

Bone Mass and Bone Turnover in Power Athletes, Endurance Athletes, and Controls: A 12-Month Longitudinal Study

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Strain magnitude may be more important than the number of loading cycles in controlling bone adaptation to loading. To test this hypothesis, we performed a 12 month longitudinal cohort study comparing bone mass and bone turnover in elite and subelite track and field athletes and less active controls. The cohort comprised 50 power athletes (sprinters, jumpers, hurdlers, multievent athletes; 23 women, 27 men), 61 endurance athletes (middle-distance runners, distance runners; 30 women, 31 men), and 55 nonathlete controls (28 women, 27 men) aged 17-26 years. Total bone mineral content (BMC), regional bone mineral density (BMD), and soft tissue composition were measured by dual-energy X-ray absorptiometry. Bone turnover was assessed by serum osteocalcin (human immunoradiometric assay) indicative of bone formation, and urinary pyridinium crosslinks (high-performance liquid chromatography) indicative of bone resorption. Questionnaires quantified menstrual, dietary and physical activity characteristics. Baseline results showed that power athletes had higher regional BMD at lower limb, lumbar spine, and upper limb sites compared with controls ($p < 0.05$). Endurance athletes had higher BMD than controls in lower limb sites only ($p < 0.05$). Maximal differences in BMD between athletes and controls were noted at sites loaded by exercise. Male and female power athletes had greater bone density at the lumbar spine than endurance athletes. Over the 12 months, both athletes and controls showed modest but significant increases in total body BMC and femur BMD ($p < 0.001$). Changes in bone density were independent of exercise status except at the lumbar spine. At this site, power athletes gained significantly more bone density than the other groups. Levels of bone formation were not elevated in athletes and levels of bone turnover were not predictive of subsequent changes in bone mass. Our results provide further support for the concept that bone response to mechanical loading depends

upon the bone site and the mode of exercise. (Bone 20: 477-484; 1997) © 1997 by Elsevier Science Inc. All rights reserved.

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Introduction

Maximizing skeletal mass has important implications, both for the individual and for the community. The risk of osteoporotic fracture in the elderly increases progressively as bone mineral density (BMD) declines²⁹ and osteoporotic fractures impose a considerable financial burden on the health care system. Despite advances in therapy, reversal of bone loss in established osteoporosis remains problematic. Exercise is one preventive measure that may optimize peak BMD.

Exercise can be an enjoyable, inexpensive, and readily available activity. In addition, it has numerous other health benefits, especially in relation to the cardiovascular system. However, studies evaluating the efficacy of exercise in promoting bone mass accrual have provided contradictory results. In part, the discrepancy may be due to the varying influences of gender, age, anatomical region, predominant bone type, and exercise regimen. It is suggested that the most effective form of exercise for the skeleton is dynamic, weight-bearing activity. Studies using controlled external loading in animal models have found that osteogenesis is maximal with high-magnitude strains applied at a high rate and that relatively few strain cycles are needed.^{39,42,44,45} If these principles are applied to humans, activities such as weight-training, sprinting, and jumping should have a greater effect on bone mass than distance running. This concept has been supported by some investigators^{17,26,43} but not others.⁵¹

Although bone mass has been extensively measured, there is little research into bone turnover associated with exercise. There is some evidence that elevated levels of bone formation may be found in athletes compared with less active individuals, although this has not been a consistent finding.^{14,28,32,36} Assessment of bone turnover using more specific and sensitive biochemical markers may increase our understanding of the mechanisms responsible for exercise-related changes in bone mass.

The sport of track and field encompasses a variety of events

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with different physical requirements. Athletes participating in power events (e.g., sprints, hurdles, jumps) train at maximal or near maximal intensity in short bursts. Conversely, athletes participating in endurance events (e.g., middle-distance and distance running) train at lower intensity levels for extended periods. Power athletes subject their skeleton to fewer total strain cycles but cycles of higher magnitude than do endurance athletes. We studied elite and subelite track and field athletes and less active controls over a 12 month period to compare baseline bone mass and bone turnover and to monitor changes in bone mass.

We hypothesized that: (i) athletes would have higher bone density than controls at loaded sites; (ii) power athletes would have greater bone density than endurance athletes; (iii) biochemical markers of bone formation would be higher in athletes, particularly power athletes, than controls; (iv) changes in bone mass would be greatest in athletes, particularly power athletes; and (v) markers of bone turnover would predict changes in bone mass.

