HHS Public Access

Author manuscript

Med Sci Sports Exerc. Author manuscript; available in PMC 2018 July 01.

Published in final edited form as:

Med Sci Sports Exerc. 2017 July ; 49(7): 1437–1442. doi:10.1249/MSS.0000000000001239.

Calcium Supplementation Attenuates Disruptions in Calcium Homeostasis during Exercise

Vanessa D Sherk[1,*], **Sarah J Wherry**[1,*], **Daniel W Barry**[1], **Karen L Shea**[1], **Pamela Wolfe**[2], and **Wendy M Kohrt**[1]

[1]Division of Geriatric Medicine, Department of Medicine, University of Colorado Anschutz Medical Campus, Aurora, CO

[2]Department of Preventative Medicine and Biostatistics, University of Colorado Anschutz Medical Campus, Aurora, CO

Abstract

An exercise-induced decrease in serum ionized calcium (iCa) is thought to trigger an increase in parathyroid hormone (PTH), which can stimulate bone resorption.

Purpose—The purpose of this study was to determine whether taking a chewable calcium (Ca) supplement 30 minutes before exercise mitigates disruptions in Ca homeostasis and bone resorption in competitive male cyclists.

Methods—Fifty-one men aged 18 to 45 y were randomized to take either 1000 mg Ca (CA) or placebo (PL) 30 minutes before a simulated 35-km cycling time trial. Serum iCa and PTH were measured before and immediately after exercise and a marker of bone resorption (collagen type-1 c-telopeptide; CTX) was measured before and 30 minutes after exercise.

Results—Serum iCa decreased in both groups from before to after exercise (mean ± SD CA: 4.89±0.16 to 4.76±0.11 mg/dL; PL: 4.92±0.15 to 4.66±0.22 mg/dL; both $p \leq 0.01$); the decrease was greater ($p=0.03$) in the PL group. There was a nonsignificant ($p=0.07$) attenuation of the increase in PTH by Ca supplementation (CA: 30.9±13.0 to 79.7±42.6 pg/mL; PL: 37.1±14.8 to 111.5±49.4 pg/mL; both $p \leq 0.01$), but no effect of Ca on the change in CTX, which increased in both groups (CA: 0.35±0.17 to 0.50±0.21 ng/mL; PL: 0.36±0.13 to 0.54±0.22 ng/mL, both $p \leq 0.01$).

Conclusion—It is possible that ingesting Ca only 30 minutes before exercise was not a sufficient time interval to optimize gut Ca availability during exercise. Further studies will be needed to

ADDRESS FOR CORRESPONDANCE: Sarah J Wherry, PhD, Division of Geriatric Medicine, University of Colorado Anschutz Medical Campus, Mail Stop B179, 12631 E. 17th Avenue, Room 8111, Aurora, CO 80045, 720-848-6475 (o), 7208487382 (f), Sarah.wherry@ucdenver.edu.

*Drs. Sherk and Wherry are co-first authors

Author roles: Study design: DB, PW, WK. Study conduct and data collection: DB, KS. Data analysis and interpretation: VS, SW, PW, WK. Drafting manuscript: VS, SW, PW, WK. Revising manuscript content: VS, SW, DB, KS, PW, WK. Approving final version of manuscript: VS, SW, DB, KS, PW, WK. WK takes responsibility for the integrity of the data.

Conflict of Interest

We have no conflicts of interest to declare. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation. Results of the present study do not constitute endorsement by ACSM.

determine whether adequate Ca supplementation before and/or during exercise can fully mitigate the exercise-induced decrease in serum iCa and increases in PTH and bone resorption.

Keywords

Calcium supplementation; bone resorption; PTH; serum ionized calcium

Introduction

Exercise can cause a decrease in serum total and ionized calcium (iCa) during prolonged moderate-to-vigorous endurance exercise, which is a trigger for parathyroid hormone (PTH) secretion (1, 2, 6, 7, 11). PTH defends serum calcium (Ca) by increasing intestinal Ca absorption, inhibiting renal Ca excretion, and stimulating the mobilization of skeletal Ca. If exercise induces an increase in bone resorption that is not accompanied by stimulation of bone formation, the repeated disruption of Ca homeostasis during exercise training may contribute to bone loss, such as that observed in cyclists (4, 8, 12).

