the men and women in the other studies were younger (10, 23). When our subjects consumed milk as the supplemental calcium source continually for 1 wk, there was no evidence (Table 3) that milk differed from the other 2 calcium sources in its ability to suppress bone resorption (serum NTX) or serum 1,25-dihydroxyvitamin D, or with regard to the observed increases in total serum calcium. These observations suggest that equal amounts of calcium were probably absorbed from all 3 supplemental calcium sources. Equivalent absorption of calcium from isotopically labeled milk and calcium carbonate was reported previously (15).

Thus, on the basis of our acute and longer-term studies of several biomarkers of calcium and bone metabolism, we conclude that calcium bioavailability from milk, calcium-fortified orange juice, and a calcium carbonate supplement is equivalent. This indicates that elderly persons may freely choose to add any of these calcium-rich supplemental sources to their usual diets to help achieve optimal dietary calcium intakes without concern about differences in calcium bioavailability.

We thank the elderly men and women who graciously volunteered to give their time to participate in this study. In addition, we appreciate all the members of the USDA HNRCA Human Study Unit and the Nutrition Evaluation Laboratory who provided assistance in conducting the metabolic study and sample analyses, especially G Dallal of the HNRCA for biostatistical consultation and Judy Lee of the Human Study Unit Dietary Department for designing and monitoring the experimental diets.

REFERENCES

1. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Institute of Medicine, Food and Nutrition Board. Dietary reference intakes for calcium, phosphorus, magnesium, vitamin D, and fluoride. Washington, DC: National Academy Press, 1997.
2. Morgan KJ, Stampley GL, Zabik ME, Fischer DR. Magnesium and calcium intakes of the U.S. population. J Am Coll Nutr 1985;4:195–206.
3. Looker AC, Loria CM, Carroll MD, McDowell MA, Johnson CL. Calcium intakes of Mexican Americans, Cubans, Puerto Ricans, non-Hispanic whites, and non-Hispanic blacks in the United States. J Am Diet Assoc 1993;93:1274–9.
4. Optimal calcium intake. NIH Consensus Conference. JAMA 1994;272:1942–8.
5. Kassarjian Z, Russell RM. Hypochlorhydria: a factor in nutrition. Annu Rev Nutr 1989;9:271–85.
6. Wood RJ, Fleet JC, Cashman K, Bruns ME, Deluca HF. Intestinal calcium absorption in the aged rat: evidence of intestinal resistance to 1,25(OH)2 vitamin D. Endocrinology 1998;139:3843–8.
7. Pattanaungkul S, Riggs BL, Yergey AL, Vieira NE, O'Fallon WM, Khosla S. Relationship of intestinal calcium absorption to 1,25-dihydroxyvitamin D [1,25(OH)2D] levels in young versus elderly women: evidence for age-related intestinal resistance to 1,25(OH)2D action. J Clin Endocrinol Metab 2000;85:4023–7.
8. Heaney RP, Recker RR, Saville PD. Menopausal changes in calcium balance performance. J Lab Clin Med 1978;92:953–63.
9. Sahi T. Genetics and epidemiology of adult-type hypolactasia. Scand J Gastroenterol Suppl 1994;202:7–20.
10. Karkkainen MU, Wiersma JW, Lamberg-Allardt CJ. Postprandial parathyroid hormone response to four calcium-rich foodstuffs. Am J Clin Nutr 1997;65:1726–30.
11. Heaney RP, Weaver CM, Recker RR. Calcium absorbability from spinach. Am J Clin Nutr 1988;47:707–9.
12. Heaney R, Weaver C. Calcium absorption from kale. Am J Clin Nutr 1990;51:656–7.
13. Wood R, Serfaty-Lacrosniere C. Gastric acidity, atrophic gastritis, and calcium absorption. Nutr Rev 1992;50:33–40.
14. Wood R, Suter P, Russell R. Mineral requirements of elderly people. Am J Clin Nutr 1995;62:493–505.
15. Recker R, Bammi A, Barger-Lux M, Heaney R. Calcium absorbability from milk products, an imitation milk, and calcium carbonate. Am J Clin Nutr 1988;47:93–5.
16. Weaver CM, Heaney RP, Martin BR, Fitzsimmons ML. Human

calcium absorption from whole-wheat products. J Nutr 1991;121: 1769–75.
17. Wood RJ, Tamura T. Methodological issues in assessing bioavailability of nutrients and other bioactive substances in dietary supplements: summary of workshop discussion. J Nutr 2001;131: 1396S–8S.
18. Guillemant J, Guillemant S. Comparison of the suppressive effect of two doses (500 mg vs 1500 mg) of oral calcium on parathyroid hormone secretion and on urinary cyclic AMP. Calcif Tissue Int 1993; 53:304–6.
19. Guillemant J, Guillemant S. Acute PTH response to oral calcium load and seasonal variation of vitamin D status in healthy young adult subjects. Eur J Clin Nutr 1996;50:469–72.
20. Need A, Horowitz M, Morris H, Nordin B. Effects of three different calcium preparations on urinary calcium and hydroxyproline excretion in postmenopausal osteoporotic women. Eur J Clin Nutr 1991;45: 357–61.
21. Guillemant J, Guillemant S. Effects on calcium and phosphate metabolism and on parathyroid function of acute administration of tricalcium phosphate. Bone 1991;12:383–6.
22. Guillemant J, Oberlin F, Bourgeois P, Guillemant S. Age-related effect of a single oral dose of calcium on parathyroid function: relationship with vitamin D status. Am J Clin Nutr 1994;60:403–7.
23. Talbot JR, Guardo P, Seccia S, et al. Calcium bioavailability and parathyroid hormone acute changes after oral intake of dairy and nondairy products in healthy volunteers. Osteoporos Int 1999;10: 137–42.
24. Clemens JD, Herrick MV, Singer FR, Eyre DR. Evidence that serum NTx (collagen-type I *N*-telopeptides) can act as an immunochemical marker of bone resorption. Clin Chem 1997;43:2058–63.
25. Rosen HN, Dresner-Pollak R, Moses AC, et al. Specificity of urinary excretion of cross-linked *N*-telopeptides of type I collagen as a marker of bone turnover. Calcif Tissue Int 1994;54:26–9.
26. Holick MF. Vitamin D: photobiology, metabolism, mechanism of action, and clinical applications. In: Favus MJ, ed. Primer on the metabolic bone diseases and disorders of mineral metabolism. 4th ed. Philadelphia: Lippincott, Williams & Wilkins, 1999:92–8.
27. Brown EM, Pollak M, Hebert SC. The extracellular calcium-sensing receptor: its role in health and disease. Annu Rev Med 1998;49: 15–29.