Materials and Methods

Study Design

A 12 month longitudinal cohort design was used.

Subjects

The cohort consisted of 50 power athletes (sprinters, jumpers, hurdlers, multievent athletes; 23 women, 27 men), 61 endurance athletes (middle-distance runners, distance runners; 30 women, 31 men), and 55 nonathlete controls (28 women, 27 men). All subjects ranged in age from 17 to 26 years. Athletes competed at the club, State, or Australian national level. In the year preceding the study, 21 (39.6%) women and 27 (47.0%) men were ranked within the top 50 Australian athletes.¹ All athletes trained at least three times per week when uninjured. Controls were recruited via advertisements placed on local university, school, and hospital noticeboards. Control subjects participated in less than 3 h per week of regular, vigorous, weight-bearing exercise in the year preceding recruitment. Subjects who were known to have used anabolic steroids or human growth hormone, had a history of disease with the potential to influence bone density, or were currently taking medication likely to influence bone density (with the exception of the oral contraceptive pill) were excluded.

During the 12 month study, the attrition rate was 14% for women and 18% for men. 11 subjects left the project due to study or work commitments, 9 retired from athletics or did not train during the year, 2 relocated, and 2 were concerned about radiation from repeated DXA measurements. Two control subjects were later excluded; one because he participated in >7 h/wk of strenuous physical activity regularly throughout the year and the other because he gained more than 14 kg during the study and represented an influential outlier in all statistical analyses. This left 20 female and 21 male power athletes, 26 female and 28 male endurance athletes, and 24 female and 21 male controls.

The project was approved by the Human Experimentation Ethics Committees of La Trobe University and The Royal Melbourne Hospital. Subjects gave written informed consent.

Menstrual, Dietary, and Physical Activity Assessment

Questionnaires were used to quantify menstrual status, dietary intake, and level of physical activity. At entry to the study, female subjects were asked about menarcheal age, use of the combined oral contraceptive pill (OCP), number of menses in the preceding year, duration of amenorrhea (A) defined as ≤ 3

menses per year, duration of oligomenorrhea (O) defined as 4-8 menses per year, duration of eumenorrhea (E) defined as ≥ 9 menses per year, and whether they had ever been pregnant. The number of years of A, O, and E were used to calculate a menstrual index for each subject. The menstrual index, adapted from that devised by Grimston et al.,¹⁵ quantified the average number of menstrual cycles per year since menarche. Menstrual status and use of the OCP during the 12 month study were obtained prospectively via the completion of a diary.

Current daily dietary intake was calculated from two 4 day (3 week days, 1 weekend day) food records completed during the summer and winter months. Data from the food records were analyzed by a registered dietitian using the DIET/1 software package, version 3.22 (Xyris Software, Brisbane, Australia), which utilized the Nuttab Australia (1990) database. Mean nutrient values from the two records were used in the statistical analysis.

Historical physical activity levels were established by asking subjects to categorize their average weekly hours of vigorous physical activity over 5 year intervals from ages 5-19. Repeatability was assessed by readministering an identical questionnaire 18 months later to a random sample of 47 subjects. Weighted κ coefficients of physical activity recall³⁸ ranged from 0.39 to 0.82, depending on the age interval, indicating that the repeatability was fair to almost perfect.³⁰ Current exercise participation was quantified according to type and duration and was obtained from questionnaires administered at baseline and at the conclusion of the study.

Bone Densitometry

Dual-energy X-ray absorptiometry (DEXA) measurements of total body BMC, regional BMD and soft tissue composition, including total fat and lean mass, were acquired and analyzed on a Hologic QDR 1000W densitometer (Hologic Inc., Waltham, MA) using the standard whole body protocol, version 5.47. Regional BMD measurements included left and right upper limb, lumbar spine (L1-4) and left and right lower limb. The lower limb was further subdivided into three regions: femur (proximal femoral neck to the knee joint); tibia/fibula (knee joint to the ankle joint); foot (below the level of the ankle joint). Values for left and right sides were averaged. Measurements were taken at baseline and approximately 12 months later. Changes in foot bone density were not analyzed due to differences in foot positioning between scans. The mean time interval between the first and second DEXA scans was 12.4 (± 1.0) months with 136 (90%) subjects scanned within 1 month of the 12 month interval. Observed DEXA values were annualized where the retesting interval was ≤ 11 months or ≥ 13 months. DEXA short-term in vivo precision was evaluated from triplicate scans in 15 normal, healthy subjects. The coefficient of variation (CV) was 0.6% for total body BMC, 0.7% for upper limb and femur BMD, 1.3% for lumbar spine BMD, 1.4% for tibia/fibula BMD, and 1.9% for foot BMD. For soft tissue composition measurements, the CVs were 1.2% for total body fat mass and 0.4% for total body lean mass.

