JOURNAL OF BONE AND MINERAL RESEARCH
Volume 13, Number 2, 1998
Blackwell Science, Inc.

Effect of Long-Term Unilateral Activity on Bone Mineral Density of Female Junior Tennis Players

HEIDI HAAPASALO, PEKKA KANNUS, HARRI SIEVÄNEN, MATTI PASANEN, KIRSTI UUSI-RASI, ARI HEINONEN, PEKKA OJA, and ILKKA VUORI

ABSTRACT

High peak bone mass in early adulthood is an important protective factor against osteoporotic fractures in later life, but little is known about the effects of exercise on growing bone. The purpose of this cross-sectional study was to determine at which state of maturity (Tanner stage), the areal bone mineral density (BMD) differences between the playing and nonplaying arms of junior tennis players become obvious, and to clarify in each developmental stage which training and background variables, if any, could explain the interindividual differences in bones' response to mechanical loading. Ninety-one 7- to 17-year-old female tennis players and 58 healthy female controls were measured. In each Tanner stage, differences in BMD in playing and nonplaying (dominant and nondominant) arms (proximal humerus, humeral shaft, and distal radius) and BMD of the lumbar spine and nondominant distal radius were compared between the controls and players. Within each Tanner stage of players, the associations between training and background variables and BMD differences were analyzed with Spearman rank correlation coefficients. In players, BMD differences between the playing and nonplaying arms were significant ($P < 0.05$– < 0.001) in all Tanner stages, with the mean difference ranging from 1.6 to 15.7%. In controls, these dominant-to-nondominant arm differences were clearly smaller (ranging from −0.2 to 4.6%), but significant at some measured sites. In comparison with the relative side-to-side arm differences between the players and controls (i.e., examination of the training effect), the mean difference was not obvious and significant until the adolescent growth spurt (i.e., the girls in Tanner stage III with a mean age of 12.6 years). In the lumbar spine, significant BMD differences between players and controls were not found until Tanner stage IV (mean age 13.5 years; 8.7%, $P < 0.05$) and V (mean age 15.5 years; 12.4%, $P < 0.05$). In a nonloaded site of the skeleton (nondominant distal radius), no significant BMD differences were found between the players and controls in any Tanner stage. In the correlation analysis, the Tanner I and II players (mean ages 9.4 and 10.8 years) showed no significant associations between any of the predictive variables and the side-to-side BMD differences, while in Tanner stages III, IV, and V, such associations could be found; the total amount of training hours during the playing career and the number of training sessions per week showed a significant and systematic correlation (r_s ranging from 0.43 to 0.80) with the side-to-side BMD differences in several measured bone sites. In conclusion, this study suggests that in a majority of female junior tennis players, the benefit of unilateral activity on bone density does not become clearly evident until the adolescent growth spurt or Tanner stage III. The total amount of training during the player's career and the current training frequency (sessions per week) seem to best explain the training effect on bone tissue, leaving, however, room for speculation on the still unknown factors that modulate the loading response of a growing bone. (J Bone Miner Res 1998;13:310–319)

Bone Research Group, UKK Institute for Health Promotion Research, Tampere, Finland.

INTRODUCTION

The peak bone mass in early adulthood and the subsequent rate of bone loss are the main determinants of bone mass in later life.[1] Genetic factors seem to account for 60–80% of the interindividual variation in the peak bone mass, the remaining 20–40% being determined by environmental factors such as nutrition and physical activity.[2–5] Several recent studies have given strong evidence that 95–99% of the peak bone mass is gained during the first two decades of life.[6–9]

The higher the peak bone mass, the more an individual may loose in adulthood without risk of osteoporosis and subsequent fractures. In other words, a high peak bone mass in early adulthood is an important protective factor against osteoporotic fractures in later life.[10–12] Therefore, emphasis should be placed on increasing the bone mass to the genetically determined maximum, especially during puberty, when nearly half of the adult bone mass is gained. Similarly, the factors that negatively influence peak bone mass attainment (physical inactivity, immobilization, inadequate nutrition, hormonal imbalance, and bone-resorbing drugs) should be eliminated during childhood and adolescence.[13]

