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# Strength training combined with plyometric jumps in adults: sex differences in fat-bone axis adaptations

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**Guadalupe-Grau A, Perez-Gomez J, Olmedillas H, Chavarren J, Dorado C, Santana A, Serrano-Sanchez JA, Calbet JA.** Strength training combined with plyometric jumps in adults: sex differences in fat-bone axis adaptations. *J Appl Physiol* 106: 1100–1111, 2009. First published February 5, 2009; doi:10.1152/japplphysiol.91469.2008.—Leptin and osteocalcin play a role in the regulation of the fat-bone axis and may be altered by exercise. To determine whether osteocalcin reduces fat mass in humans fed ad libitum and if there is a sex dimorphism in the serum osteocalcin and leptin responses to strength training, we studied 43 male (age 23.9 2.4 yr, mean ± SD) and 23 female physical education students (age 23.2 ± 2.7 yr). Subjects were randomly assigned to two groups: training (TG) and control (CG). TG followed a strength combined with plyometric jumps training program during 9 wk, whereas the CG did not train. Physical fitness, body composition (dual-energy X-ray absorptiometry), and serum concentrations of hormones were determined pre- and posttraining. In the whole group of subjects (pretraining), the serum concentration of osteocalcin was positively correlated ($r = 0.29–0.42$, $P < 0.05$) with whole body and regional bone mineral content, lean mass, dynamic strength, and serum-free testosterone concentration ($r = 0.32$). However, osteocalcin was negatively correlated with leptin concentration ($r = -0.37$), fat mass ($r = -0.31$), and the percent body fat ($r = -0.44$). Both sexes experienced similar relative improvements in performance, lean mass (+4–5%), and whole body (+0.78%) and lumbar spine bone mineral content (+1.2–2%) with training. Serum osteocalcin concentration was increased after training by 45 and 27% in men and women, respectively ($P < 0.05$). Fat mass was not altered by training. Vastus lateralis type II MHC composition at the start of the training program predicted 25% of the osteocalcin increase after training. Serum leptin concentration was reduced with training in women. In summary, while the relative effects of strength training plus plyometric jumps in performance, muscle hypertrophy, and osteogenesis are similar in men and women, serum leptin concentration is reduced only in women. The osteocalcin response to strength training is, in part, modulated by the muscle phenotype (MHC isoform composition). Despite the increase in osteocalcin, fat mass was not reduced.

exercise; adipose; performance; testosterone; cortisol

THERE IS A CROSS TALK BETWEEN the adipose tissue and the skeleton (30). Osteocalcin is a hormone produced by osteoblasts, which acts as a negative regulator of fat mass, protecting against diet-induced obesity in rodents (16). The adipose tissue produces leptin, whose concentration in blood is proportional to the fat mass (20). Contradictory effects of leptin on bone mass have been reported (17, 26). In cell cultures, leptin promotes proliferation of osteoblasts and collagen synthesis and mineralization (18). However, studies in rodents have revealed that, via a hypothalamic relay, leptin promotes bone loss (46). Human epidemiological studies have reported a positive association between leptin and bone mineral content (BMC) and density (BMD) in men and women (5, 38). However, when these analyses are adjusted for fat mass, leptin has been reported to be either positively (47) or negatively (5) associated with BMD.

Load-wearing exercises, strength training, and, in general, exercises that generate high tensions and impact on bones enhance plasma osteocalcin concentration and bone mass (19, 39, 50, 52). Few studies have directly compared the osteogenic response to mechanical stimulation in men and women (6, 19). However, no single study has determined the osteocalcin response to a training program combining strength training with jumping exercises in women and men, with their corresponding control groups. This information is needed to ascertain whether the osteoblastic response to exercise is similar in men and women.

An important factor, often overlooked, when studying the osteogenic effects of an exercise program is the accompanying level of muscle hypertrophy and the muscle phenotype [myosin heavy chain (MHC) composition] at the start of the training program. Muscle mass is independently associated with bone mass (22, 42, 51), and animal models like the myostatin knockout mouse showed increased bone mass associated with muscle hypertrophy (22). Fast-twitch muscle fibers are able to achieve higher rates of force development (41), eliciting faster strains on bones (53). Studies using training protocols equal in volume, intensity, and duration for both sexes indicate that, despite women having less initial strength and smaller myofibers than men, relative gains in strength (1, 21, 31, 44) and muscle hypertrophy (24, 31, 45) are similar in men and women. Although in men strength training does not seem to influence plasma leptin concentration, at least when body fat mass is not reduced (4), less is known about the effect of strength training on plasma leptin concentration in women. It remains unknown if there is interaction between the changes in plasma leptin, osteocalcin, fat mass, and muscle fiber type in response to strength training in humans. Since women train

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with lower absolute loads and submit their bones to lower absolute tensions, we hypothesized that women compared with men would have an attenuated osteogenic response to strength training that would be reflected in lower enhancement of plasma osteocalcin concentrations. We also hypothesized that subjects with a higher percentage of MHC II will experience greater elevation of plasma osteocalcin with strength training. Finally, we hypothesized that the increase in osteocalcin will be associated with a reduction in leptin and fat mass with training.

The aims of this study were to determine whether there is a sex dimorphism in the serum osteocalcin and leptin responses to strength training, and whether the changes in osteocalcin, leptin, and fat mass are associated, as well as to determine whether the osteocalcin and leptin responses to exercise are influenced by the skeletal muscle phenotype.

## METHODS

*Subjects.* The minimal sample size required to show a significant increase in osteocalcin of 4 ng/ml with training was estimated to be of five subjects per group, with a significance level of 5% and a statistical power of 80%. The sample size required to show a significant difference in osteocalcin concentration between men and women of 1.5 SD was estimated to be of eight subjects per group. Subjects were recruited through advertisements in the Faculty of Sports Sciences, at the University of Las Palmas de Gran Canaria. In total, 57 male and 31 female physical education students agreed to participate in the study. Twenty-one men and 13 women were randomly assigned to the strength training group (TG) and the rest, i.e., 36 men and 18 women, served as the control group (CG) (Fig. 1). All subjects were queried about their usual daily intake of dairy products to calculate the amount of calcium ingested, as previously reported (53). Exclusion criteria included a history of endocrine, renal, or metabolic diseases, or any medication known to influence calcium metabolism. None of the subjects taking part in the study had any medical conditions known to affect bone metabolism, eating disorders, or amenorrhea. None of our volunteers accomplished exclusion criteria.

As described in Fig. 1, 13 men and 3 women from the CG were excluded due to incomplete adherence to the study requisites (starting an exercise program during the study and missing tests were the main reasons). One man and five women from the TGs were also excluded for the same reasons. Thus the study was finalized with 43 male physical education students (age 23.9 ± 2.4 yr, height 176.7 ± 7.1 cm, body mass 73.2 ± 10.1 kg; means ± SD) and 23 female physical education students (age 23.2 ± 2.7 yr, height 164.6 ± 6.3 cm, body mass 59.9 ± 5.8 kg; means ± SD). As depicted in Table 1, CG and TG had comparable physical characteristics and similar levels of fitness at the start of the training program. All of the subjects were healthy, physically active, nonsmokers and were not taking drugs or medications. CGs were physical education students, with moderate

