# Effects of strength training on bone mineral density: hormonal and bone turnover relationships

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*Departments of Kinesiology and of Human Nutrition and Food Systems, University of Maryland, College Park 20742; Division of Gerontology, University of Maryland School of Medicine and Veterans Affairs Medical Center, Baltimore 21201; Osteoporosis Assessment Center, Wheaton, Maryland 20902; Mineral Metabolism Unit, Veterans Affairs Hospital, Loma Linda University, Loma Linda, California 92357; and Department of Orthopedics and Rehabilitation, Yale University School of Medicine, New Haven, Connecticut 06510*

**Ryan, A. S., M. S. Treuth, M. A. Rubin, J. P. Miller, B. J. Nicklas, D. M. Landis, R. E. Pratley, C. R. Libanati, C. M. Gundberg, and B. F. Hurley.** Effects of strength training on bone mineral density: hormonal and bone turnover relationships. *J. Appl. Physiol.* 77(4): 1678–1684, 1994.—The effects of a 16-wk strength-training program on bone mineral density (BMD) was assessed by dual-energy X-ray absorptiometry in 21 men [age 61 ± 1 (SE) yr]. Sixteen men (age 59 ± 2 yr) served as control subjects. To investigate the possible hormonal relationships underlying the effects on BMD, serum concentrations of growth hormone, insulin-like growth factor I, and testosterone were determined before and after training. In addition, osteocalcin and skeletal alkaline phosphatase (markers of bone formation) and tartrate-resistant acid phosphatase (a marker of bone resorption) were measured before and after training to assess bone turnover. The training program resulted in a 2.8 ± 0.6% increase in femoral neck BMD (1.004 ± 0.037 vs. 1.031 ± 0.037 g/cm$^2$; $P < 0.001$). However, there were no significant changes in total body, anterioposterior spine, lateral spine, Ward's triangle, or greater trochanter BMD. Moreover, there were no significant changes in growth hormone, insulin-like growth factor I, testosterone, osteocalcin, or skeletal alkaline phosphatase. There were no changes in the control group. Thus, strength training can increase femoral neck BMD, and this effect does not appear to be accompanied by changes in anabolic hormones or markers of bone formation and resorption.

weight training; anabolic hormones; insulin-like growth factor I

ALTHOUGH OSTEOPOROSIS is more prevalent among women, bone loss does occur with age in men, beginning at ~40–45 yr and proceeding at a rate of 3–4% per decade (22). These losses are thought to be related to the decline in strength and muscle mass that occurs with age (33). Muscle fiber atrophy and muscle weakness are more prevalent among elderly individuals who develop hip fractures than those individuals of similar age who do not (1). Moreover, bone mineral density (BMD) is related to the strength of the anatomically related muscles (10, 35).

It has been suggested that activities that require heavy loading with few repetitions (31), resulting in high strain rates (21), may provide optimal stimuli for increases in BMD. Cross-sectional studies have recently revealed that individuals who participate in strength-training (ST) programs appear to have a greater BMD than inactive control subjects (7, 18). However, these studies have the usual limitation of selection bias inherent in cross-sectional investigations. For this reason, many investigators have tried to determine by performing prospective studies whether ST can increase BMD (6, 13, 23, 25, 26, 28, 34, 35a).

Results from these studies are equivocal. Significant increases in BMD have been observed after ST, whereas a loss in BMD was found in inactive control subjects during this same time period (34). Nevertheless, one study showed no significant increase in lumbar BMD after 12 mo of training (13), whereas another actually showed significant decreases at 4.5 and 9 mo of training (30). Recent results from our lab revealed significant increases in regional BMD and in indexes of bone formation after heavy-resistance ST (23). However, as pointed out in the report (23), that study had the limitations of a small sample size ($n$ = 9) and low initial BMD values relative to the control group. Hence, we could not rule out the possibility that our results were affected by regression toward the mean. Furthermore, no data were collected on potential mechanisms that could explain these results. Growth hormone (GH), insulin-like growth factor I (IGF-I), and testosterone are believed to influence bone metabolism (17, 29, 32). Low levels of GH and testosterone associated with aging may be related to the age-related loss of BMD because both testosterone therapy (17) and GH administration (32) result in an increase in BMD in those individuals with deficiencies. In addition, administration of recombinant human GH in normal young males stimulates osteoblasts and activates bone remodeling (4). Because of the pulsatile release of GH, blood levels of IGF-I are often measured as an indicator of GH secretion (32). Acute resistive exercise may increase the blood concentration of GH, IGF-I, and testosterone in young and elderly male subjects (8, 19). Thus, we hypothesized that ST may increase BMD through elevations in IGF-I, GH, and testosterone levels.