Biochemical Markers of Bone Turnover

Bone formation and bone resorption were assessed at baseline by measuring serum osteocalcin and urinary excretion of pyridinium crosslinks, respectively. Blood for serum osteocalcin was collected between 0800 and 1100 h, following an overnight fast. For female subjects, samples were collected during the early follicular phase of the menstrual cycle, defined as within 7 days of the onset of menstrual bleeding. Serum osteocalcin was measured by

Table 1. Baseline and 12 month change characteristics of female and male power athletes, endurance athletes, and controls given as the mean (\pm SD)

Characteristics	Females			Males		
	Power athletes	Endurance athletes	Controls	Power athletes	Endurance athletes	Controls
Age (years)	20.1 (1.9)	20.8 (2.3)	20.2 (2.1)	19.9 (1.7)	20.7 (2.1)	20.4 (2.5)
Height (cm)	170.1 (5.4) ^{b,d}	165.3 (5.9)	162.3 (5.8)	179.7 (6.3)	178.9 (6.1)	177.6 (7.4)
Δ height (cm)	0.3 (0.8)	0.3 (0.8) ^e	0.2 (0.6)	0.1 (0.7) ^c	0.4 (0.7) ^f	0.6 (0.6) ^f
Weight (kg)	60.3 (4.6) ^e	58.0 (6.1)	56.0 (7.0)	73.9 (7.5) ^a	67.2 (6.7)	71.3 (10.7)
Δ weight (kg)	0.6 (2.0)	0.3 (2.5)	-0.1 (3.3)	1.4 (1.7) ^f	0.6 (2.6)	0.6 (2.7)
Total lean mass (kg)	47.4 (3.5) ^d	45.1 (3.6) ^d	39.6 (4.0)	63.8 (6.6) ^{b,d}	58.3 (5.5)	58.0 (7.9)
Δ total lean mass (kg)	0.3 (1.3)	0.04 (1.4)	-0.01 (1.0)	1.0 (1.6) ^f	0.4 (1.8)	0.4 (1.8)
Total fat mass (kg)	10.0 (2.1) ^d	10.5 (3.0) ^d	13.6 (3.5)	7.0 (1.7) ^d	5.6 (1.7) ^d	10.1 (3.4)
Δ total fat mass (kg)	0.3 (1.1)	0.3 (1.2)	0.3 (1.2)	0.1 (1.0)	0.4 (1.1)	0.3 (1.2)
Calcium intake (mg)	977 (236) ^d	996 (338) ^d	680 (267)	1341 (624) ^d	1246 (354) ^d	920 (269)
Energy intake (kJ)	8524 (2523) ^e	8058 (2022)	6772 (1808)	12,901 (2959) ^d	13,361 (2410) ^d	11,011 (1868)
Age commenced competition	10.4 (4.8)	12.1 (5.0)	—	10.1 (4.1)	12.3 (4.5)	—
Baseline training hrs/wk	13.4 (5.7) ^d	10.9 (5.1) ^d	0.5 (0.9)	13.2 (4.4) ^d	12.0 (3.7) ^d	0.6 (0.8)

^aSignificantly different from endurance athletes, $p < 0.05$.

^bSignificantly different from endurance athletes, $p < 0.01$.

^cSignificantly different from controls, $p < 0.05$.

^dSignificantly different from controls, $p < 0.01$.

^eSignificant change from baseline, $p < 0.05$.

^fSignificant change from baseline, $p < 0.01$.

a human immunoradiometric assay (Immutopics, Inc. San Clemente, CA). The intra- and interassay CVs were 7% and 9%, respectively.

To measure pyridinium crosslinks [pyridinoline (Pyr) and deoxypyridinoline (D-Pyr)], a second void, early morning, 2 h urine collection was made after an overnight fast. Samples were collected between 0800 and 1100 h. The concentration of total pyridinium crosslinks in each urine sample was measured by high-performance liquid chromatography. Fluorescence was measured at 297 nm (ex) and 395 nm (em). Pyr and D-Pyr were quantitated using an external standard prepared from sheep cortical bone and calibrated against a highly purified pyridinoline standard (D. Eyre, University of Washington, Seattle, WA). The intra- and interassay CVs for this assay are both less than 9%. Recoveries of samples for this assay are, on average, 99.6% (range 88.5%–125.0%). Urinary Pyr and D-Pyr values were adjusted for urine creatinine (Cr) excretion. Urinary Cr concentration was measured by the Jaffe reaction using a Beckman Synchron CX7 Analyzer (Beckman Instruments Inc., Palo Alto, CA) prior to hydrolysis. The CV for Cr measurement was less than 8%.