Consuming a Ca-enriched beverage before and during endurance exercise has been found to be effective in reducing the disruption in Ca homeostasis during exercise. The decrease in iCa and increase in PTH were attenuated when a Ca-enriched beverage or meal was consumed before and/or during exercise (1, 6, 7, 11). This Ca supplementation was effective in attenuating the increase in bone resorption in some experiments (6, 7, 11), but not others (1, 11). The discordance may relate to the timing of Ca dosing.

Beverages that are likely to be consumed during exercise usually contain little or no Ca. This is due, in part, to the difficulty of getting calcium into solution. Other Ca supplements are typically in pill or chewable form, but whether this mode of delivery mitigates the disruption of Ca homeostasis during exercise has not been studied. Timing is also important for exercise planning. Most experiments have investigated pre-exercise supplementation beginning 60 minutes or more before the onset of exercise (7, 11). While this may be appropriate for a planned research exercise visit, this degree of advanced planning may be difficult for regular exercise. Investigating timing of ingestion that may be more suitable (i.e. as close to the onset of exercise as possible) is important in designing future studies to determine whether Ca supplementation prior to exercise has a favorable effect on bone health.

Accordingly, the aim of this study was to determine whether consuming a chewable Ca supplement (1000 mg) 30 minutes before exercise attenuates the decrease in serum iCa and increases in serum PTH and bone resorption (as measured by c-terminal telopeptide of type I collagen; CTX) when compared with a placebo supplement.

Methods

Study Design

This was a randomized, double-blinded, placebo-controlled study of the effects of pre-exercise Ca supplementation on the iCa, PTH, and CTX responses to vigorous exercise. Eligible participants were randomized to take a chewable supplement containing either Ca

citrate (1000 mg elemental Ca) or placebo (PL) 30 minutes before exercise. Active and placebo products were provided by Marigot Ltd (Cork, Ireland). The study was approved by the Colorado Multiple Institutional Review Board and all volunteers provided written informed consent to participate.

Participants

Participants were 51 competitive male road cyclists, aged 18 to 45 years. To be considered a competitive cyclist, prospective participants must have competed in road cycling races for at least one year with plans to participate in at least 10 more races in the next calendar year. Exclusion criteria included thyroid stimulating hormone (TSH) level <0.5 or >5.0 mU/L, creatinine clearance <50 mL/min, parathyroid hormone (PTH) >69 pg/mL, 25(OH) vitamin D <20 ng/mL, hypercalcuria determined by spot urine calcium-to-creatinine ratio ≥0.31, bone mineral density (BMD) T-score <−2.5, and use of drugs known to influence bone metabolism (teriparatide, calcitonin, oral steroids, sex hormones, bisphosphonates) within the past 6 months. If participants reported taking a calcium supplement or a vitamin supplement containing calcium, they were asked to discontinue use 24 hours prior to the research visit. Participants were instructed to fast overnight (minimum of 8 hours) prior to their exercise visit. No dietary instructions were given regarding the meals they consumed the day prior to the visit.

Dual-energy X-ray Absorptiometry (DXA)

Total body, lumbar spine (L1–L4), and proximal femur (total hip, trochanter, femoral neck, subtrochanter) BMD and T-scores were measured on a Discovery-W DXA instrument (Hologic Inc, Waltham, MA). Fat-free mass (FFM) and fat mass (FM) were obtained from the total body scan. All scans were performed by trained technicians. Intra-instrument CVs for scans completed on men <50 yr of age are: − 0.8% total mass, 2.6% FM, 1.1% FFM; 0.8% lumbar spine BMD, 0.9% total hip BMD, 1.9% femoral neck BMD, 1.1% trochanter BMD, and 0.99% subtrochanteric BMD.

Simulated 35-km Time Trial

Participants performed a laboratory-based cycling bout (simulated 35-km time trial) in the fasted state and took the CA or PL supplement 30 minutes before exercise. Dermal Ca loss was estimated during exercise as previously described (2). Nude, dry body weight was measured before and after exercise. Participants were allowed to drink deionized water *ad libitum* during the exercise bouts and total fluid intake was recorded. Sweat volume was estimated from change in weight adjusted for fluid intake and urine production.