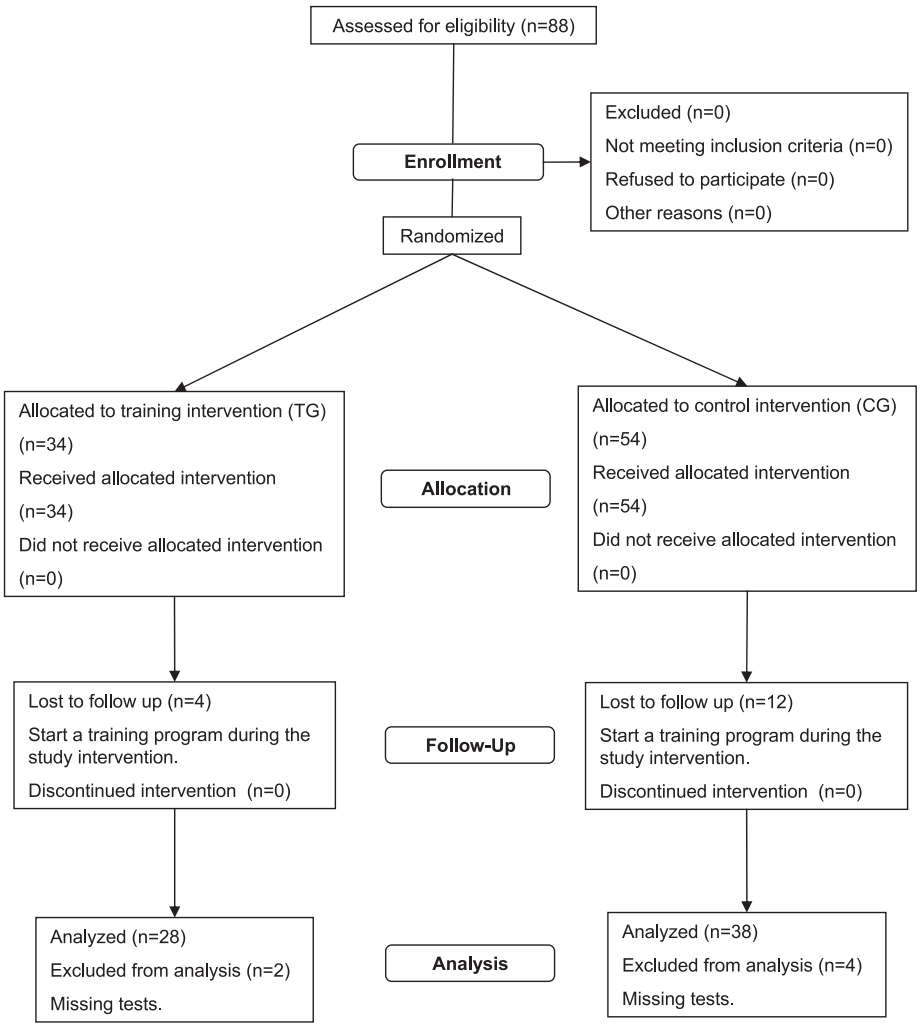


Fig. 1. Flow chart describing recruitment, allocation, and dropout. TG, training group; CG, control group.

Table 1. *Subject's characteristics at the beginning of the study*

| | Men | | Women | |
|---|---|---|---|---|
| | Control group | Training group | Control group | Training group |
| Age, yr | 24.6±2.4 | 23.13±2.2 | 23.7±2.7 | 22.3±2.7 |
| Height, cm | 174.8±6.8† | 174.7±7.1† | 162.5±5.5 | 168.5±6.0* |
| Weight, kg | 76.3±11.2† | 69.5±7.3*† | 58.9±6.2 | 61.7±4.8 |
| BMI, kg/m$^2$ | 24.0±3.6 | 22.8±2.0 | 22.3±2.1 | 21.7±1.2 |
| Fat mass, % | 14.5±3.9† | 15.8±6.4† | 27.6±5.7 | 28.0±4.6 |
| Calcium intake, mg·kg body mass$^{-1}$·day$^{-1}$ | 10.6±5.7 | 11.0±6.3 | 9.6±6.4 | 13.0±6.4 |
| ILP, kg | 253.9±73.5 | 211.8±42.0 | 155.9±19.3 | 119.4±19.4 |
| LE, kg | 70.5±13.2 | 68.0±11.4 | 42.8±6.8 | 47.5±8.0 |
| HS, kg | 156.6±36.6 | 150.3±28.5 | 110.8±18.3 | 106.3±17.7 |
| LC, kg | 58.4±10.0 | 55.4±8.2 | 32.8±6.3 | 37.5±5.3 |
| MIF, kg | 115.6±23.8 | 112.0±20.4 | 77.5±12.2 | 73.0±8.7 |
| 30 m, s | 4.4±0.2 | 4.4±0.2 | 5.0±0.2 | 4.8±0.2 |
| 300 m, s | 47.0±2.8 | 46.3±2.1 | 59.6±3.7 | 55.3±2.6 |

Values are means ± SD. BMI, body mass index; ILP, inclined leg press; LE, leg extension; HS, half squat; LC, leg curl; MIF, maximal isometric force; 30 m, 30-m sprint test; 300 m, 300-m all-out test. Calcium intake is expressed as milligrams per kilogram body mass per day (mg·kg body mass$^{-1}$·day$^{-1}$). *$P < 0.05$ vs. respective control subjects; †$P < 0.05$, men vs. women in the same group.

levels of physical activity, as required by their academic activities. Control subjects that start any kind of regular training were excluded from the analysis. The study was performed in accordance with the Helsinki Declaration of 1975, as last modified in 2000 regarding the conduct of clinical research, and was approved by the Ethical Committee of the University of Las Palmas de Gran Canaria. Subjects provided their written consent before participating in the study.

*Study design.* The study protocol consisted of 9 wk, 3 days/wk of supervised experimental strength training combined with plyometric jumps (see below). Each subject performed two test sessions, one before and one after 9 wk of training. In both sessions, body composition was measured by dual-energy X-ray absorptiometry (DXA).

The maximum weight that could be lifted at least once per exercise [one repetition maximum (1 RM)] was used to determine the training loads before and after the training intervention period and to assess the improvements in strength. Baseline testing was completed during the first week. Resting blood samples were drawn at baseline and at the end of the study, 48–72 h after the last training session. Venous blood samples from an antecubital vein were taken between 7 and 8 AM after an overnight fasting. The blood samples were allowed to clot at 4°C and then were centrifuged (3,500 rpm, 15 min) at the same temperature (Allegra 21R, Beckman Instruments, Fullerton, CA). The serum obtained was separated and frozen at −80°C for later analysis.

*Anthropometry and body composition.* Anthropometric measurements were obtained on each subject. Height was measured in the upright position to the nearest millimeter (Atlántida, Barcelona, Spain). Body mass was determined using a balance with a 50-g imprecision (Atlántida, Barcelona, Spain), calibrated with M1 calibration masses (tolerance <0.005% in mass). Total and regional body composition were assessed by DXA (Hologic QDR-1500, Hologic, software version 7.10, Waltham, MA), as described elsewhere (10, 11). DXA equipment was calibrated using a lumbar spine phantom and following the Hologic guidelines. Subjects were scanned in the supine position, and the scans were performed in high resolution. Lean mass (g), fat mass (g), total area (cm$^2$), and BMC (g) were calculated from total and regional analysis of the whole body scan. BMD (g/cm$^2$) was calculated using the formula BMD = BMC·area$^{-1}$. Two additional examinations were conducted to estimate bone mass at the lumbar spine and proximal region of the femur. BMC and BMD values of the femoral neck, greater trochanter, intertrochanteric, and Ward's triangle subregions are also reported. The coefficient of variation for the assessment of whole body BMC (WBBMC) and BMD was 0.4 and 0.7, respectively. The coefficient of variation for the assessment of lumbar spine BMC and BMD was below 0.7% and that for the assessment of femoral regions below 1.7%. The coefficient of variation for the assessment of whole body fat and lean masses was 3 and 1%, respectively (11).

*Strength assessment and strength-training program.* The TG followed a training program consisted of three sessions per week, during 9 wk (Table 2). During the first part of the training session, subjects performed plyometric exercises: drop jumps and hurdles, five hurdles 1 m apart fixed at 50 cm of height. The second part of each training session consisted of four weight-lifting exercises performed in this order: inclined leg press (ILP), leg extension (LE), half squat (HS), and leg curl (LC). These exercises were executed on weight-lifting exercise machines (Technogym, Barcelona, Spain). The HS and ILP exercises were performed with a range of motion between full extension and a knee angle of 90°. All of the available range of motion was used during the LE. For the LC, each subject lifted the device until contact with the thigh. Verbal feedback and encouragement were provided to facilitate a correct execution of exercises. A 90-s rest period was allowed between exercise sets (Table 2).