The primary purpose of the present study was to reinvestigate the effects of heavy-resistance ST on regional and total body BMD using a larger sample size ($n$ = 21 in the training group) and subjects with higher initial BMD values than were used in our previous study (23). To determine whether these factors could affect the results, it was necessary to keep the training protocol the same as in our previous investigation. A second purpose was to assess the possible hormonal and bone turnover relationships underlying the effects on bone density.

## METHODS

*Subjects.* Twenty-one subjects between the ages of 51 and 71 yr volunteered to participate in the ST program. Sixteen similarly aged men served as control subjects. Subjects were screened by medical history questionnaire, physical examination, and a graded exercise test. Only persons that had not participated in a regular exercise program for a minimum of 6 mo before the study were included. All subjects were nonsmokers, free of cardiovascular disease or recent skeletal fracture, and not on any medications that would alter calcium or bone metabolism. Each participant signed appropriate consent forms. All methods and procedures were approved by the Institutional Review Board of the University of Maryland.

*Standardized diet.* In an attempt to keep diet constant throughout the study and to help control for the effects of diet on BMD, body composition, bone markers, or hormones, subjects were instructed on the American Heart Association Phase I diet by a nutritionist 4 wk before the initiation of the study. This diet consists of 50–55% carbohydrates, 15–20% protein, ≤30% fat, and ≤300 mg of cholesterol per day. Subjects were asked to maintain this diet throughout the study's duration. Compliance was monitored by food records taken every 3 wk throughout the study. As an additional control, the subjects in the training group recorded dietary intake for 5 days before the initial blood samples and then were given a copy of this diet to replicate for the same time period before the final blood sampling while they were still training. They were asked to record any deviation from the initial diet record on the final diet record. Similar procedures were followed for the control group. Computerized food analysis programs were used to analyze the total calories; percentage of calories from carbohydrates, proteins, and fats; and the amount of calcium and phosphorus.

*Body composition and maximal $O_2$ uptake ($\dot{V}O_{2\,max}$).* Hydrostatic weighing was utilized for the determination of body density after correction for residual volume. Percent body fat and fat-free mass were estimated from body density values using the Brozek formula (5).

$\dot{V}O_{2\,max}$ was measured before and after training in the training group to confirm that the subjects were untrained before the study and that they were not engaged in regular aerobic exercise during the study. A continuous treadmill test protocol was used in which the speed was kept constant but was varied for each individual depending on his level of conditioning. The grade was increased from 0 to 4% at 2 min and was then increased 2% every minute after the 3rd min until the subject was unable to continue. Expired air was collected during the last 3–4 min of exercise, and gas volumes were measured with a Collins Tissot 120-liter chain-compensated spirometer. The fractional concentrations of oxygen and carbon dioxide in the collected gas samples were determined with the use of a model 2000 AirSpec mass spectrometer. Validation that $\dot{V}O_{2\,max}$ had been reached was established if two of the following three criteria were met: *1*) a plateau in oxygen uptake with an increased work load as evidenced by a difference in oxygen uptake of <2 $ml \cdot kg^{-1} \cdot min^{-1}$, *2*) a respiratory exchange ratio >1.10, and *3*) a maximal heart rate within 10 beats/min of the age-predicted maximal value. All subjects who were included in the data analysis met these criteria.

*BMD.* Dual-energy X-ray absorptiometry (model DPX, LUNAR Radiation, Madison, WI) was used to scan the total body, anterioposterior (AP) lumbar spine ($L_2$–$L_4$), and femur in seven subjects in the training group and in seven control subjects. The LUNAR model DPX-L was used to scan these areas as well as the lateral spine ($B_2$–$B_4$) in 14 subjects in the training group and in 9 control subjects. The LUNAR version 3.4 DPX and 1.2i DPX-L programs were used for all BMD determinations. The same scanner and program used for initial baseline assessments were used for the final assessments for all subjects. All bone density tests took place during one visit at the same time of day after an overnight fast.