Blood Sampling and Analysis

An indwelling intravenous catheter was placed prior to exercise for all blood sampling. Blood samples were collected before, immediately after, and 30 minutes after exercise. Serum iCa, pH, and hematocrit (Hct) were measured immediately after sample collection using a cartridge-based whole blood analyzer (iSTAT, Abbott, East Windsor, NJ); the reported CV for ionized calcium is 1.1%. Intact PTH before and immediately after exercise was measured in duplicate using a two-site chemiluminescent enzyme-labeled

immunometric assay on an Immulite 1000 analyzer (Siemens, Tarrytown, NY). Intra- and inter-assay CVs for PTH are 2.9–3.5% and 4.8–6.8%. CTX before and 30 minutes after exercise was measured in duplicate to assess change in bone resorption (CTX; Nordic Bioscience Diagnostics, Herlev, Denmark). The intra- and inter-assay CVs for CTX are 2.7–10.3% and 2.5–9.2%.

Serum values of iCa, PTH, and CTX were adjusted for plasma volume (PV) shifts based on change in Hct (16). Because the iSTAT measures Hct based on conductivity, Hct values were not adjusted for trapped plasma and no conversion from venous to whole body Hct was performed. Adjusted and unadjusted values are identified using the subscripts “adj” and “unadj” (e.g., iCa_{adj} vs. iCa_{unadj}).

Statistical Analyses

The sample size for the study was based on the observation in a previous study that Ca supplementation 60 minutes before exercise attenuated the increase in PTH by 17.8 ± 20.0 pg/mL (11). A sample size of 21 participants per group provided 80% power to detect this magnitude of difference between groups. Statistical analyses were performed using SAS version 9.3 (SAS Institute Inc., Cary, NC). Data are reported as mean ± SD unless otherwise specified. Changes in outcomes of interest were compared between groups using a linear contrast in a regression model, controlling for the baseline value of the outcome measure to improve the precision of the estimates. The same analysis was repeated for PV-adjusted outcome measures and relative changes in PV-adjusted measures. Within-group changes in outcomes were evaluated as a secondary analysis using paired *t* tests. Pearson’s product-moment correlation coefficients were used to estimate the linear associations among changes in iCa, PTH, and CTX. Statistical significance was accepted as $p<0.05$.

Results

Subject characteristics (Table 1)

Self-reported weekly training data were collected over a 4- to 10-month period. Training frequency (CA: 3.9 ± 0.8 d/wk; PL: 3.9 ± 1.1 d/wk, $p>0.95$) and duration (CA: 523.8 ± 137.9 min/wk; PL: 501.4 ± 172.0 min/wk, $p>0.63$) were similar between the groups. Prevalence of low bone mass, defined as a T-score ≤−1.0, was 31% (6 CA, 10 PL) at the lumbar spine, 8% (2 CA, 2 PL) at the total hip, and 29% at the femoral neck (7 CA, 8 PL).

Responses to Exercise

The simulated time trial was completed in 58.7 ± 3.2 min by the CA group and 60.4 ± 4.4 min by the PL group ($p=0.13$). Estimated sweat loss (CA: 1.41 ± 0.22 L; PL: 1.43 ± 0.25 L; $p=0.78$) and dermal calcium loss (CA: 89.6 ± 34.3 mg; PL: 94.6 ± 49.0 mg; $p=0.69$) were similar in both groups. Plasma volume shifts were also similar between the groups. The decrease in serum iCa_{unadj} from before to immediately after exercise was greater ($p=0.03$) in the PL group than in the CA group ((mean (95% CI) CA: −0.14 (−0.22, −0.07) mg/dL vs. PL: −0.25 (−0.32, −0.19) mg/dL) (Table 2). Attenuation of the increase in PTH_{unadj} from before to immediately after exercise by Ca supplementation approached significance (mean (95% CI) CA: 49.4 (31.6, 67.2) pg/mL vs. PL: 72.3 (55.7, 88.9) pg/mL; $p=0.07$), but there

was no indication of an effect of Ca to attenuate the increase in CTX_{unadj} (p=0.68) from before to 30 minutes after exercise (CA: 0.16 (0.10, 0.22) ng/mL; PL: 0.17 (0.12, 0.23) ng/mL). Adjusting for plasma volume shifts did not change these conclusions (Table 2). Comparisons based on relative changes in CTX_{adj}, PTH_{adj}, and iCa_{adj} were consistent to the absolute changes (Figure 1).