Maximum strength (1 RM) for all exercises used during training was assessed immediately before and at the end of the strength training period. Before the 1-RM attempt, subjects warmed up by doing 10 min of stationary cycling followed by 10 repetitions lifting ~50% of perceived maximum. Then they performed two lifts with progressively heavier weights until the 1 RM was determined. To minimize fatigue, 3- to 5-min resting periods were allowed between trials. The 1-RM values obtained were subsequently used to calculate the relative loads for the training protocol. The relative loads for each exercise ranged between 50 and 90% of the 1-RM load (Table 2). To ensure this training intensity, the 1-RM test was repeated every 3 wk, and the load adjusted accordingly. All subjects finished the training program, completing a total of 536 sets. The adherence of the subjects to the training program with regard to the total number of sessions planned was 85%.

*Maximal isometric force.* The maximal isometric force (MIF) during LE in the squat position (knees bent at 90°, hip bent at 110°) was measured with a force plate (Kistler, Winterthur, Switzerland), as described previously (11). Briefly, during 6 s, subjects were encouraged to exert the highest strength against a fixed bar positioned across the shoulders in the lowest time. The best of three attempts, with 5-min resting periods in between, was recorded. MIF (N) was determined as the highest value of the force produced. MIF per kilogram body weight was also assessed.

*Anaerobic capacity.* A 300-m running test was used to estimate the anaerobic capacity. The anaerobic capacity is the first determinant of performance in maximal all-out efforts eliciting exhaustion between 30 and 60 s (9). The test was performed in a 400-m track, and timings were measured manually with a digital stopwatch. Subjects were asked to run the 300 m as fast as possible.

*Running speed test.* Following an individual warm-up, subjects performed three maximal indoor short sprint trials, each separated by at least 5 min. The time needed to cover 30 m was measured with photoelectric cells (General ASDE, Valencia, Spain). The timer is automatically activated when the subject crosses the first cell and every 5 m thereafter. The subjects were encouraged to run as fast as possible. A standing start was used, and the best of three trials was selected as the representative value of this test (52).

*Hormonal assays.* Blood samples were obtained at baseline and after 9 wk of training to determine serum concentrations of osteocalcin, leptin, free testosterone, and cortisol. All concentrations were determined in duplicate by ELISA (ELx800 Universal Microplate Reader, Bioteck Instruments), using reagent kits from Nordic Bioscience Diagnostics (Herlev, Denmark) for osteocalcin, Linco Re-

Table 2. *Training program*

| Week | Session | Weight Lifting | | | Plyometric Exercises | | |
|---|---|---|---|---|---|---|---|
| | | 1 RM, % | Sets, no. | Repetitions, no. | Drop jump sets, no. | Hurdles, no. | Jumps, no. |
| 1 | 1 | 50–70–90 | 1-1-1 | 12-6-2 | 4 × 5 (40 cm) | 4 × 5 | 40 |
| | 2 | 50–70–90 | 1-2-1 | 12-6-2 | 5 × 5 (40 cm) | 5 × 5 | 50 |
| | 3 | 50–70–90 | 1-3-1 | 12-6-2 | 6 × 5 (40 cm) | 6 × 5 | 60 |
| 2 | 1 | 50–70–90 | 1-3-1 | 12-6-2 | 5 × 5 (40 cm) | 5 × 5 | 50 |
| | 2 | 50–70–90 | 1-3-2 | 12-8-3 | 6 × 5 (40 cm) | 6 × 5 | 60 |
| | 3 | 50–70–90 | 1-3-1 | 12-8-2 | 7 × 5 (40 cm) | 7 × 5 | 70 |
| 3 | 1 | 50–80–90 | 1-3-2 | 12-8-3 | 5 × 5 (50 cm) | 5 × 5 | 50 |
| | 2 | 50–80–90 | 1-3-2 | 12-8-3 | 6 × 5 (50 cm) | 6 × 5 | 60 |
| | 3 | 50–80–90 | 1-3-2 | 12-8-3 | 7 × 5 (50 cm) | 7 × 5 | 70 |
| 4 | 1 | 50–70–90 | 1-3-1 | 12-6-2 | 6 × 5 (50 cm) | 6 × 5 | 60 |
| | 2 | 50–70–90 | 1-2-1 | 12-8-2 | 7 × 5 (50 cm) | 7 × 5 | 70 |
| | 3 | 50–70–90 | 1-3-1 | 12-10-2 | 8 × 5 (50 cm) | 8 × 5 | 80 |
| 5 | 1 | 50–70–90 | 1-3-1 | 12-10-2 | 6 × 5 (60 cm) | 6 × 5 | 60 |
| | 2 | 50–70–90 | 1-3-1 | 12-10-3 | 7 × 5 (60 cm) | 7 × 5 | 70 |
| | 3 | 50–70–90 | 1-3-2 | 12-10-2 | 8 × 5 (60 cm) | 8 × 5 | 80 |
| 6 | 1 | 50–80–90 | 1-3-1 | 12-8-3 | 7 × 5 (60 cm) | 7 × 5 | 70 |
| | 2 | 50–80–90 | 1-3-2 | 12-8-3 | 8 × 5 (60 cm) | 8 × 5 | 80 |
| | 3 | 50–80–90 | 1-3-2 | 12-8-3 | 9 × 5 (60 cm) | 9 × 5 | 90 |
| 7 | 1 | 50–70–90 | 1-3-1 | 12-6-2 | 5 × 5 (70 cm) | 5 × 5 | 50 |
| | 2 | 50–70–90 | 1-2-1 | 12-8-2 | 6 × 5 (70 cm) | 6 × 5 | 60 |
| | 3 | 50–70–90 | 1-3-1 | 12-10-2 | 7 × 5 (70 cm) | 7 × 5 | 70 |
| 8 | 1 | 50–70–90 | 1-3-1 | 12-3-2 | 9 × 5 (60 cm) | 9 × 5 | 75 |
| | 2 | 50–70–90 | 1-3-1 | 12-10-3 | 10 × 5 (60 cm) | 10 × 5 | 85 |
| | 3 | 50–70–90 | 1-3-2 | 12-10-2 | 11 × 5 (60 cm) | 11 × 5 | 95 |
| 9 | 1 | 50–80–90 | 1-3-1 | 12-8-3 | 5 × 5 (80 cm) | 5 × 5 | 50 |
| | 2 | 50–80–90 | 1-3-2 | 12-8-3 | 6 × 5 (70 cm) | 6 × 5 | 60 |
| | 3 | 50–80–90 | 1-3-2 | 12-8-3 | 7 × 5 (70 cm) | 7 × 5 | 70 |

1 RM, one repetition maximum.

search (St. Charles, MO) for leptin, and Diagnostic Systems Laboratories (Webster, TX) for free testosterone and cortisol. Intra- and interassay coefficients of variation were 6.7 and 6.7% for osteocalcin, 2.6 and 3.7% for leptin, 6.5 and 3.1% for free testosterone, and 5.9 and 8.7% for cortisol, respectively. 17β-Estradiol was measured by a competitive electrochemiluminescence immunoassay intended for use on Modular Analytics analyzer E170 using E2 reagents (Roche/Hitachi, 03000079122, Indianapolis, IN). Results were determined via a calibration curve with analytic sensitivity of 18.4 pmol/l.

The free testosterone-to-cortisol molar ratio was calculated as an indication of the balance between anabolic and catabolic activity within the tissues and was calculated as free testosterone in nanomoles per liter and cortisol in micromoles per liter, as previously reported (2). Low-end sensitivities for osteocalcin, leptin, free testosterone, and cortisol were 0.5 ng/ml, 0.5 ng/ml, 0.19 pg/ml, and 0.1 μg/dl, respectively.