The positive effect of physical activity on human bone mass is well documented in many cross-sectional studies comparing athletes with sedentary controls,[14–16] as well as in longitudinal follow-ups.[17–19] In studies of tennis players, where the playing arm is compared with the nondominant counterpart, the side-to-side differences have been up to 40% in favor of the playing arm, which quite indisputably proves the positive effect of physical loading on bone.[20–22]

Animal studies, in turn, have suggested that a growing body has a greater capacity to add new bone to the skeleton than its adult counterpart,[23–26] and that this prior skeletal-maturity added bone mass could be retained through adulthood. It has also been speculated that the higher bone mass in adult athletes compared with sedentary subjects may be due to the fact that they started regular training already in childhood. If true, this could explain the relatively small increases in the bone mineral mass in the exercise interventions of adults.[18,19,25] The human evidence of good response of a growing bone to exercise arises from our two recent studies,[27,28] where we demonstrated with adult female squash and tennis players that the percentage side-to-side difference in bone mineral content between the dominant and nondominant arms was about two times greater if females had started playing at or before menarche rather than after it.

Related to our above noted finding, it has been suggested that within the period of longitudinal growth, the effects of exercise may vary considerably depending on the developmental stage, or the Tanner stage, of the individual, but clear-cut data and evidence have not been presented.[26,29,30] Despite this, it is interesting to review the findings and conclusions of these studies. Slemenda and Johnston[29] observed that in young female figure skaters the areal bone mineral densities (BMD) in the lower parts of the body were higher than in controls, but the difference was not evident until the midteens, despite the fact that all the girls had started training very early in life. VandenBergh and colleagues,[30] in turn, studied the relationship between physical activity, calcium intake, and bone mineral content (BMC) in children aged 7–11 years. They found increased BMC only in those with very high levels of physical activity, and the association was most pronounced in the more mature children.

However, the above noted studies have left many questions unanswered concerning the effects of physical activity on the bone mineral accumulation during growth. Specifically, the type, intensity, frequency, and duration of most beneficial exercise, and especially the age-phase or developmental stage at which the effects of physical activity would be most crucial on bone, are still largely unknown.

The purpose of this study was, therefore, to find out at which stage of maturity (Tanner stage), the side-to-side BMD differences between the playing and nonplaying arms of tennis players become obvious, and to clarify (within each Tanner stage) which training or background variables, if any, could explain the interindividual differences in bone's response to mechanical loading. In the first study question, the use of athletes with a known history of unilateral loading, adequately matched nonplaying controls, and a study design with side-to-side comparison allowed us to control the confounding factors encountered in earlier cross-sectional studies (the intrinsic factors such as age, height, weight, and hormonal status, and the extrinsic factors such as nutrition and medications). In the second study question, however, our cross-sectional study design allowed us to analyze the predicting variables *within* each Tanner stage only, i.e., due to a number of obvious and inevitable differences in the training and background variables among the Tanner groups I–V, our study design was not adequate to explain the interindividual side-to-side BMD differences across the Tanner groups.

MATERIALS AND METHODS

Participants

Ninety-one 7- to 17-year-old female tennis players were recruited for the study through the Finnish Tennis Federation and the tennis clubs in southern Finland. All subjects were clinically healthy, were not receiving medication known to affect bone metabolism, and none of them had had severe upper extremity injuries. Only players who had trained on a regular basis longer than 1 year (at least 1–2 sessions/week) were included in the study.

The control group was comprised of 58 similarly healthy Finnish girls who were recruited from local schools. Although some of them did participate in casual sports, none of them had been involved in physical activity affecting the dominant or nondominant extremity only. The study protocol was approved by an independent ethical committee for clinical investigation. Written informed consent was obtained from the participants and their parents.