Statistical Analysis

Comparisons between power athletes, endurance athletes, and controls for physical activity, soft tissue composition, menstrual characteristics, and dietary factors were made using one-way analysis of variance or chi-square tests. Post hoc Tukey's HSD tests were used to locate the source of any significant effects.²⁷ Comparisons between power athletes, endurance athletes, and controls for baseline and change in total body BMC and regional BMD were made separately in men and women using analysis of covariance with height and weight as covariates. Because the bone turnover marker data were not normally distributed, logarithmic transformations preceded all statistical analyses. Comparisons of markers of bone turnover between groups were made separately in men and women using analysis of variance or covariance. Total body BMC was included as the covariate for Pyr and D-Pyr to correct for any differences in skeletal size. Post hoc Tukey's HSD tests were used to locate the source of any significant effects. To assess whether markers of bone turnover

predicted 12 month change in total body BMC and lumbar spine BMD after controlling for age, gender, and subject group, forward stepwise multiple regression was used. The significance level was set at $p < 0.05$.

Results

Anthropometric and Dietary Characteristics

The means (\pm SD) for baseline age, height, weight, soft tissue composition, dietary intake, and physical activity together with 12 month changes are provided for both men and women in **Table 1**. Female athletes had more total lean mass, less fat mass, and a higher energy and calcium intake than controls. In addition, female power athletes were taller and weighed more than both endurance athletes and controls. The only significant change in anthropometric characteristics over 12 months in the females was a small height increase in the endurance athletes.

Male athletes had less fat mass and a higher energy and calcium intake than controls. In addition, power athletes had greater lean mass compared with controls. Power athletes were heavier and had more total lean mass than their endurance counterparts. During the 12 month follow-up, male power athletes showed a significant increase in weight and lean mass while endurance athletes and controls showed increases in height.

Training History and Physical Activity

Endurance athletes ran greater distances per week but participated in fewer plyometric activities and resistance training than power athletes. However, both athlete groups commenced competitive training at the same age and performed similar amounts of weekly training. Neither athlete group changed their training volume from the year preceding the study to the year of the study. All control subjects reported performing <2.5 h/wk of regular moderate-to-vigorous weight-bearing physical activity during the 12 month study period. Female controls participated in $0.7 (\pm 0.7)$ h/wk of regular physical activity compared with $11.3 (\pm 6.1)$ h/wk of athletics training for female athletes ($p < 0.001$). Male controls performed $0.6 (\pm 0.9)$ h/wk of regular physical

Table 2. Means (\pm SD) of *unadjusted* baseline and percentage change in total body BMC and regional BMD and baseline bone turnover in female and male groups