*Muscle biopsies.* Needle muscle biopsies were obtained from the middle section of the vastus lateralis muscle under local anesthesia without suction, but with mild pressure on the lateral aspect of the thigh. Pre- and posttraining muscle biopsies were obtained from 48 of the subjects (22 from the TGs and 26 from the CGs). The muscle samples were immediately mounted with Tissue-Tek and frozen in isopentane cooled with liquid nitrogen and stored at −80°C. MHC analyses were performed on the muscle biopsies using SDS-PAGE, as reported by Larsson et al. (29). From each biopsy, 20–40 serial cross sections (10 μm) were cut and placed in 200–500 μl of lysing buffer and heated for 3 min at 90°C. Between 2 and 12 μl of the myosin-containing samples were loaded on a SDS-PAGE. Gels were run at 70 V for 43 h at 4°C. Subsequently, the gels were Coomassie stained, and MHC isoform bands (I, IIa, IIx) were determined based on known migration patterns and quantified with the image analysis software Quantity One from Bio-Rad Laboratories (Hemel, Hempstead, Hertfordshire, UK).

*Statistical analyses.* Repeated-measures ANOVA was used to determine changes between baseline and the end of the training program; a group by sex interaction was included in this analysis. Analysis of covariance with the percentage of body fat as covariate was used to determine whether changes in serum leptin concentrations could be accounted for by changes in fat mass. Pearson correlation coefficients were determined to evaluate relationships among variables of body composition, hormones, and physical performance tests. All statistical tests were two tailed. Data are expressed as means ± SD, and significance was set at the $P \leq 0.05$ level.

## RESULTS

*Cross-sectional analysis.* In the whole group of subjects, the basal serum concentration of osteocalcin was positively correlated with WBBMC ($r = 0.31$; $P < 0.05$) (Fig. 2), whole body lean mass ($r = 0.43$; $P < 0.01$) (Fig. 2), lower extremities BMC ($r = 0.36$; $P < 0.01$), BMD ($r = 0.29$; $P < 0.05$), and lean mass ($r = 0.42$; $P < 0.01$), ILP 1 RM ($r = 0.27$; $P < 0.05$), LE 1 RM ($r = 0.27$; $P < 0.05$), LC 1 RM ($r = 0.35$; $P < 0.01$), and serum-free testosterone concentration ($r = 0.41$; $P < 0.01$) (Fig. 3). In contrast, osteocalcin concentration was negatively correlated with leptin serum concentration ($r = -0.37$; $P < 0.01$) (Fig. 2), cortisol serum concentration ($r = -0.39$; $P < 0.01$) (Fig. 3), body fat mass ($r = -0.31$; $P < 0.05$) (Fig. 2), the percentage of body fat mass ($r = -0.44$; $P < 0.01$) (Fig. 2), and 30-m running time ($r = -0.43$; $P < 0.01$).

Serum leptin concentration correlated with the percentage of MHC IIx in men ($r = 0.36$, $P < 0.05$, $n = 31$).

*Effects of strength training on performance.* CGs and TGs were comparable at the beginning of the study (Table 1). Both sexes experienced improvements with training in dynamic strength (1 RM) and MIF [maximal voluntary contraction (MVC)] (Table 3, Fig. 4*A*). There was a large variation in the relative improvement in dynamic strength between exercises

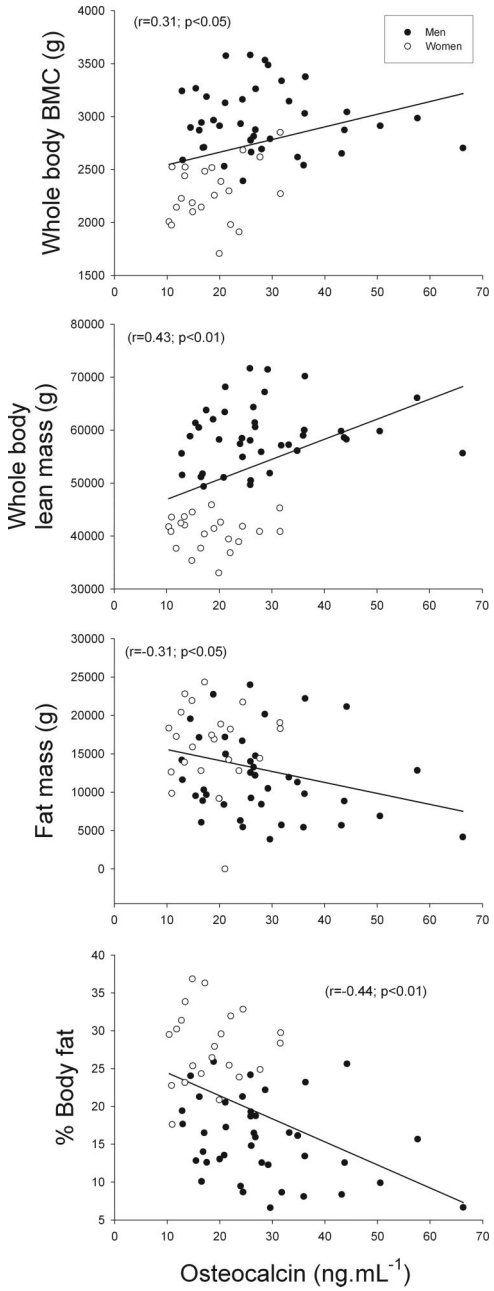


Fig. 2. Relationship between serum osteocalcin concentration and whole body bone mineral content (BMC), lean mass, fat mass, and percentage of body fat in 40 men (●) and 22 women (○).

ranging from 16 to 92%, but dynamic strength was enhanced similarly in men and women (Fig. 4*A*). A sex dimorphism was observed in the improvement of MVC (+17.2 and +14.0% in men and women, respectively, $P < 0.01$; time × sex interaction, $P < 0.01$). However, the MVC per kilogram of lean mass in the lower extremities was increased similarly in both sexes with training (+8.3 and +7.4% in men and women, respectively, $P < 0.01$ and $P < 0.05$; group by time interaction, $P < 0.01$) (Fig. 4*B*).

Only the women from the TG significantly improved the 30-m running speed (time by sex interaction, $P < 0.05$, Table 3). Performance in the 300-m running test was not significantly affected by training (group by time interaction, $P = 0.83$; sex by group by time interaction, $P = 0.97$, Table 3).

*Hormonal responses to training.* A between-sex comparison in serum hormone concentrations before and after training is reported in Table 4. Women had higher serum cortisol, leptin, and estradiol concentrations than men. Men had higher serum free testosterone and osteocalcin concentrations than women (Table 4).

The basal serum concentrations of free testosterone, cortisol, the free testosterone-to-cortisol ratio, and 17β-estradiol were not affected by training (group by time interaction: $P = 0.32$, $P = 0.21$, $P = 0.22$, and $P = 0.96$, respectively, Table 4).

Serum leptin concentration was reduced with training (group by time interaction: $P < 0.05$, Table 4), but only in women (sex by time interaction in the TG: $P = 0.009$) (Fig. 3, *top*). This difference remained significant after accounting for the percentage of body fat as a covariable.

Serum osteocalcin concentration was increased after strength training by 45 and 27% in men and women, respectively (ANOVA time effect: $P < 0.001$, Table 5), while it remained unchanged in the control subjects (group by time interaction,

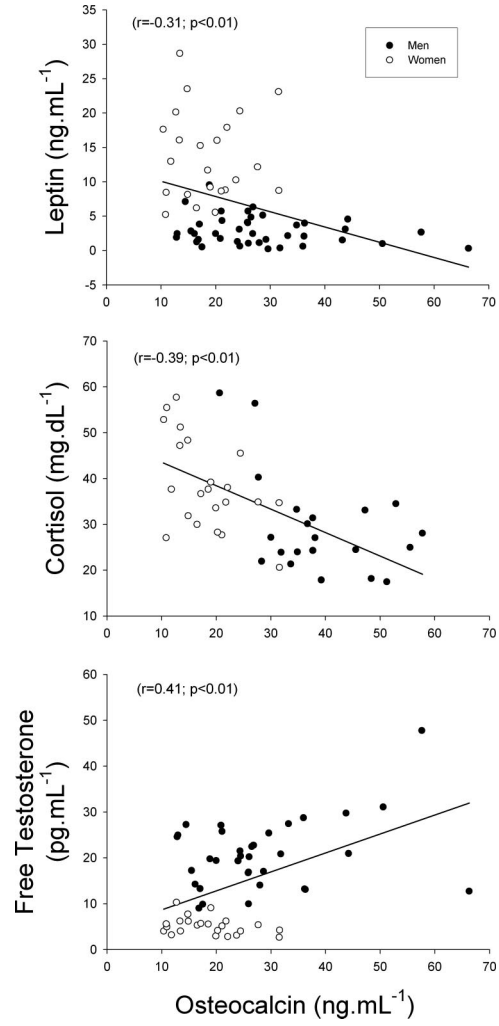


Fig. 3. Relationship between serum osteocalcin concentration and the serum concentrations of leptin ($n = 63$), cortisol ($n = 61$), and free testosterone ($n = 59$).