To ensure investigator reliability, five repeat scans of the total body and three repeat scans of the lateral spine were performed on the DPX-L in two healthy volunteers who were repositioned for each scan. The coefficient of variations (CV) were 0.5 and 2.6% for total body and lateral spine, respectively. For the DPX, the CVs were 0.6, 0.4, 2.3, and 0.8% for the AP spine, femoral neck, Ward's triangle, and greater trochanter, respectively, and for the DPX-L, the CVs were 0.8, 1.3, 4.2, and 1.3%, respectively, for these same regions in five repeat scans. To ensure system precision of the DPX-L, an aluminum spine phantom was scanned for BMD at the medium speed according to the manufacturer's specifications five times before the study (CV = 0.3%) and five times on completion (CV = 0.6%). Five identical scans were performed using the DPX, demonstrating a CV of 0.8%.

*Indexes of bone turnover.* Bone formation was estimated from serum concentrations of osteocalcin and skeletal alkaline phosphatase (SAP) (2). Serum levels of tartrate-resistant acid phosphatase (TrACP) were used as an index of bone resorption (2). Blood samples were taken before and after ~16 wks of ST. Samples taken after training were drawn ~24 h after the last exercise session. None of these markers changed significantly from 24 to 48 h in our previous study (23). All blood samples were taken after an overnight fast at approximately the same time (8:30 A.M.) in the morning. Samples were allowed to clot at room temperature for 20 min and then centrifuged. After separation, aliquots of ~1.0 ml of serum were immediately pipetted into prechilled 4.0-ml polystyrene culture tubes. The samples were stored at −70°C until assayed. To eliminate interassay variations, samples were analyzed in the same assay and performed in duplicate for osteocalcin, SAP, and TrACP.

The radioimmunoassay (RIA) described by Price and Nishimoto (27) and later modified by Gundberg et al. (15) was utilized to determine serum concentrations of osteocalcin. Because human osteocalcin cross-reacts identically with the antibody raised against purified bovine osteocalcin, all assays were performed with rabbit antiserum to bovine osteocalcin. Bovine osteocalcin was purified to homogeneity and was used for standards and tracer. Bound and free fractions were separated by goat anti-rabbit serum. The intra-assay variation was 5.3%. The sensitivity and specificity of the assay were 0.1–0.3 μg/l and 99%, respectively.

The determination of serum concentrations of SAP isoenzyme and the preparation of hepatic and bone isoenzyme standards for this spectrophotometric assay have been described by Farley et al. (11) using *p*-nitrophenyl phosphate as the substrate. The intra-assay variation was <5%. When compared with alkaline phosphatase standards containing from 25 to 75% SAP isoenzyme, the amount of SAP isoenzyme activity can be calculated with an accuracy of ~10% and a sensitivity of <0.2 U/l (11).

Serum TrACP concentration was analyzed using the procedures described by Lau et al. (20). The aliquots of human serum samples were diluted fivefold with distilled water to decrease the concentration of inhibitors. Samples were heated to inactivate TrACP activity from erythrocytes. The intra-assay variation was <5%. The sensitivity and specificity of the assay were 0.2 U/l and 90–95%, respectively (20).

*Anabolic hormones and growth factors.* Blood samples were drawn before and after ~16 wk of ST in both the training and control groups. All blood samples were taken on two separate days, both for initial baselines before and after training. Posttraining samples for GH, IGF-I, and testosterone were taken 24 and 48 h after the last exercise session. All blood samples were drawn after an overnight fast at a similar time of morning.

Samples were allowed to clot at room temperature for ~20 min and then centrifuged. After separation, aliquots of ~1 ml of serum were pipetted into culture tubes. The blood was stored at −70°C until the samples were assayed.

RIAs were utilized to determine serum concentrations of GH, IGF-I, and testosterone in 14 training and 9 control subjects. To eliminate interassay variation, all samples were analyzed in duplicate in the same assay. GH was measured using the Allegro human GH immunoradiometric assay (Nichols Laboratories, San Juan Capistrano, CA), which incorporated two monoclonal antibodies, specifically $^{125}$I-labeled monoclonal human GH antibody and bioton-coupled monoclonal human GH antibody. The intra-assay CV was 12% at a concentration of 0.5 μg/l and 2% at a concentration of 5 μg/l. Sensitivity was ~0.05–0.1 μg/l. Serum IGF-I was determined by double-antibody RIA after acid ethanol extraction at Endocrine Sciences Laboratories (Calabasas Hills, CA) and represents total IGF-I levels. The intra-assay variation was 6.4% at 250 μg/l and 10% at 125 μg/l. The sensitivity of the assay was 10 μg/l. Testosterone was quantified by a $^{125}$I solid-phase RIA supplied by ICN Biomedicals (Carson, CA). The intra-assay CV was 5% at a dose of 1 μg/l and 6% at a dose of 10 μg/l, and the sensitivity was 0.05 μg/l.