When pooled across groups, the change in serum iCa_{unadj} in response to exercise was inversely associated with the change in PTH_{unadj} ($r=-0.49$, $p<0.001$) and the change in PTH_{unadj} was directly related to change in CTX_{unadj} ($r=0.36$, $p=0.01$) (Figure 2).

Discussion

The aim of this study was to determine whether taking a chewable Ca supplement before vigorous exercise mitigates the exercise-induced decrease in serum iCa and increases in PTH and bone resorption. Our major finding was that Ca supplementation 30 minutes before exercise attenuated the decrease in serum iCa_{adj} and iCa_{unadj} and the attenuation in PTH_{adj} and PTH_{unadj} approached significance. However, the use of a chewable calcium supplement did not alter the CTX_{adj} or CTX_{unadj} response. The decrease in iCa_{adj} and iCa_{unadj} and increase in PTH_{adj} and PTH_{unadj} from before to after exercise, despite pre-exercise Ca supplementation, indicates that the rate of Ca loss from the vascular compartment exceeded the rates of Ca absorption from the gut and mobilization from bone.

Ingestion of a 1000 mg Ca supplement 30 minutes before exercise in the current study attenuated the PTH_{adj} and PTH_{unadj} responses by a similar magnitude as in our previous study of competitive cyclists, in which participants consumed a Ca-enriched beverage 20 minutes before exercise or in 250-mg aliquots during exercise (total of 1000 mg in both cases) (1). In that study, PTH was similarly attenuated by both dosing protocols. Neither protocol was successful in significantly attenuating the CTX response; there was a nonsignificant decline only when Ca was consumed before exercise. In contrast, the timing of Ca supplementation influenced the PTH response to 60 minutes of vigorous walking in a study of older women (11). Women consumed a Ca-enriched beverage every 15 minutes, starting either 15 minutes or 60 minutes before exercise, and continuing during exercise. Starting Ca supplementation 15 minutes before exercise attenuated the decrease in iCa, but not the increases in PTH or CTX. In contrast, starting Ca supplementation 60 minutes before exercise prevented the decrease in iCa, attenuated the increase in PTH by almost 70%, and also tended (p=0.08) to suppress the CTX response (11). In young female cyclists, a high Ca meal (1352 mg) 120 minutes before exercise attenuated the changes in iCa, PTH, and CTX in response to 90 minutes of moderate intensity exercise when compared with a low Ca meal (46 mg) (7). Collectively, these studies indicate that intestinal Ca absorption can play an important role in defending serum Ca during exercise.

The availability of Ca in the small intestine for absorption may reduce the extent to which skeletal Ca is mobilized during exercise to defend serum Ca. There is some evidence that the timing of Ca supplementation relative to the performance of exercise has the potential to enhance skeletal adaptations to exercise training. Collegiate basketball players were found to have a marked decrease in total and leg bone mineral content (BMC) over a year of practice

and competition, but the following year, when supplemental Ca was provided during practices and games, BMC increased in these athletes (8). In contrast, taking supplemental Ca with meals (i.e., not timed with exercise) did not influence the trajectory of BMD change over 1 year in competitive road cyclists (1). Further research will be necessary to determine whether minimizing the disruption of Ca homeostasis during acute exercise sessions can enhance the skeletal adaptations to exercise training.