Table 3. *Physical performance before and after strength training combined with jumps*

| Performance Test | Men | | | | | | Women | | | | | | Interaction Group × Time | Interaction Group × Time × Sex |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Control group | | | Training group | | | Control group | | | Training group | | | | |
| | Pre | Post | %Change | Pre | Post | %Change | Pre | Post | %Change | Pre | Post | %Change | | |
| ILP, kg | 253.9±73.5 | 265.9±75.2 | 4.7 | 211.8±42.0 | 360.3±68.0 | 70.1* | 155.9±19.3 | 160.0±24.4 | 2.6 | 119.4±19.4 | 228.7±19.8 | 91.5* | $P = 0.001$ | $P = 0.08$ |
| LE, kg | 70.5±13.2 | 72.3±15.4 | 2.5 | 68.0±11.4 | 85.3±14.8 | 25.5* | 42.8±6.8 | 42.4±6.8 | −0.9 | 47.5±8.0 | 62.3±9.9 | 31.1* | $P = 0.001$ | $P = 0.85$ |
| HS, kg | 156.6±36.6 | 157.5±34.2 | 0.6 | 150.3±28.5 | 221.3±39.0 | 47.2* | 110.8±18.3 | 114.2±20.0 | 3.1 | 106.3±17.7 | 155.5±19.6 | 46.3* | $P = 0.001$ | $P = 0.12$ |
| LC, kg | 58.4±10.0 | 58.5±10.4 | 0.2 | 55.4±8.2 | 64.0±8.4 | 15.5* | 32.8±6.3 | 32.3±6.4 | −1.5 | 37.5±5.3 | 46.1±4.4 | 22.9* | $P = 0.001$ | $P = 0.86$ |
| MIF, kg | 115.6±23.8 | 107.5±23.7 | −7.0* | 112.0±20.4 | 131.2±26.7 | 17.1* | 77.5±12.2 | 78.6±16.5 | 1.4 | 73.0±8.7 | 83.1±8.7 | 13.8* | $P = 0.001$ | $P = 0.005$ |
| RMIF, kg/kg | 1.52±0.27 | 1.41±0.31 | −7.2† | 1.61±0.33 | 1.85±0.39 | 14.9* | 1.33±0.20 | 1.33±0.20 | 0.0 | 1.17±0.12 | 1.32±0.17 | 12.8* | $P = 0.001$ | $P = 0.03$ |
| MIF/LLM, kg/kg | 11.40±2.06 | 10.41±2.23 | −9.5† | 12.15±2.71 | 13.53±2.92 | 11.36* | 12.17±1.79 | 12.01±2.19 | −1.31 | 10.8±1.2 | 11.67±1.32 | 8.1* | $P = 0.001$ | $P = 0.09$ |
| 30 m, s | 4.4±0.2 | 4.4±0.2 | 0.0 | 4.4±0.2 | 4.4±0.1 | 0.0 | 5.0±0.2 | 5.1±0.2 | 2.0* | 4.8±0.2 | 4.7±0.1 | −2.1* | $P = 0.02$ | $P = 0.034$ |
| 300 m, s | 47.0±2.8 | 47.2±3.0 | 0.4 | 46.3±2.1 | 46.4±2.6 | 0.2 | 59.6±3.7 | 59.2±3.8 | −0.7 | 55.3±2.6 | 54.8±3.1 | −0.9 | $P = 0.84$ | $P = 0.97$ |

Values are means ± SD. Pre, pretraining; Post, posttraining; RMIF, maximal isometric per kilogram body weight; MIF/LLM, MIF per lower limb lean mass. Dynamic strength tests were missed in 4 women from the control group. MIF was missed by 6 subjects (2 training men, 2 training women, and 2 control women).The 30-m test was missed by 5 subjects (2 training men and 2 training women, and 1 control woman). The 300-m test was missed by 6 subjects (2 training men, 1 training woman, and 3 control women). *$P < 0.05$ and †$P < 0.01$: Pre vs. Post comparison.

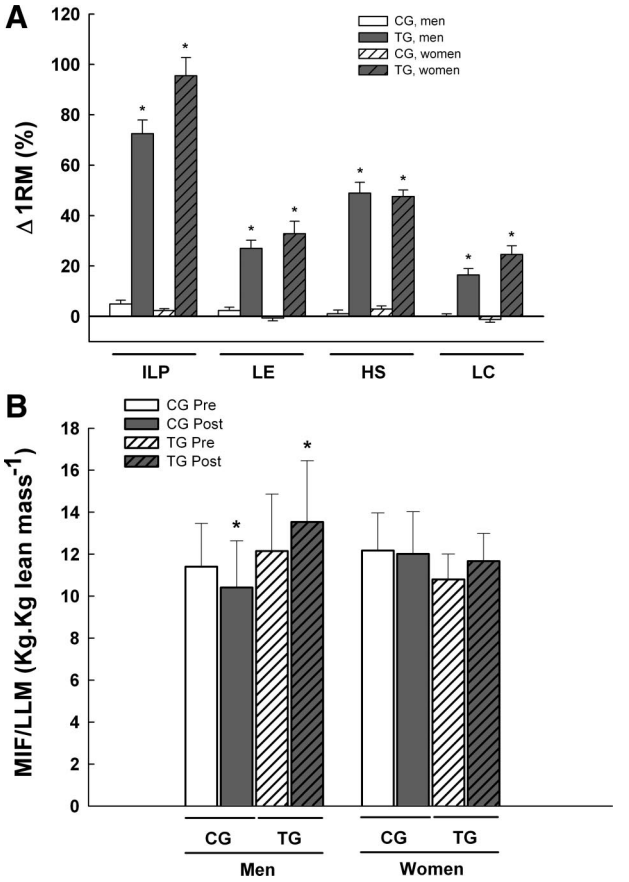


Fig. 4. *A*: effects of strength training combined with jumps on one repetition maximum (1 RM). ILP, inclined leg press; LE, leg extension; HS, half squat; LC, leg curl; %Δ1RM, relative increases of 1 RM expressed as percentage. Open bars, CGs; shaded bars, TGs; hatched bars, women; nonhatched bars, men. *$P < 0.01$, group by time interaction. *B*: changes in maximal isometric force per kilogram lower extremity lean mass (MIF/LLM) expressed in kilograms per kilogram lean mass, after a 9-wk intervention period. Values are means ± SE. *$P < 0.01$ before vs. after training.

$P < 0.01$, Table 4) (Fig. 3, *middle*). The sex differences in the osteocalcin response to training did not reach statistical significance (sex × time interaction: $P = 0.15$, power = 0.30, SE = 0.09).

*Fat and lean body mass.* Fat mass was not significantly altered by strength training. Lean body mass was increased by 1.6 and 1.4% in men and women from the TG, respectively (ANOVA time effect: $P < 0.001$, Table 5), without group by time effects ($P = 0.37$, Table 5), or sex by group by time effects ($P = 0.50$, Table 5). The increase in lean body mass with training occurred principally in the lower extremities, where it increased by 4.5 and 5.3% in men and women from the TG, respectively (Table 5). Although the lean mass of the lower extremities was increased by 2.0 and 2.8% in men and women from the CG, respectively (Table 5), a group by time interaction was also found (ANOVA group by time effect, $P < 0.03$, Table 5), meaning that the enhancement in lean mass was significantly greater in the TG compared with the CG.