Bone variable	Females			Males		
	Power athletes	Endurance athletes	Controls	Power athletes	Endurance athletes	Controls
Total body BMC (g)						
Baseline	2267.7 (235.0)	2082.8 (237.9)	2030.7 (316.5)	2829.9 (371.0) ^d	2533.8 (324.9) ^c	2538.9 (452.6)
12 month % Δ	1.9 (2.2) ^c	1.5 (3.0) ^c	2.2 (2.9) ^c	1.7 (2.4) ^c	1.6 (2.5) ^c	1.8 (2.5) ^c
Upper limb BMD (g/cm ²)						
Baseline	0.814 (0.042) ^c	0.775 (0.040)	0.766 (0.047)	0.938 (0.067) ^d	0.881 (0.050)	0.884 (0.060)
12 month % Δ	0.6 (1.7) ^c	0.5 (1.3) ^c	1.9 (2.2) ^c	0.5 (1.9)	0.5 (1.7)	0.9 (2.3)
Lumbar spine BMD (g/cm ²)						
Baseline	1.167 (0.122) ^{b,d}	1.036 (0.130)	1.020 (0.120)	1.244 (0.135) ^{b,d}	1.095 (0.107)	1.051 (0.143)
12 month % Δ	2.6 (3.3) ^c	1.7 (3.5) ^c	0.4 (3.8) ^c	3.0 (3.8) ^{a,d,e}	0.9 (3.4) ^{c,e}	-0.4 (4.1)
Femur BMD (g/cm ²)						
Baseline	1.220 (0.103) ^d	1.190 (0.096)	1.123 (0.103)	1.389 (0.124) ^d	1.333 (0.110) ^d	1.243 (0.132)
12 month % Δ	2.0 (1.6) ^c	1.4 (1.6) ^c	1.4 (1.8) ^c	1.6 (1.6) ^c	0.7 (1.7) ^c	1.5 (2.9) ^c
Tibia/fibula BMD (g/cm ²)						
Baseline	1.143 (0.072) ^d	1.094 (0.073) ^d	1.014 (0.085)	1.282 (0.118) ^d	1.205 (0.079) ^d	1.109 (0.109)
12 month % Δ	-0.2 (3.7)	0.8 (3.3)	-0.01 (1.5)	-0.6 (4.7)	0.4 (2.6)	0.3 (3.1)
Foot BMD (g/cm ²)						
Baseline	1.065 (0.090) ^{a,d}	1.011 (0.083) ^d	0.888 (0.064)	1.246 (0.121) ^d	1.206 (0.089) ^d	1.038 (0.094)
Pyridinoline (nmol:mmol Cr)	90.73 (26.88) ^a	71.31 (21.20)	84.90 (21.10)	79.20 (29.94)	90.75 (40.54)	86.91 (32.25)
Deoxypyridinoline (nmol:mmol Cr)	15.14 (4.94) ^a	11.66 (5.66)	16.04 (10.14)	15.24 (8.96)	15.43 (8.19)	15.82 (7.53)
Osteocalcin (ng/mL)	7.61 (3.96)	6.34 (2.48)	7.42 (2.61)	10.72 (4.76)	9.59 (3.62)	11.81 (5.22)

Results of statistical tests using data adjusted for height and weight.

^aSignificantly different from endurance athletes, $p < 0.05$.

^bSignificantly different from endurance athletes, $p < 0.01$.

^cSignificantly different from controls, $p < 0.05$.

^dSignificantly different from controls, $p < 0.01$.

^eSignificant change from baseline, $p < 0.01$.

activity compared with 11.2 (\pm 4.9) h/wk of athletics training for male athletes ($p < 0.001$).

Self-reported physical activity levels throughout childhood and adolescence showed significant differences in the number of hours per week of vigorous physical activity between athlete and control groups. At all age ranges, there were more athletes than controls engaged in >7 h/wk of vigorous activity, and fewer athletes than controls engaged in 0–1 h/wk. There was no difference in historical physical activity levels between power and endurance athletes of either gender.

Menstrual Characteristics

Female athletes had a later age of menarche than controls [P: 14.3 (\pm 1.2); E: 14.3 (\pm 1.8); C: 13.2 (\pm 1.2), $p < 0.01$], with 50.9% of athletes commencing menstruation at age 14 or older compared with only 14.9% of controls. All females were more than 3 years postmenarcheal. Endurance athletes reported fewer menses in the year preceding the study than controls [E: 9.8 (\pm 3.9); C: 11.6 (\pm 1.4), $p < 0.05$]. Menstrual history also appeared to differ among endurance athletes and controls. 53% of endurance athletes had a history of oligo- and/or amenorrhea compared with only 21% of controls ($p < 0.05$). This finding was further supported by a lower menstrual index in the endurance athletes [7.8 (\pm 2.8)] than the controls [10.1 (\pm 1.0), $p < 0.05$], indicating on average fewer menses per year since menarche. Baseline and past use of the oral contraceptive pill did not differ between athletes or controls. All subjects were nulliparous.

During the 12 month study, all female subjects, except two athletes (one endurance, one power), were eumenorrheic (≥ 9 menses). 23 (50.0%) athletes and 13 (54.2%) controls used the OCP during the study. There was no significant difference in OCP use over the 12 months when comparing power and endurance athletes.

Bone Mass in Power Athletes, Endurance Athletes, and Controls—Baseline Results

In general, the baseline results revealed that athletes had greater bone density than controls at most loaded sites and that power athletes had greater bone density than endurance athletes at the lumbar spine (Table 2 and Figure 1). Compared with controls, female power athletes had higher baseline BMD at lower limb, lumbar spine, and upper limb sites, whereas female endurance athletes had higher baseline BMD only at the foot and tibia/fibula. Female power athletes had higher baseline BMD than their endurance counterparts at the lumbar spine and foot. Despite these regional differences in BMD between power athletes, endurance athletes, and controls, there was no difference in total body BMC between groups.