*Strength tests and training program.* Upper and lower body strengths were tested before and after training by a three-repetition maximum (3-RM) test using Keiser K-300 pneumatic variable-resistance machines. A 3 RM was defined as the maximal resistance that could be moved three times through the full range of motion. Four training sessions were conducted as an accomodation period before ST so that the subjects could become accustomed to the equipment. The subjects were allowed three warm-up repetitions. Approximately the same number of trials were performed before reaching the 3-RM weight after training as before training. The strength measures were completed on the leg press, leg extension, chest press, latissimus pull down, upper back row, and military press exercise machines. These exercises were chosen because they were the only ones used in training that could be easily tested using an objective criteria.

The ST program consisted of 3 exercise sessions/wk on nonconsecutive days for ~16 wk using 14 exercises of Keiser K-300 variable-resistance machines, dumbbells, and floor exercises. None of the subjects who entered the training program dropped out before it was over. Each training session included a warm-up of low-intensity cycling for 3 min. This warm-up period was followed by 10 min of static stretching. After the subjects had completed the accommodation period of training and all strength testing, the subjects began each training session at a resistance of ~5 RM (90% of 3 RM) for the first three to four repetitions. Starting with the fourth or fifth repetition, resistance was reduced just enough to permit the subject to complete another few repetitions. This process was repeated until a total of 15 repetitions were completed. The Keiser equipment allowed for this decrease in resistance without having to interrupt the normal cadence of each repetition so that 15 continuous repetitions were completed while using a resistance that required near maximal effort on every repetition. Starting and finishing resistances were recorded for each exercise. The resistance level was periodically checked and adjusted to accommodate strength gains for each individual. Upper and lower body exercises were alternated to minimize fatigue, with a rest interval of 1–2 min between exercises and no rest interval between repetitions. The exercises were performed in the following order: leg press, chest press, leg curl, latissimus pull down, leg extension, military press, adductor, abductor, upper back, triceps, lower back, upper abdominals, biceps curl (dumbbells), and lower abdominals (floor). A second set of exercises was performed on the leg press, leg curl, and leg extension machines after the entire circuit was completed. Only one set of all other exercises was performed. Blood pressures and heart rates were recorded before warm-up, after the military press, after the second circuit on the leg machines, and before departure. Subjects recorded their body weight before every third exercise session.

TABLE 1. *Physical characteristics of training and control groups*

| | Training Group | | Control Group | |
|---|---|---|---|---|
| | Before | After | Initial | Final |
| Age, yr | 61±1 | | 59±2 | |
| Height, cm | 175±1 | | 173±2 | |
| Weight, kg | 83.4±3.1 | 83.7±3.0 | 80.4±2.7 | 81.1±2.8 |
| %Fat | 25.6±1.4 | 24.0±1.5† | 25.2±1.7 | 26.2±1.7 |
| Fat-free mass, kg | 61.4±1.7 | 63.0±1.8† | 59.5±1.5 | 59.1±1.4 |
| $\dot{V}O_{2\,max}$, ml·kg$^{-1}$·min$^{-1}$ | 28.3±1.2 | 29.2±1.2* | | |

Values are means ± SE; $n$ = 21 and 16 men for training and control groups, respectively. $\dot{V}O_{2\,max}$, maximal $O_2$ uptake. Significantly different from before training: * $P < 0.05$; † $P < 0.001$.

*Statistical analyses.* A repeated-measures analysis of variance (ANOVA) was used to determine differences between and within the training and control groups for the dependent variables. When ANOVA revealed significant results, $t$-tests were performed on specific planned comparisons of interest. Data are expressed as means ± SE, and significance was set $P < 0.05$ with Bonferroni adjustments. All analyses were performed with Statview 512$^+$ (BrainPower) and the Super ANOVA statistical package (Abacus Concepts) for the Macintosh personal computer.

## RESULTS

*Subject characteristics.* There was no significant difference between the training and control groups in initial baseline values for age, height, or body mass (Table 1). The training group did not change body mass, but percent body fat decreased ($P < 0.001$) and fat-free mass increased ($P < 0.001$) after training. $\dot{V}O_{2\,max}$ increased for the training group (28.3 ± 1.2 vs. 29.2 ± 1.2 ml·kg$^{-1}$·min$^{-1}$; $P < 0.05$), but this change was too small to be considered physiologically meaningful. The control group did not change weight, percent body fat, or fat-free mass over the study period.