*Effects on bone mass.* WBBMC was increased with training by 0.78% ($P < 0.001$, Table 5), while it remained unchanged in the CG (group by time interaction: $P < 0.05$, Table 5). The response was similar in men and women (sex × time interac-

Table 4. *Serum hormones before and after strength training combined with jumps*

| | Men | | | | | | Women | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Control group | | | Training group | | | Control group | | | Training group | | | Interaction Group × Time | Interaction Group × Time × Sex |
| | Pre | Post | %Change | Pre | Post | %Change | Pre | Post | %Change | Pre | Post | %Change | | |
| Free testosterone, pg/ml | 21.1±8.9 | 19.6±6.6 | −7.1 | 24.0±14.3 | 25.3±11.5 | 5.4 | 5.0±1.8† | 4.9±1.9 | −2.0 | 5.4±2.2† | 5.8±3.5 | 7.4 | $P = 0.32$ | $P = 0.51$ |
| Cortisol, mg/dl | 29.1±10.9 | 28.4±6.9 | −2.4 | 33.0±8.7 | 31.0±9.9 | −6.0 | 39.7±8.0† | 40.5±9.4 | 2.0 | 36.9±13.2 | 33.3±8.2 | −9.7 | $P = 0.21$ | $P = 0.49$ |
| FTCR | 0.11±0.04 | 0.10±0.03 | −9.1 | 0.11±0.06 | 0.12±0.05 | 9.1 | 0.02±0.007† | 0.02±0.007 | 0.0 | 0.02±0.009† | 0.02±0.01 | 0.0 | $P = 0.22$ | $P = 0.29$ |
| Leptin, ng/ml | 3.0±2.5 | 2.9±2.0 | −3.3 | 2.7±1.4 | 2.3±1.1 | −4.8 | 13.7±6.8† | 15.5±7.7 | 13.1 | 13.6±6.1† | 11.7±6.0 | −14.0 | $P = 0.02$ | $P = 0.05$ |
| Estradiol, pg/ml | 21.5±16.2 | 21.3±15.5 | −0.9 | 25.5±15.8 | 23.4±11.8 | −8.2 | 62.5±66.5† | 49.1±41.3 | −21.5 | 80.6±57.6† | 68.2±82.0 | 15.4 | $P = 0.96$ | $P = 0.89$ |
| Osteocalcin, ng/ml | 31.9±14.0 | 31.5±11.5 | −1.2 | 23.5±7.1 | 34.0±11.4 | 44.7* | 17.7±4.4† | 18.8±7.1 | 6.2 | 20.3±8.9† | 25.8±11.0 | 27.1±14.0* | $P = 0.007$ | $P = 0.24$ |

Values are means ± SD. FTCR, free testosterone cortisol ratio. There were 3 missing values for osteocalcin (2 training men and 1 control man), 2 for leptin (2 training men), 4 for cortisol (2 training men, 1 control man, and 1 control woman), 3 for estradiol (3 training men), and 6 for free testosterone (4 training men and 2 control men). *$P < 0.05$, Pre vs. Post comparison. †$P < 0.05$ compared with men (same time point).

tion in the TG, $P = 0.68$, Table 5). In contrast, whole body BMD was only increased in the men from the TG (from 1.25 ± 0.07 to 1.26 ± 0.07; g/cm$^2$, $P < 0.01$, Table 5), revealing a significant group by time by sex interaction ($P < 0.02$, Table 5).

Lumbar spine ($L_1 + L_2 + L_3 + L_4$) BMC was increased with training similarly in both groups (2.0 and 1.2%, in men and women, respectively, $P < 0.001$, Table 5). However, the group × time interaction did not reach statistical significance ($P = 0.21$, Table 5), and there was no significant sex effect. Although overall effects on lumbar spine BMD were not significant, there was a trend for a group × time interaction in the TG ($P = 0.07$, Table 5). This effect was due to the 2.2% increase in lumbar BMD observed in men from the TG ($P < 0.01$, Table 4).

There was no significant effects in the BMC and BMD of the hip regions except for femoral neck BMC, which increased with training (group × time interaction: $P < 0.05$, Table 5), without significant differences in the responses between sexes (sex × time interaction in the TG: $P = 0.31$, Table 5).

*MHC isoform distribution.* Men and women had a similar MHC distribution before strength training (Table 6). Strength training in men resulted in an increased proportion of MHC type IIa (+22%; ANOVA time effect: $P < 0.01$) and a reduction in the amount of MHC type I (−9.22%; ANOVA time effect: $P < 0.02$) and type IIx (−89% $P < 0.05$). No significant changes were observed in women (Table 5, Fig. 5), due to insufficient statistical power ($n = 6$).

*Relationships between changes in body composition, physical performance, and hormonal concentrations.* In the whole group of subjects, we observed correlations between the changes in osteocalcin and the changes in lower extremity lean mass ($r = 0.31$, $n = 62$, $P < 0.05$) and the changes in HS 1 RM ($r = 0.27$, $n = 59$, $P < 0.05$). In the whole female group, the changes in osteocalcin correlated with the changes in serum free testosterone ($r = 0.57$, $n = 23$, $P < 0.05$), lower extremity lean mass ($r = 0.42$, $n = 22$, $P < 0.05$), and LP 1 RM ($r = 0.46$, $n = 20$, $P < 0.05$).

There was no relationship between the changes in osteocalcin and the changes in either body composition or performance with training. The changes in serum leptin concentration with training correlated positively with the changes in body fat mass and percentage of body fat ($r = 0.49$ and 0.45, respectively, $n = 25$, both $P < 0.05$), and negatively with the changes in serum cortisol ($r = −0.41$, $n = 25$, $P < 0.05$). The association between leptin changes and fat mass changes was statistically significant in men ($r = 0.47$, $n = 18$, $P = 0.05$) but not in women ($r = 0.46$, $n = 8$, $P = 0.25$). The gain of lean mass in the lower extremities correlated with the gain in BMC in this region ($r = 0.45$, $n = 27$, $P < 0.05$).

In the TG, the change in osteocalcin tended to be positively associated with the change in leptin in men ($r = 0.46$, $n = 18$, $P = 0.056$) and inversely in women ($r = −0.57$, $n = 8$, $P = 0.14$).

*Muscle phenotype influences the osteocalcin response to strength training.* There was a positive correlation between the percentage of MHC II at the beginning of the training program and the increase in osteocalcin with strength training ($r = 0.50$, $P < 0.05$, $n = 22$) (Fig. 6).

Table 5. *Body composition before and after strength training combined with jumps*