Compared with controls, male power athletes had higher baseline BMD at all regional sites, whereas male endurance athletes had higher BMD at lower limb sites only. Both power and endurance athletes had higher total body BMC than the controls after adjusting for height and weight. Comparing the two athlete groups, male power athletes had greater BMD than endurance athletes at the lumbar spine.

Bone Mass in Power Athletes, Endurance Athletes, and Controls—12 Month Results

The data (adjusted for changes in height and weight) showed small but significant increases in total body BMC in all athlete and control groups (Table 2). The annual change in total body BMC ranged from 1.5%–2.2% in the female groups and from 1.6%–1.8% in male groups. Similarly, all groups demonstrated significant increases in femur BMD of the order of 1%–2%.

At the lumbar spine, there was a difference in the rate of change in BMD between athlete and control groups. Power

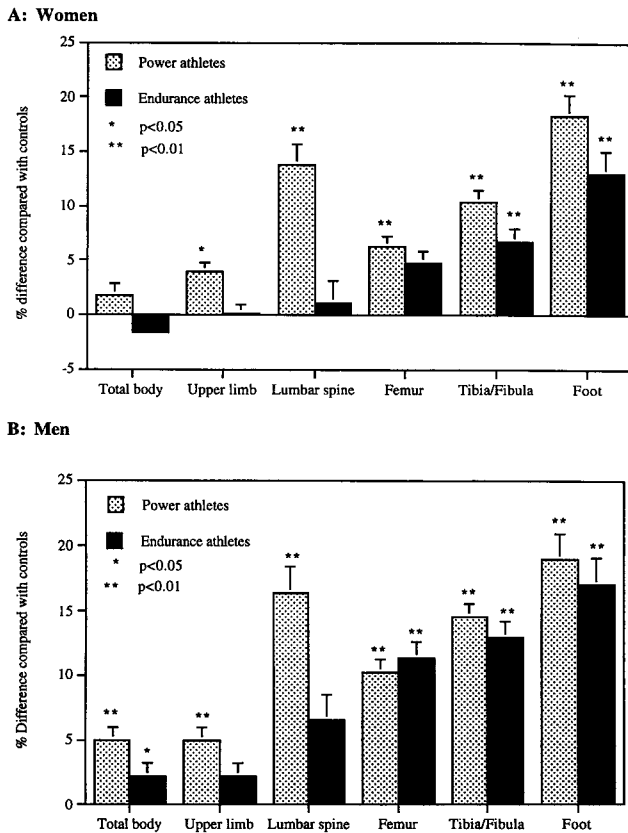


Figure 1. Percentage (SE) difference in baseline total body bone mineral content and regional bone density (adjusted for height and weight) of power and endurance athletes compared with controls.

athletes gained more BMD than controls in both men ($p < 0.001$) and women ($p = 0.06$). Power athletes also gained more BMD than endurance athletes in both men ($p < 0.05$) and women ($p = 0.06$).

Tibia/fibula BMD did not change in any group. At the upper limb, only female controls exhibited a significant increase in BMD.

Bone Turnover in Power Athletes, Endurance Athletes, and Controls

Bone resorption was significantly higher in female power athletes than in female endurance athletes—6.9% higher Pyr and 18.8% higher D-Pyr levels (adjusted for total body BMC, both $p < 0.05$; Table 2). Levels of Pyr and D-Pyr in female power and endurance athletes did not differ from controls. There was no significant difference in OC levels between female groups. For the men, there was no significant difference between groups for any of the bone turnover markers.

None of the biochemical markers of bone turnover predicted 12 month changes in bone mass. Instead, age was a significant predictor of the change in total body BMC ($\beta = -7.37$; SE = 2.32; $p < 0.01$) accounting for 7% of the variation. For change in lumbar spine BMD, subject group was a significant predictor ($\beta = -0.019$; SE = 0.005; $p < 0.001$) with the greatest increase being in power athletes, followed by endurance athletes, and finally controls. This variable accounted for 11% of the variation.

Discussion

Baseline Differences in Bone Mass Between Athletes and Controls

Both male and female athletes had greater bone mass than their controls at sites where athletes loaded bone. Power athletes had greater bone mass than controls at virtually every site, whereas endurance athletes had greater bone mass than controls at lower limb sites.