*Dietary analysis.* Analysis of the 5-day food records in the training group revealed no changes before vs. after training in caloric intake (2,247 ± 57 vs. 2,265 ± 61 kcal) and percent calories from carbohydrates (52 ± 1 vs. 52 ± 1%), fat (30 ± 1 vs. 30 ± 1%), and protein (18 ± 1 vs. 18 ± 1%). In addition, there were no significant differences in calcium (939 ± 41 vs. 951 ± 40 mg) or phosphorus levels (1,464 ± 29 vs. 1,381 ± 38 mg). Likewise, no significant differences were observed between initial and final food records for the control group.

*Muscular strength.* There was a substantial improvement in muscular strength for the training group as indicated by increases of 37% in the leg press (331 ± 67 vs. 455 ± 100 kg), 43% in the leg extension (54 ± 11 vs. 77 ± 14 kg), 28% in the chest press (53 ± 9 vs. 67 ± 10 kg), 33% in the latissimus pull down (55 ± 11 vs. 73 ± 14 kg), 44% in the military press (28 ± 5 vs. 40 ± 5 kg), and 55% in upper back strength (48 ± 9 vs. 74 ± 14 kg) (Table 2). When all upper and lower body exercises were pooled

TABLE 2. *3-RM strength values for training and control groups*

| | Training Group | | Control Group | |
|---|---|---|---|---|
| | Before | After | Initial | Final |
| Upper body, kg | 174±7 | 240+9* | 182+8 | 190+9 |
| Lower body, kg | 387±15 | 532±22* | 409±16 | 422±14 |
| Total body, kg | 561±20 | 772±29* | 591±20 | 612±17 |

Values are means ± SE; $n$ = 21 and 15 men for training and control groups, respectively. Upper body strength, sum of values for chest press, military press, latissumus pull down, and upper back row; lower body strength, sum of bilateral leg press and unilateral leg extension; total body strength, sum of upper and lower body values. 3-RM, 3-repetition maximum. * Significantly different from before training, $P < 0.001$.

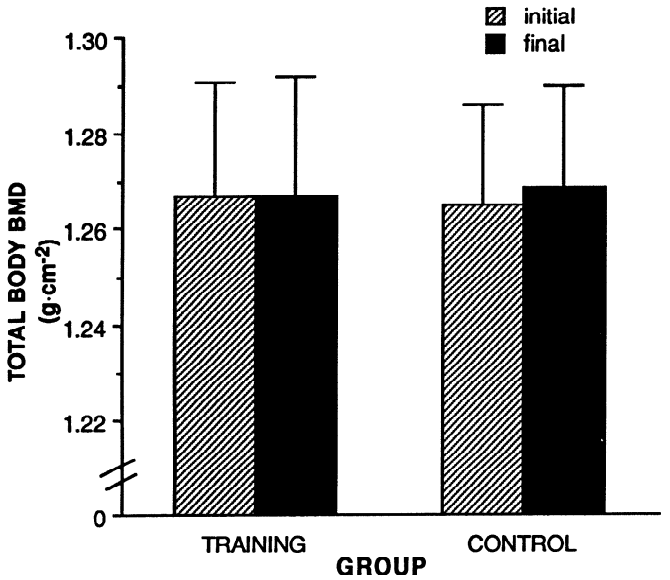


FIG. 1. Bone mineral density (BMD) of total body in strength-trained and control groups. Values are means ± SE. No differences were significant.

separately, there was a 39 ± 2% average increase in the upper body (3-RM) strength test and a 38 ± 3% average increase in lower body strength. No significant changes occurred between initial and final values for the control group in any of the 3-RM strength tests.

*BMD.* There was no significant change in total body BMD (1.267 ± 0.024 vs. 1.267 ± 0.025 g/cm$^2$) or AP lumbar spine ($L_2$–$L_4$) BMD (1.285 ± 0.046 vs. 1.284 ± 0.045 g/cm$^2$) with training (Figs. 1 and 2, respectively). Although the changes within each group were not statistically significant for lateral spine ($B_2$–$B_4$) BMD, the mean values in each group tended to move in opposite directions ($P$ = 0.09 for interaction; Fig. 3). The training program resulted in a 2.8 ± 0.6% increase in femoral neck BMD (1.004 ± 0.037 vs. 1.031 ± 0.037 g/cm$^2$; $P < 0.001$; Fig. 4). There was no change in the BMD of Ward's triangle (0.882 ± 0.044 vs. 0.880 ± 0.044 g/cm$^2$) or the greater trochanter (0.943 ± 0.034 vs. 0.944 ± 0.037 g/cm$^2$) in the training group. Similarly, there were no significant differences between initial and final BMD values for the total body, AP spine, lateral spine, femoral neck (Figs. 1–4), Ward's triangle (0.821 ± 0.027 to 0.834 ± 0.022 g/cm$^2$), or the greater trochanter (0.895 ± 0.020 to 0.909 ± 0.016 g/cm$^2$) in the control group.