The optimal dose and timing of Ca supplementation to minimize the disruption of Ca homeostasis during exercise are not known. The minimal dose that has been demonstrated to be effective in attenuating PTH was 1000 mg, but lower doses have not been evaluated (1). The failure to attenuate the CTX response to vigorous exercise with a chewable Ca supplement 30 minutes before exercise in the current study may reflect the need to ingest Ca sooner. If Ca is not positioned in the gut to be absorbed early in an exercise session, it is possible that resorption is stimulated and remains activated for some period of time, even after PTH returns to baseline (9). Consumption of a Ca-enriched beverage starting 60 minutes before and continuing during exercise was effective in attenuating the increase in CTX (6, 11). However, consumption of a Ca-enriched beverage closer to the onset of exercise failed to diminish the CTX response (1, 11), suggesting that it was the timing of Ca supplementation in the current study, and not the use of a chewable supplement, that was the reason for the lack of efficacy.

Although CTX provides a relative index of bone resorption activity, it does not provide a quantitative estimate of the amount of Ca that is mobilized from bone (15). In the current study, the provision of supplemental Ca did not attenuate the exercise-induced increase in CTX_{adj} and CTX_{unadj}, but samples were obtained only 30 minutes after exercise. It is possible that CTX returned to the basal level faster in the CA than the PL condition, reflecting less reliance on skeletal Ca to restore serum Ca homeostasis, but a longer sampling protocol would be necessary to evaluate this. Quantifying the mobilization of Ca from the skeleton and absorption of intestinal Ca during exercise would provide insight for the development of strategies (e.g., Ca supplementation) to optimize the skeletal adaptations to exercise training. It may be possible to do this using stable isotope kinetics but, to the best of our knowledge, this approach has never been used during exercise.

Although the exercise-induced increase in PTH leads to an activation of bone resorption, resulting in what appears to be a catabolic response, it remains possible that PTH could have an anabolic effect on bone. Paradoxically, PTH has both anabolic and catabolic actions on bone, whereby transient increases in PTH are anabolic and chronic elevation is catabolic (13). Teriparatide, a PTH analog that is used to treat osteoporosis, increases bone resorption in a manner similar to vigorous exercise, and yet has anabolic effects on bone because it also stimulates bone formation (17). This has been shown to occur in a dynamic fashion, whereby bone resorption was increased within hours after a single dose of teriparatide, but the increase in bone formation was not apparent until 24 h later (14). After 28 days of daily teriparatide dosing, bone formation (i.e., P1NP) was increased by ~100%, whereas CTX was increased by <10% (5). It is possible that the exercise-induced increase in PTH also has a delayed effect on bone formation, but evidence does not support this (9, 10). Future research

is needed to fully investigate the bone formation potential of exercise in light of the bone resorption response we observed.

It is also possible that people with low bone mass may respond to this type of intervention differently than people with normal bone mass. Our study population included individuals with t-scores between −1.0 and −2.5, which would meet criteria for low bone mass. However, due to the low number of participants in this category, we did not analyze whether they responded differently than their peers with t-scores greater than −1.0. However, we think it is unlikely that the difference in BMD scores would influence the outcomes because of the consistent findings across multiple labs that endurance exercise stimulates PTH secretion (3, 7, 10). Our working hypothesis is that the repeated activation of PTH and CTX by exercise is the cause of the bone loss observed in this population, and we hope to address the impact of bone health status on these markers in a future clinical trial.

In conclusion, taking a chewable Ca supplement 30 minutes before exercise attenuated the decrease in iCa and increase in PTH during exercise, but did not prevent an increase in CTX. Ca supplementation may need to occur earlier before exercise to reduce the mobilization of skeletal Ca during exercise. Further studies are needed to determine whether adequate Ca supplementation before and/or during exercise can fully mitigate the exercise-induced increases in PTH and bone resorption. Given the paradoxical actions of PTH on bone, it must also be determined whether preventing the increase in PTH is likely to be beneficial or harmful to bone.

Acknowledgments

This study was supported in part by NIH awards R01 AG018857, UL1 TR001082, P30 DK048520, T32 DK007658, and T32 AG000279. Marigot Ltd provided Ca and placebo supplements.

Funding Sources: Supported in part by NIH awards R01 AG018857, UL1 TR001082, P30 DK048520, T32 DK007658, and T32 AG000279. Marigot Ltd provided product support.