Ground reaction forces are attenuated as they propagate upward.^{31,52} Thus, during athletic training, the bones of the foot and leg may be expected to undergo the greatest mechanical deformation and receive a more potent osteogenic stimulus than would the proximal bones. Consistent with this model, we found that differences in lower limb bone mass between athletes and controls were least at proximal sites (e.g., femur) and progressively greater distally with maximal difference at the foot. This suggests that there may be a local effect of mechanical loading on net bone formation.

The upper limb bone density results provide further evidence for local skeletal adaptation to mechanical loading. Power athletes, who performed substantial amounts of upper limb strength training, had higher bone density than controls in the upper limb. Endurance athletes, on the other hand, who minimally stressed their upper limbs, did not differ from controls in upper limb bone density.

We found regional differences in bone density between female athlete and control groups despite them having similar amounts of total body BMC. These results indicate that exercise may influence the pattern in which bone mineral is distributed throughout the skeleton without necessarily increasing the total amount of bone mineral. This phenomenon has also been reported by Lohmann et al.³² in a randomized, 18 month weight-training study of young women. They found significant increases in bone density at the lumbar spine and proximal femur without changes in total body bone mass.

Our results suggest that participation in track and field training may influence bone mass. However, cross-sectional comparisons cannot establish a causal relationship between exercise and bone density. Self-selection may confound athlete-control studies because athletes may have a genetic predisposition to higher bone mass. This trait, in turn, may convey greater resistance to injury or be associated with other factors that promote athletic success, and therefore select those individuals most suited to sporting participation.

Baseline Differences in Bone Mass Between Power and Endurance Athletes

We hypothesized that power athletes would have greater bone mass than endurance athletes. This was found to be the case only at the lumbar spine in both men and women. Why do power athletes have greater bone mass than endurance athletes at the lumbar spine and not at the lower limb sites? This may be related to the concept of strain thresholds for osteogenesis. At lower limb sites, both endurance and power training may generate strains that reach the threshold for bone adaptation. Because ground reaction forces undergo attenuation progressively from the foot,⁵² there may be insufficient load applied to the lumbar spine in endurance athletes to reach the strain threshold, whereas this threshold may be reached by the higher loads generated by power event training. This supports the theory that strain magnitude may be more important than strain frequency.

In addition to variation in loading patterns, hormonal factors may contribute to the differences in lumbar spine bone mass

between power and endurance athletes. Although we did not measure hormone levels, it has been shown that reproductive hormones in both genders can be influenced by endurance exercise.^{2,6,50} In female athletes this can lead to bone loss at axial sites.^{10,20,46} Female endurance athletes in our study reported a strong trend toward a greater prevalence of clinical menstrual disturbances and this may have modified the mechanical effects of exercise at the hormonally sensitive lumbar spine site. In addition, subclinical menstrual disturbances such as shortened luteal phase length may have been present in the endurance athletes. These have been associated with spinal bone loss in physically active women.⁴¹ Although there have been reports of lower spinal bone density in male endurance athletes^{5,19,33} evidence for a link between low reproductive hormone levels and low bone density remains scanty in men. Other systemic hormones altered by exercise and capable of influencing bone and calcium metabolism may also explain the observed differences in bone mass between athlete groups.

Differences in mechanical loading patterns and hormonal status are not the only possible mechanisms for the differences in bone mass between power and endurance athletes. As mentioned above, self-selection is a well-known confounding factor in cross-sectional BMD studies. Recent research has highlighted the effect of starting age of exercise on adult bone density.²³ In our study, power athletes commenced training 2 years earlier than endurance athletes and, although there was no significant difference between groups, it may have contributed to the baseline bone density differences.

Changes in Bone Mass Over 12 Months

There was a small but significant increase in total BMC in both athletes and controls during the 12 month study. The greatest gains in bone mineral were seen in the younger individuals. Although this study was not designed to establish the timing of peak bone mass, our longitudinal results suggest that bone mineral accrual is still possible in early adulthood. This is consistent with a recent study which showed attainment of peak bone mass around age 20.¹⁶

At the lumbar spine, there were greater increases in bone density in athletes than in controls. Three other longitudinal studies have also monitored changes in lumbar spine BMD of athletes and less active controls and reported similar findings to our own.^{8,22,36} These studies showed greater gains in spinal bone density in athletes ranging from 1.3% in female gymnasts (6 months) to 2.9% in male rowers (7 months) and 5.9% in female dancers (12 months). Spinal bone density of controls remained essentially unchanged.