| DXA Measurement | Men | | | | | | Women | | | | | | Interaction Group × Time | Interaction Group × Time × Sex |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Control group | | | Training group | | | Control group | | | Training group | | | | |
| | Pre | Post | % Change | Pre | Post | % Change | Pre | Post | % Change | Pre | Post | % Change | | |
| Whole body | | | | | | | | | | | | | | |
| BMC, g | 2,986±327 | 2,994±317 | 0.3 | 2,903±269 | 2,921±266 | 0.6* | 2,207±269 | 2,208±280 | 0.0 | 2,418±267 | 2,441±271 | 1.0* | $P = 0.018$ | $P = 0.38$ |
| BMD, g/cm$^2$ | 1.24±0.07 | 1.24±0.07 | 0.0 | 1.25±0.07 | 1.26±0.07 | 0.8* | 1.11±0.08 | 1.12±0.08 | 0.9* | 1.15±0.08 | 1.15±0.08 | 0.0 | $P = 0.97$ | $P = 0.017$ |
| LM, g | 60,669±5,725.6 | 61,087±5,683.4 | 0.7 | 5,6397±5,199.6 | 57,330±5,751.8 | 1.6* | 40,025±3,650.6 | 40,552±3,706.8 | 1.3 | 42,172±1,661.0 | 42,774±2,420.8 | 1.4* | $P = 0.37$ | $P = 0.50$ |
| FM, g | 12,690.6±6,572.1 | 12,827.3±6,301.7 | 1.1 | 10,244.3±3,330.7 | 10,334.4±2,829.5 | 0.9 | 16,680.2±3,900.2 | 16,200±3,840.0 | −2.8 | 17,240.3±4,656.0 | 16,809.8±4,086.6 | −2.5 | $P = 0.99$ | $P = 0.89$ |
| FM, % | 15.8±6.4 | 16.0±6.0 | 1.2 | 14.5±3.9 | 14.5±3.1 | 0.0 | 28.0±4.6 | 27.2±4.2 | −2.8 | 27.6±5.7 | 26.9±5.1 | −2.5 | $P = 0.97$ | $P = 0.75$ |
| Lumbar spine | | | | | | | | | | | | | | |
| BMC, g | 74.3±8.2 | 74.4±7.8 | 0.1 | 73.2±4.33 | 74.7±10.9 | 2.0* | 59.2±9.2 | 59.6±9.0 | 0.7 | 67.4±8.1 | 68.2±7.7 | 1.2 | $P = 0.21$ | $P = 0.49$ |
| BMD, g/cm$^2$ | 1.09±0.08 | 1.10±0.08 | 0.0±0.9 | 1.10±0.12 | 1.12±0.11 | 2.7* | 1.05±0.13 | 1.05±0.14 | 0.0 | 1.12±0.09 | 1.12±0.09 | 0.0 | $P = 0.25$ | $P = 0.13$ |
| Lower limbs | | | | | | | | | | | | | | |
| BMC, g | 639.8±86.2 | 644.4±85.8 | 0.7* | 600.6±66.1 | 604.6±65.6 | 0.6* | 436.0±59.2 | 436.2±61.3 | 0.0 | 488.6±56.6 | 491.2±57.7 | 0.5 | $P = 0.66$ | $P = 0.50$ |
| BMD, g/cm$^2$ | 1.48±0.10 | 1.48±0.11 | 0.0 | 1.46±0.07 | 1.46±0.06 | 0.0 | 1.21±0.09 | 1.22±0.09 | 0.8 | 1.26±0.09 | 1.27±0.08 | 0.8 | $P = 0.72$ | $P = 0.56$ |
| LM, g | 10,163.3±1,166.3 | 10,374±1,120.5 | 2.1* | 9,307±1,094.8 | 9,725±1,153.3 | 4.5* | 6,358±641.5 | 6,547.4±680.4 | 2.9* | 6,715±384.5 | 7,071±556.6 | 5.3* | $P = 0.03$ | $P = 0.94$ |
| Hip | | | | | | | | | | | | | | |
| Femoral neck BMC | 6.3±0.8 | 6.2±0.8 | −1.6 | 6.3±0.5 | 6.3±0.5 | 0.0 | 4.8±0.7 | 4.7±0.7 | −2.1 | 5.0±0.5 | 5.1±0.5 | 2.0* | $P = 0.05$ | $P = 0.31$ |
| Femoral neck BMD | 1.07±0.14 | 1.06±0.14 | −1.0 | 1.08±0.08 | 1.06±0.09 | −1.8 | 0.93±0.11 | 0.93±0.11 | 0.0 | 1.01±0.12 | 1.00±0.12 | −1.0 | $P = 0.07$ | $P = 0.19$ |
| Ward's triangle BMD | 0.96±0.16 | 0.95±0.16 | −1.0 | 0.91±0.09 | 0.91±0.09 | 0.0 | 0.86±0.12 | 0.85±0.14 | −1.1 | 1.00±0.18 | 0.97±0.16 | −3.0 | $P = 0.58$ | $P = 0.35$ |
| Trochanter BMD | 0.88±0.09 | 0.89±0.09 | 1.1 | 0.90±0.06 | 0.89±0.08 | −1.1 | 0.78±0.11 | 0.78±0.11 | 0.0 | 0.81±0.12 | 0.81±0.12 | 0.0 | $P = 0.13$ | $P = 0.54$ |
| Intertrochanter BMD | 1.35±0.15 | 1.36±0.14 | 0.7 | 1.35±0.11 | 1.36±0.11 | 0.7 | 1.15±0.15 | 1.15±0.15 | 0.0 | 1.23±0.13 | 1.23±0.13 | 0.0 | $P = 0.91$ | $P = 0.63$ |

Values are means ± SD. BMC, bone mineral content; BMD, bone mineral density; LM, lean mass; FM, fat mass. *$P < 0.05$, Pre vs. Post comparison. dual-energy X-ray absorptiometry tests were missed by one woman from the control group.

Table 6. *Vastus lateralis myosin heavy chain isoform composition before and after strength training combined with jumps*

| | Men | | | | | | Women | | | | | | Interaction Group × Time | Interaction Group × Time × Sex |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | Control group ($N$ = 15) | | | Training group ($N$ = 16) | | | Control group ($N$ = 11) | | | Training group ($N$ = 6) | | | | |
| MHC | Pre | Post | % Change | Pre | Post | % Change | Pre | Post | % Change | Pre | Post | % Change | | |
| Type I | 53.5±12.1 | 49.4±11.5 | −7.6 | 57.2±11.6 | 51.9±8.4 | −9.2* | 58.8±7.3 | 56.1±10.9 | −4.6 | 64.3±3.5 | 61.5±8.6 | −4.3 | $P$ = 0.83 | $P$ = 0.86 |
| Type IIa | 40.3±14.8 | 45.8±13.4 | 13.6 | 39.0±12.9 | 47.6±9.2 | 21.3* | 41.2±7.3 | 40.4±10.5 | −1.9 | 33.6±7.6 | 36.2±9.9 | 7.7 | $P$ = 0.33 | $P$ = 0.98 |
| Type IIx | 5.7±9.5 | 5.2±10.6 | −8.7 | 3.8±6.4 | 0.4±0.9 | −89* | 0.0±0.0 | 3.4±8.1 | | 2.0±5.0 | 2.2±5.4 | 10 | $P$ = 0.13 | $P$ = 0.91 |

Values are means ± SD; $N$, no. of subjects. *$P < 0.05$.

## DISCUSSION

In rodents, osteocalcin reduces fat mass and enhances insulin sensitivity (16, 30). In agreement, we found that osteocalcin is negatively associated with serum cortisol and leptin concentrations, fat mass, and the percentage of body fat in healthy physically active humans. However, despite the marked increase in osteocalcin, there was no significant reduction of fat mass with strength training. We also found that osteocalcin is positively associated with free testosterone and that men have greater circulating levels of osteocalcin than women. In contrast with our hypothesis, serum osteocalcin concentration was increased similarly in men and women after a strength training program that elicited similar relative gains in muscle mass and strength in both sexes. We observed that women who develop more muscle hypertrophy and improved their strength more experienced greater enhancement of their basal osteocalcin concentration with strength training. In agreement with our hypothesis, a higher percentage of MHC II isoforms predicted greater increase in basal serum osteocalcin concentration with strength training. This study also shows that there is a sex dimorphism in the serum leptin response to strength training.

*Performance enhancement and muscle hypertrophy with strength training is similar in men and women.* The effect of the training program on performance was rather similar in both sexes. In agreement with previous studies, men and women showed similar relative improvements in maximal dynamic force (1 RM) (31, 40, 44). However, men improved significantly more MVC than women, although the sex effect was small. In contrast, running speed in 30 m was improved only in women. These effects were in part explained by a similar degree (4–5%) of muscle hypertrophy, in agreement with previous studies (13, 24, 31, 32, 45, 55).

*Osteocalcin response to strength training, fat mass, and muscle phenotype.* Studies with rodents indicate that osteocalcin reduces fat mass (16, 30). In agreement, our cross-sectional data also show a negative correlation between osteocalcin and fat mass, the percentage of body fat and leptin concentration in serum. Similar results have been reported in elderly men (27). However, our longitudinal data show that, in exercising humans fed ad libitum, a marked (but physiological) increase in osteocalcin concentration is not associated with a reduction of fat mass, regardless of sex. In agreement, no significant correlations were observed between changes in osteocalcin and changes in visceral fat in 11 perimenopausal obese women submitted to hypocaloric diet combined with strength training (15). It remains to be determined whether a higher elevation (or pharmacological levels) of osteocalcin could reduce fat mass in humans.