Reference List

1. Barry DW, Hansen KC, van Pelt RE, Witten M, Wolfe P, Kohrt WM. Acute calcium ingestion attenuates exercise-induced disruption of calcium homeostasis. Med Sci Sports Exerc. 2011; 43(4): 617–23. [PubMed: 20798655]
2. Barry DW, Kohrt WM. Acute effects of 2 hours of moderate-intensity cycling on serum parathyroid hormone and calcium. Calcified Tissue International. 2007; 80(6):359–65. [PubMed: 17549534]
3. Barry DW, Kohrt WM. Acute effects of 2 hours of moderate-intensity cycling on serum parathyroid hormone and calcium. Calcif Tissue Int. 2007; 80(6):359–65. [PubMed: 17549534]
4. Barry DW, Kohrt WM. BMD decreases over the course of a year in competitive male cyclists. Journal of Bone and Mineral Research. 2008; 23(4):484–91. [PubMed: 18072875]
5. Glover SJ, Eastell R, McCloskey EV, et al. Rapid and robust response of biochemical markers of bone formation to teriparatide therapy. Bone. 2009; 45(6):1053–8. [PubMed: 19679211]
6. Guillemant J, Accarie C, Peres G, Guillemant S. Acute effects of an oral calcium load on markers of bone metabolism during endurance cycling exercise in male athletes. Calcified Tissue International. 2004; 74(5):407–14. [PubMed: 14735261]
7. Haakonssen EC, Ross ML, Knight EJ, et al. The effects of a calcium-rich pre-exercise meal on biomarkers of calcium homeostasis in competitive female cyclists: a randomised crossover trial. PloS one. 2015; 10(5):e0123302. [PubMed: 25970439]

8. Klesges RC, Ward KD, Shelton ML, et al. Changes in bone mineral content in male athletes. Mechanisms of action and intervention effects. Journal of the American Medical Association. 1996; 276(3):226–30. [PubMed: 8667568]
9. Scott JP, Sale C, Greeves JP, Casey A, Dutton J, Fraser WD. The effect of training status on the metabolic response of bone to an acute bout of exhaustive treadmill running. The Journal of clinical endocrinology and metabolism. 2010; 95(8):3918–25. [PubMed: 20519353]
10. Scott JP, Sale C, Greeves JP, Casey A, Dutton J, Fraser WD. The role of exercise intensity in the bone metabolic response to an acute bout of weight-bearing exercise. J Appl Physiol (1985). 2011; 110(2):423–32. [PubMed: 21127210]
11. Shea KL, Barry DW, Sherk VD, Hansen KC, Wolfe P, Kohrt WM. Calcium supplementation and parathyroid hormone response to vigorous walking in postmenopausal women. Med Sci Sports Exerc. 2014; 46(10):2007–13. [PubMed: 24576866]
12. Sherk VD, Barry DW, Villalon KL, Hansen KC, Wolfe P, Kohrt WM. Bone loss over 1 year of training and competition in female cyclists. Clinical Journal of Sport Medicine. 2014; 24(4):331–6. [PubMed: 24326929]
13. Silva BC, Costa AG, Cusano NE, Kousteni S, Bilezikian JP. Catabolic and anabolic actions of parathyroid hormone on the skeleton. Journal of endocrinological investigation. 2011; 34(10):801–10. [PubMed: 21946081]
14. Sugimoto T, Nakamura T, Nakamura Y, Isogai Y, Shiraki M. Profile of changes in bone turnover markers during once-weekly teriparatide administration for 24 weeks in postmenopausal women with osteoporosis. Osteoporosis international : a journal established as result of cooperation between the European Foundation for Osteoporosis and the National Osteoporosis Foundation of the USA. 2014; 25(3):1173–80.
15. Swaminathan R. Biochemical markers of bone turnover. Clinica chimica acta; international journal of clinical chemistry. 2001; 313(1–2):95–105. [PubMed: 11694245]
16. van Beaumont W, Strand JC, Petrofsky JS, Hipskind SG, Greenleaf JE. Changes in total plasma content of electrolytes and proteins with maximal exercise. J Appl Physiol. 1973; 34(1):102–6. [PubMed: 4697366]
17. Zikan V, Stepan JJ. Marker of Bone Resorption in Acute Response to Exogenous or Endogenous Parathyroid Hormone. Biomarker insights. 2008; 3:19–24. [PubMed: 19578490]