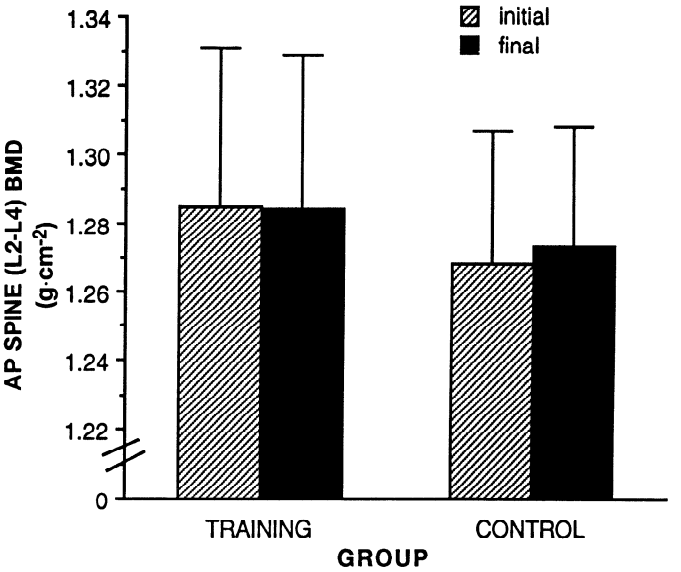


FIG. 2. BMD of anterioposterior (AP) spine ($L_2$–$L_4$) in strength-trained and control groups. Values are means ± SE. No differences were significant.

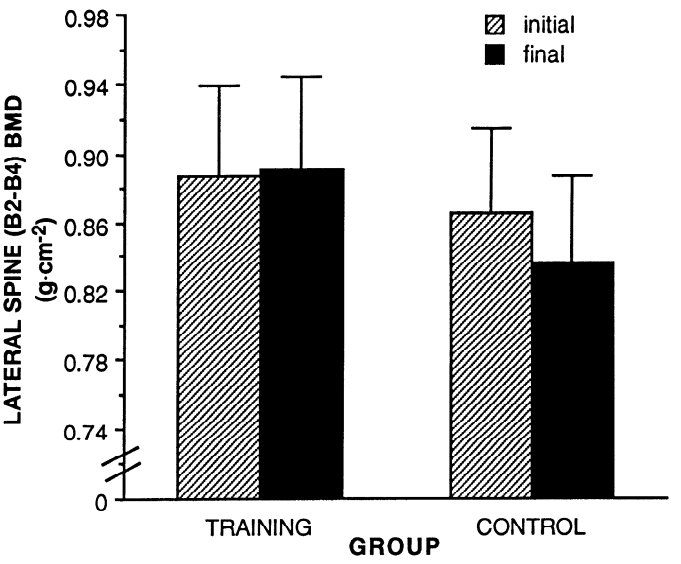


FIG. 3. BMD of lateral spine ($B_2$–$B_4$) in strength-trained and control groups. Values are means ± SE. No differences were significant.

*Indexes of bone remodeling.* The training program did not change osteocalcin or SAP isoenzyme levels (Table 3). Likewise, there were no significant differences between initial and final values for osteocalcin or SAP in the control group. Serum TrACP levels increased significantly in both groups, but there were no differences between the two groups.

*Anabolic hormones and IGF-I.* There were no significant changes in baseline levels of serum GH (0.32 ± 0.12 vs. 0.36 ± 0.12 μg/l), IGF-I (192 ± 8 vs. 191 ± 13 μg/l), or testosterone (6.98 ± 0.60 vs. 6.64 ± 0.51 μg/l) as a result of training (Table 4). Initial baseline and after-training values were based on the means of two samples taken on two separate days because there were no significant differences between these time points for GH, IGF-I, or testosterone. The after-training values represent the means of the values obtained 24 and 48 h after the last training session. All baseline and after-training values were