Our study allowed comparison of two vastly different types of track and field competitors—power athletes and endurance athletes. Power athletes had greater increases in lumbar spine BMD than endurance athletes in both men and women. This is consistent with the baseline findings and adds considerable support to the argument that mechanical loading is responsible for spinal bone density differences rather than purely self-selection. Because power athletes had the greater gains in lumbar spine BMD, this emphasizes the possible importance of strain magnitude rather than strain frequency as an osteogenic stimulus at this site.

An important finding in this investigation of bone density at multiple sites in elite and subelite athletes was that changes in bone mass at the lower limb were independent of exercise status in contrast to the lumbar spine. Similar findings were noted in the gymnastic³⁶ and ballet²² studies discussed previously, as changes in femoral neck and metatarsal bone density, respectively, were not influenced by exercise participation. Given a slower bone turnover rate, it is possible that cortical sites take longer than

trabecular sites to manifest exercise-related changes in bone density.

Another reason why lower limb bone density changes were independent of exercise status may be related to the athlete's training history. Because the athletes in our study maintained baseline levels of exercise, it is conceivable that lower limb bones had already adapted to this degree of mechanical loading. Although elevated mechanical strain would be expected to augment bone density, an equilibrium point could be reached where bone density remains relatively constant. To achieve additional gains in bone mass, the training intensity may need to be increased to provide the bone with the stimulus necessary to reach the new elevated threshold for bone adaptation. This is supported by the results of two recent exercise intervention studies in previously inactive premenopausal women.^{3,32} Initial significant gains in bone density following a period of exercise were not improved upon with continued training.

Bone Turnover Markers in Athletes and Controls

Although we hypothesized that the observed baseline differences in bone density between athletes and controls might be mediated by higher levels of bone formation in the athletes, there was no difference in bone formation markers. There are several possible explanations as to why this was the case. First, if the genesis of observed bone mass differences between athletes and controls occurred during the childhood years when athletes were also more physically active, there would be no difference in bone turnover levels between athletes and controls at the age we studied these individuals. Second, it must be remembered that bone turnover markers reflect the sum of all bone remodeling throughout the skeleton.⁴⁰ In our study, total body BMC changes were similar in athletes and controls, which would be consistent with levels of bone turnover markers being similar in athletes and controls. Furthermore, although our results differ from those in other studies of sports participants,^{4,14,19,32,36,37} the studies are difficult to compare because of varying assay techniques, athlete populations, and control group characteristics.

Female power athletes had higher bone resorption rates than endurance athletes, but these findings were not explained by factors such as current menstrual status, OCP use, or calcium intake. Perhaps the pattern of bone strain resulting from power event training could trigger local growth factors, such as IGF-I, which alter the remodeling process. For example, in normal postmenopausal women, there is a dose-dependent effect of IGF-I on increasing bone turnover.¹² The finding of accelerated resorption in power athletes might also reflect greater bone microdamage in these athletes and a more substantial reparative process.

While the presence of increased bone resorption but similar bone formation in the female athlete groups might imply a degree of uncoupling of bone remodeling, a combination of bone formation markers may be necessary to fully clinically characterize osteoblastic activity.^{7,11,13} Thus, a single measurement of one bone formation marker as used in our study may have been inadequate to detect higher formation rates in the power athletes.

Biochemical markers of bone turnover did not predict changes in total body BMC or lumbar spine BMD. While markers of bone turnover can predict bone density and rates of bone loss in postmenopausal populations,^{21,25,47,49} most other studies in young adults have not observed any relationship at a variety of sites, even in large cohorts.^{18,24,34,48} This may be due to the greater and more rapid loss of bone around the time of menopause compared with the relative stability of bone mass around skeletal maturity. In addition, given the biological vari-

ability of these markers,⁹ a single measurement may be an inadequate measure of bone turnover levels.

Summary

Baseline comparison of athletes and controls revealed an association between greater bone mass and participation in track and field training. Bone mass differences were greatest at the sites subjected to mechanical loading. Bone density at the lumbar spine was highest in athletes training for power events. The longitudinal component of the study showed modest but significant increases in total body BMC and femur BMD in both athletes and controls. Gains in BMD at the lumbar spine were greatest in power athletes. When considered together, these results provide evidence for an osteogenic effect of mechanical loading, particularly at the lumbar spine, and suggest that strain magnitude may be a more potent stimulus than strain frequency. Levels of bone formation were not elevated in athletes and levels of bone turnover were not predictive of subsequent changes in bone mass.

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