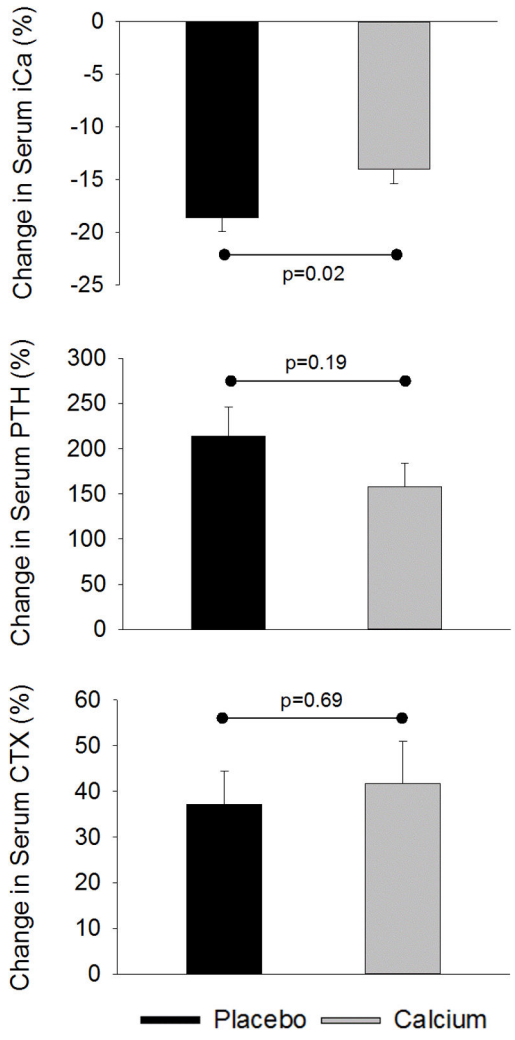

Figure 1.

Relative changes (mean ± SE) in ionized calcium (iCa) and parathyroid hormone (PTH) from before to immediately after exercise and change in C-terminal telopeptide of type-I collagen (CTX) from before to 30 minutes after exercise; data were adjusted for shifts in plasma volume.

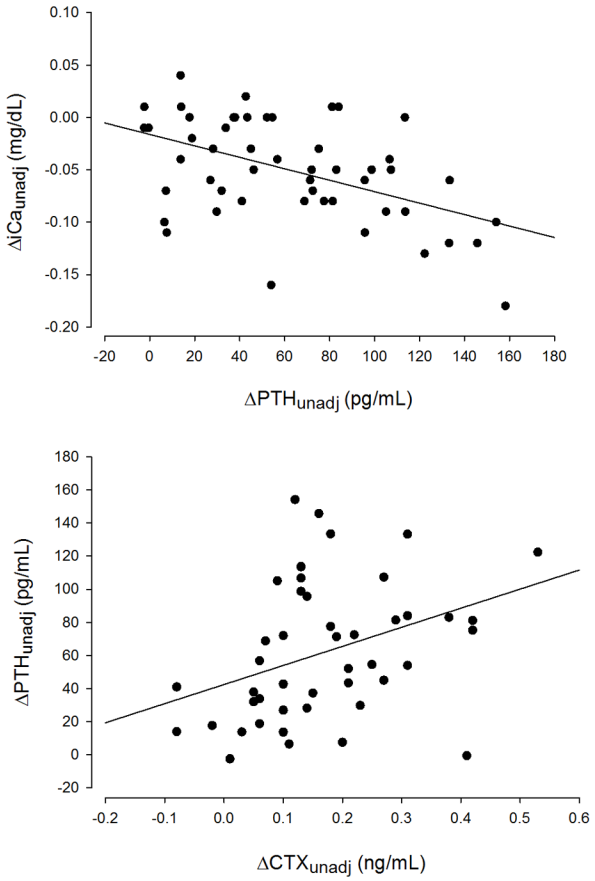

Figure 2.