In agreement with our hypothesis, we have observed that muscle phenotype influences the osteocalcin response to strength training, such that the muscle phenotype alone explains 25% of the osteocalcin response to exercise training. Subjects having a high percentage of MHC II experience greater elevation of basal osteocalcin. The association between muscle phenotype and osteocalcin could be explained by the greater ability of fast-type muscle fibers to generate higher and faster strain levels in the bones where they attach (41). Further strength training studies of longer duration will be needed to determine whether this increased osteoblastic activity could also translate into greater bone mass acquisition.

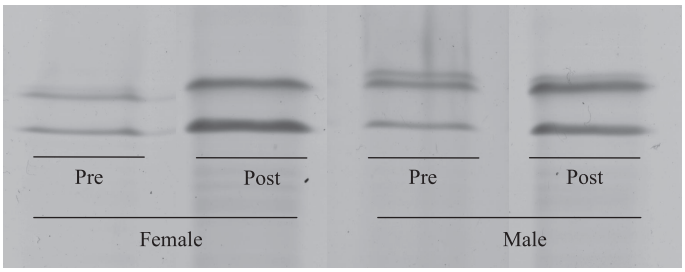


Fig. 5. Identification of the three bands corresponding to MHC isoforms I, IIa, and IIx in muscle biopsies using SDS-PAGE.

*The short-term osteogenic response to strength training is similar in young men and women.* Our study shows that even 9 wk of strength training combined with jumping exercise are long enough to elicit a small, but significant, increase in BMC, whose magnitude and regional distribution are rather similar in young adult men and women. In addition, the present investigation shows for the first time that the short-term osteogenic response to exercise is similar in young adult men and women, as reflected by similar increases in osteocalcin serum concentration and similar enhancements of whole body and lumbar spine BMC. These findings contrast with the marked sex dimorphism observed in adolescents (peri- to postpubertal), who participated in an 8-mo in-school intervention, which consisted of 10 min of jumping activity in place of regular physical education warm-up (56).

No single study had previously examined the osteogenic responses to the same strength training program in men and women, with comparable CGs. The few studies published until now lacked appropriate CGs (40). Ryan et al. (40) reported in 10 men and 7 women (age range: 20–29 yr) that, after 6 mo of training 3 times a week with moderate loads (12–15 RM), WBBMC was improved only in young men; however, there was no sex effect in the ANOVA test, implying that this finding provides only little, if any, evidence in favor of greater osteogenic responsiveness in men. The latter basically concurs with the present investigation, with the difference that the effects here reported were achieved in shorter time, by using a more intense strength training program supplemented with jumps. We have, however, observed a trend for greater improvements in BMD in men than women. It remains to be determined whether a longer intervention would lead to greater enhancement of BMD in men than women.

In contrast with our results, no significant effects on bone mass or density have been reported in young women submitted to short-term strength training programs (without jumping exercises) (12, 37). This discrepancy is likely attributable to the differences in the training program and highlights the importance of including jumping or other high-impact exercises in the training program.

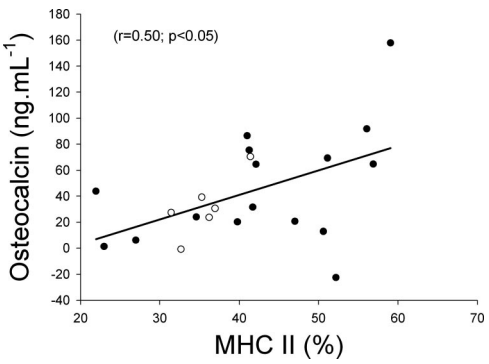


Fig. 6. Relationship between serum MHC II percentage in the vastus lateralis before the start of the training program and the change in osteocalcin concentration with strength training in 16 men (●) and 6 women (○).

*The enhancement of muscle mass with strength training is associated with the increase in bone mass and in basal serum osteocalcin concentration.* This study shows that the change in osteocalcin concentration is positively associated with the level of muscle hypertrophy elicited by the strength training program. This may just reflect the concurrent mechanical stimulation of bones and muscle fibers by the strength and jumping exercises. In agreement with our hypothesis, the gain in lower extremity lean mass explained 22% of the variance in the gain of WBBMC. This finding agrees with previous studies with transgenic animals, showing that phenotypes of increased muscle mass also show enhanced bone mass (22). In humans, a twin study that included 56 monozygotic and 56 dizygotic female pairs of twins of mean age of 45 yr (range 24–67 yr) concluded that genetic factors accounted for 60–80% of the variance in hip BMD and 60–80% of the variance in lean mass and more than 50% of their covariance (42). The latter was interpreted as an indication that the association between greater muscle mass and greater BMD is likely to be determined by genes regulating size (42). However, later studies on humans have shown an association between the increase in muscle mass and the gain in bone mass (51).

*Strength training reduces basal serum leptin concentration, even in the absence of changes in fat mass, but only in women.* In agreement with our results, Ryan et al. (40) and Walts et al. (55) reported no changes in fat mass (40), thigh subcutaneous fat (assessed by magnetic resonance imaging), or intermuscular fat in young and aged (50–85 yr) men and women who endured a strength training program lasting 6 mo (40) or 10 wk (55). In young men, we have also observed a small reduction in fat mass with only 6 wk of strength training but using a harder exercise program (4). Lemmer et al. (32) reported that there is a sex dimorphism in the effects of strength training on resting metabolic rate (RMR), which increases in trained young men but not in women, even after adjusting RMR for fat-free mass. No change in RMR has been reported with strength training in other studies (8, 48, 49). Moreover, despite the elevation of RMR reported by Lemmer et al. (32), strength training did not affect fat mass, either in men or in women in their study.

In agreement with our laboratory's previous study (4) and others (25), no changes in basal serum leptin concentration were observed in men. However, leptin concentration diminished by 14% in the women of the TG, and this effect remained significant even after accounting for differences in body fat.

This finding concurs with a 17% decrease in fasting serum leptin levels after 12 wk of aerobic training in women, without significant changes in men, despite the fact that fat mass was not altered after training in either group (23). Thus, for a given fat mass, young women have less circulating leptin after strength training. This finding is compatible with enhanced leptin sensitivity after strength training in women. A similar sex dimorphism has been reported in response to a 6-mo hypocaloric diet, eliciting similar losses in fat mass percentage (−13 and −16%, in men and women, respectively), but greater relative decline in circulating leptin in women than men (−45 and −21%, respectively), even after accounting for the changes in fat mass (36).

In agreement with animal studies showing that leptin has a negative influence on bone mass (14), in women there was a trend for a negative association between the changes in serum osteocalcin and leptin. However, in men, osteocalcin and leptin changed in the same direction. The latter could indicate that the interaction between osteocalcin and leptin may be modulated by sex hormones. In fact, a clear association between free testosterone changes and osteocalcin was observed in women. Serum testosterone, 17β-estradiol, and cortisol are hormones that may fluctuate in response to strength training (3, 21, 28, 44) and may influence bone metabolism (6) and leptin concentrations in plasma (7, 33–35, 43, 54, 57). However, in the present study, these hormones did not change significantly with strength training.

In summary, this study shows that osteocalcin serum concentration is positively associated with bone mass and density, lean body mass, muscle strength, and free testosterone, while osteocalcin is negatively associated with serum cortisol and leptin concentrations, fat mass, and the percentage of body fat. In addition, we have shown that basal serum osteocalcin concentration increases similarly in men and women submitted to strength training combined with plyometric exercises. This response depends in part on the muscle MHC composition at the start of the training program. Basal serum leptin concentration was only reduced in women, implying a sex dimorphism in the leptin response to strength training. This sex disparity in the leptin response to strength training was not explainable by differences in the fat mass, free testosterone, 17β-estradiol, or cortisol responses to strength training. Finally, our longitudinal data show that, in exercising healthy humans fed ad libitum, an increase in osteocalcin concentration is not associated with a reduction in fat mass. Further studies are needed to determine whether an increase in osteocalcin could elicit a reduction of fat mass in obese people.

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