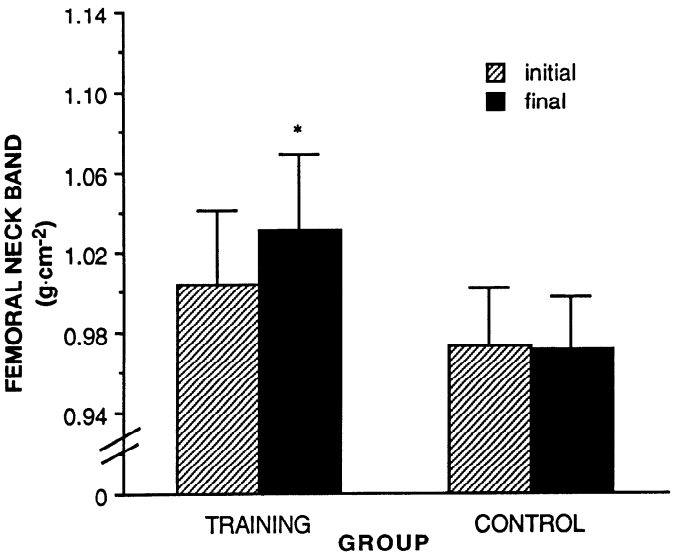


FIG. 4. BMD of femoral neck in strength-trained and control groups. Values are means ± SE. * Significantly different compared with initial value, $P < 0.001$.

within normal limits for this age group in both groups. There were no significant differences between initial and final serum GH (0.60 ± 0.15 vs. 0.75 ± 0.28 μg/l), IGF-I (171 ± 15 vs. 183 ± 18 μg/ml), or testosterone (8.63 ± 0.62 vs. 8.84 ± 0.59 μg/l) levels for the control group.

## DISCUSSION

The increases in femoral neck BMD observed in this study confirm those of a previous study from our laboratory but do not support the increases in indexes of bone formation also reported in that study (23). Furthermore, the results do not support the hypothesis that increases in BMD with ST are accompanied by elevations in serum concentrations of GH, IGF-I, or testosterone.

Portions of the present study were designed to replicate our previous study (23) while using a larger sample size and subjects who have higher baseline BMD values. It was stated in our previous report that "the possibility of regression toward the mean contributing to the BMD results cannot be ruled out" because the baseline values of the training group were lower than those of the control group (23). To determine the likelihood that the small sample size, low baseline BMD values, or regression toward the mean may have contributed to the findings in our previous study, it was necessary to keep all aspects of the training program in this study the same as they were

TABLE 3. *Bone markers for training and control groups*

| | Training Group | | Control Group | |
|---|---|---|---|---|
| | Before | After | Initial | Final |
| Osteocalcin, μg/l | 10.9±0.8 | 10.4±0.8 | 10.6±0.6 | 10.8±0.8 |
| Skeletal alkaline phosphatase, U/l | 7.6±0.9 | 9.3±1.4 | 8.2±0.4 | 8.2±0.7 |
| Tartrate-resistant acid phosphatase, U/l | 7.6±0.4 | 8.9±0.6* | 8.9±0.6 | 10.2±0.5* |

Values are means ± SE; $n$ = 21 and 16 men for training and control groups, respectively. * Significantly different from before training and initial value, $P < 0.05$.

TABLE 4. *Anabolic hormone values for training and control groups*

| | Training Group | | Control Group | |
|---|---|---|---|---|
| | Before | After | Initial | Final |
| Growth hormone, μg/l | 0.32±0.12 | 0.36±0.12 | 0.60±0.15 | 0.75±0.28 |
| Insulin-like growth factor I, μg/l | 192±8 | 191±13 | 171±15 | 183±18 |
| Testosterone, μg/l | 6.98±0.60 | 6.64±0.51 | 8.63±0.62 | 8.84±0.59 |

Values are means ± SE; $n$ = 13 and 9 men for training and control groups, respectively. None of the differences was significant.

in that study. The data from both of these reports suggest that the identical ST program can increase femoral neck BMD in older men regardless of whether baseline BMD values are low (23) or high (present study). In contrast, it appears that ST can increase BMD in the AP lumbar spine region in subjects with low BMD (23) but not in subjects with high BMD (present study). Nevertheless, it should be recognized that both studies had the limitation of a relatively short training period (4 mo). Frost (12) has argued that short-term increases in BMD measured by photon absorptiometry may reflect transient increases. In this regard, two of our subjects continued ST for an additional 5 mo after this study, for a total of 9 mo. They experienced an additional mean increase of 2% in femoral neck BMD. We realize that it would be improper to draw any conclusions from the data of only two subjects, but it does provide preliminary support for the hypothesis that our reported increases in femoral neck BMD in both studies may be more than mere transient rises. Clearly, longer duration studies are needed to clarify this issue.