All data pooled across groups. Solid lines are the simple linear regression of Y on X. (A) Change in unadjusted serum ionized calcium (iCa_{unadj}) versus change in unadjusted parathyroid hormone (PTH_{unadj}) from before to after exercise. (B) Change in PTH_{unadj} versus change in unadjusted C-terminal telopeptide of type-I collagen (CTX_{unadj}) from before to after exercise.

Table 1

Baseline characteristics (mean ± SD)

Variable	Calcium (n = 23)	Placebo (n = 28)	p value
Age (y)	35.1 ± 6.5	36.8 ± 8.0	0.41
Body mass index (kg/m^2)	23.8 ± 3.0	23.1 ± 2.0	0.34
Weight (kg)	79.6 ± 12.4	75.3 ± 6.8	0.15
Fat-free mass (kg)	63.4 ± 8.2	60.9 ± 4.9	0.20
Fat mass (kg)	13.1 ± 6.4	11.6 ± 3.9	0.33
Years in competition	5.5 ± 4.4	7.2 ± 6.9	0.29
Maximal aerobic power (mL/kg/min)	53.7 ± 7.4	54.1 ± 6.2	0.84
Serum 25-hydroxyvitamin D (ng/mL)	28.0 ± 6.0	32.0 ± 8.5	0.06
Lumbar spine BMD (g/cm^2)	1.189 ± 0.186	1.152 ± 0.153	0.44
Total hip BMD (g/cm^2)	1.083 ± 0.136	1.027 ± 0.105	0.10
Femoral neck BMD (g/cm^2)	0.902 ± 0.129	0.871 ± 0.110	0.36
Trochanter BMD (g/cm^2)	0.820 ± 0.107	0.789 ± 0.102	0.30
Subtrochanter BMD (g/cm^2)	1.278 ± 0.159	1.207 ± 0.116	0.07

Table 2

Serum ionized calcium (iCa), parathyroid hormone (PTH), C-terminal telopeptide of type I collagen (CTX), and hematocrit (Hct) responses to a simulated 35-km time trial, with and without adjustment for plasma volume changes.

	Calcium (n=23)			Placebo (n=28)		
Variable	**Before exercise (SD)**	**After exercise (SD)**	**Mean Change (95% CI)**	**Before exercise**	**After exercise**	**Mean Change (95% CI)**
Unadjusted						
iCa, mg/dL	4.89 (0.16)	4.76 (0.11) *	−0.14 (−0.22, −0.07) #	4.92 (0.15)	4.66 (0.22) *	−0.25 (−0.32, −0.19)
PTH, pg/mL	30.9 (13.0)	79.7 (42.6) *	49.4 (31.6, 67.2)	37.1 (14.8)	111.5 (49.4) *	72.3 (55.7, 88.9)
CTX, ng/mL	0.35 (0.17)	0.50 (0.21) *	0.16 (0.10, 0.22)	0.36 (0.13)	0.54 (0.22) *	0.17 (0.12, 0.23)
Hct, %	46.6 (1.9)	49.2 (2.2) *	2.7 (2.1, 3.3)	46.5 (2.6)	49.6 (2.4) *	3.1 (2.6, 3.6)
Adjusted						
iCa, mg/dL	4.89 (0.16)	4.20 (0.25) *	−0.70 (−0.84, −0.57) #	4.92 (0.15)	4.00 (0.37) *	−0.91 (−1.03, −0.79)
PTH, pg/mL	30.9 (13.0)	76.4 (42.3) *	45.9 (27.93, 63.76)	37.1 (14.8)	106.1 (48.6) *	67.7 (50.9, 84.4)
CTX, ng/mL	0.35 (0.17)	0.46 (0.19) *	0.12 (0.06, 0.17)	0.36 (0.13)	0.49 (0.20) *	0.12 (0.07, 0.17)

iCa and PTH were measured in all participants before and immediately after exercise. CTX was measured in a subset (21 Calcium, 26 Placebo) before and 30 minutes after exercise. Mean change was conditioned on baseline values.

* $p<0.01$, within-group change;

# $p<0.05$, between-group difference in change.