The magnitude of change in the femoral neck BMD in the present study (2.8%) is greater than the magnitude of error in the technique (1%) previously reported (3), as well as in the variation observed in the control group (0.2%) or in the CV from the repeated trials testing in this study. Some investigators have not found any changes in femoral neck (26, 30) or lumbar spine BMD with ST (13), but they studied premenopausal women and used a less intense training program. Others have found increases in lumbar spine BMD (25, 35a) or a reduction in the loss over time with ST (28). No one factor could be identified to explain the discrepancies in these findings.

We are not sure why femoral neck BMD, but not lumbar spine BMD, increased with training. It is thought that changes in BMD from ST are related to the load placed on the proximal muscle groups during training. Although direct evidence for this belief is lacking, there were greater loads used during the leg press and leg extension exercises than were used for the abdominal and back musculature in the present study. Futhermore, two sets were performed for the lower limb exercises, whereas only one set was performed for the other exercises.

It was thought that the trabecular bone of the vertebral bodies ($B_2$–$B_4$) would be more responsive than cortical bone to ST because the trabecular bone is more metabolically active than cortical bone. Yet, no significant in-

creases in BMD of the vertebrae in the lateral spine were observed with training. Longer term studies are needed to see whether significant differences exist between training and control groups over a prolonged time period.

GH, IGF-I, and testosterone may be associated with bone remodeling (17, 29). Concentrations of GH and IGF-I decline with increasing age, and a decrease in bone-derived growth factors may contribute to the decrease in osteoblastic renewal in elderly individuals (16). Moreover, older men with low plasma IGF-I concentrations increase their lumbar BMD when given synthetic human GH (32). Testosterone levels also decline with age at approximately the same time as reductions in bone mass are observed (9, 24) and hyperprolactinemic hypogonadal males show increases in regional BMD when serum levels of testosterone are normalized (14). On the basis of these findings, it was hypothesized that IGF-I and anabolic hormones would contribute to increased BMD after ST, but the results failed to confirm this hypothesis. No changes in serum GH, IGF-I, or testosterone were observed in the training group. Thus, these factors did not explain the increases in femoral neck BMD. However, serum values may not adequately reflect what is occurring at the local site of action (bone cells). In addition, serum IGF-I levels represent total IGF-I because we were not able to isolate the concentration of free (biologically active) IGF-I. It may also be that starting with normal levels of GH, IGF-I, and testosterone makes it more difficult to change than when starting with deficiencies. Nevertheless, a related study by Craig et al. (8) also found no changes with ST despite higher initial baseline levels of serum GH. Although GH samples were taken on two separate days at the same time of day before and after training, more frequent sampling may have produced a better representation of baseline levels. Future studies should assess anabolic hormones and growth factors more frequently before and after a longer duration training program.

In contrast to the previous investigation from our lab (23), indexes of bone remodeling used in this study did not seem to explain the increase in femoral neck BMD. No significant changes in osteocalcin or SAP were observed with training, suggesting no significant changes in osteoblastic activity. The significant increase in TrACP in the training group does not indicate a training-induced increase in resorption because the values in the control group also increased significantly. One possible explanation for these results is a seasonal variation in TrACP, although we are not aware of any evidence to support this possibility. Some markers of bone turnover do show seasonal variations, whereas other indicators do not (36). Thus, it is unclear why the marker used for bone resorption increased in both groups, whereas neither marker of bone formation changed.

We were unable to identify any specific factors that would explain the discrepancy in findings of bone markers between our previous study (23) and the present one because both studies used almost identical procedures. The dietary intake of nutrients important for normal bone metabolism was similar between the training and control groups in both studies, and there were no dietary changes in the study groups while dietary records were kept. The gender and ages of the subjects as well as the training regime were almost identical. No factors related to either BMD or bone turnover that would explain responders vs. nonresponders could be identified. Only two subjects did not increase their femoral neck BMD. Further examination of the data revealed that BMD decreased in the other sites for these subjects. One of these subjects was the oldest and had the greatest increase in TrACP after training, but no other variables were significantly affected.

In conclusion, ST led to increases in femoral neck BMD but not in total body, AP or lateral lumbar spine, Ward's triangle, or trochanter BMD. This regional increase in BMD was not accompanied by changes in the markers of bone formation. Although a marker of bone resorption did increase significantly, it also increased in the control group, indicating that the increase was not a training effect. Furthermore, this increase in BMD was not associated with changes in serum levels of GH, IGF-I, or testosterone.

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