Table 1. Characteristics of the Subjects

	Tanner I		*Tanner II*		*Tanner III*		*Tanner IV*		*Tanner V*	
Characteristic	*Controls (n = 11)*	*Players (n = 16)*	*Controls (n = 10)*	*Players (n = 18)*	*Controls (n = 10)*	*Players (n = 19)*	*Controls (n = 17)*	*Players (n = 20)*	*Controls (n = 10)*	*Players (n = 18)*
Age (years)	9.4 (0.9)	9.4 (0.9)	11.0 (0.8)	10.8 (0.6)	12.0 (1.4)	12.6 (1.1)	13.2 (1.0)	13.5 (1.3)	15.4 (1.2)	15.5 (1.0)
Height (cm)	137.3 (6.8)	135.8 (4.9)	147.6 (6.9)	148.7 (5.5)	157.9 (7.0)	157.5 (5.5)	163.2 (4.7)	163.6 (5.5)	166.1 (5.8)	166.4 (6.1)
Weight (kg)	31.9 (6.2)	30.5 (3.2)	43.5 (6.1)	41.6 (6.4)	46.6 (9.7)	45.2 (3.7)	53.1 (6.8)	52.3 (6.1)	57.2 (5.9)	58.8 (7.0)
BMI (kg/cm^2)	16.9 (2.9)	16.6 (1.4)	20.0 (2.5)	18.8 (2.6)	18.6 (2.9)	18.2 (1.6)	19.9 (2.0)	19.5 (1.8)	20.7 (1.8)	21.2 (2.5)
Daily calcium (mg)	959 (256)	1011 (524)	1193 (334)	1117 (445)	1041 (468)	1045 (429)	1073 (411)	1148 (584)	835 (421)	1045 (352)
Age at menarche (years)	—	—	—	—	11.0*	12.3 (1.2)†	12.6 (1.0)	12.3 (1.6)	12.3 (1.1)	12.9 (1.2)
Menstrual status (number of subjects)										
normal cycle	—	—	—	—	—	—	12	12	10	15
any irregularity							5	8	0	3
Menstrual history (number of subjects)										
no disturbances	—	—	—	—	—	—	14	12	5	13
some disturbances‡							3	8	5	5
Training										
starting age of playing		6.3 (1.3)		7.2 (1.2)		7.6 (1.6)		8.2 (1.6)		8.5 (1.4)
[range]		[5–9]		[6–9]		[4–10]		[6–11]		[6–11]
number of training sessions/week [range]		1.7 (0.7) [1–3]		2.1 (1.2) [1–5]		2.7 (1.1) [1–6]		3.9 (2.0) [1–10]		3.8 (1.8) [1–6]
session length (minutes) [range]		60 (11) [45–90]		59 (7) [40–75]		72 (12) [60–90]		73 (15) [60–105]		78 (14) [60–90]
years of training [range]		2.2 (1.2) [1–5]		2.4 (1.0) [1–5]		2.5 (0.8) [1–4]		2.5 (1.1) [1–6]		3.9 (1.9) [1–8]
total training hours (h) [range]§		272 (223) [52–778]		360 (249) [104–910]		744 (424) [182–1794]		1010 (571) [52–1989]		1131 (413) [364–1799]

* One subject had reached menarche in this group.

† Three subjects had reached menarche in this group.

‡ Cycle length <23 days or >35 days, irregularity in the duration of bleeding.

§ Total training hours = total number of training hours during entire playing career.

Values are mean ± SD.

TABLE 2. DOMINANT VERSUS NONDOMINANT ARM STRENGTH COMPARISONS

Strength measurement (kg)	Tanner I		Tanner II		Tanner III		Tanner IV		Tanner V	
	Nondominant	Dominant	Nondominant	Dominant	Nondominant	Dominant	Nondominant	Dominant	Nondominant	Dominant
Controls	(n = 11)		(n = 10)		(n = 10)		(n = 17)		(n = 10)	
elbow extension	6.7 (1.9)	6.6 (1.8)	9.5 (2.2)	9.1 (2.1)	9.3 (1.9)	9.7 (2.1)	9.5 (2.0)	9.9 (2.4)	10.2 (1.6)	10.1 (1.5)
elbow flexion	7.2 (1.5)	7.7 (1.7)	10.1 (2.1)	10.5 (1.5)	11.7 (2.2)	11.6 (2.4)	12.8 (2.3)	13.1 (2.3)	15.3 (3.6)	15.9 (3.6)
grip strength	13.9 (2.9)	14.5 (3.3)	15.2 (5.9)	15.9 (5.9)	21.4 (7.3)	22.8 (6.6)*	22.4 (7.7)	23.8 (7.3)*	21.0 (5.5)	22.5 (3.8)
Players	(n = 16)		(n = 18)		(n = 19)		(n = 20)		(n = 18)	
elbow extension	6.6 (1.7)	6.5 (1.8)	8.3 (1.6)	8.5 (1.8)	9.0 (1.4)	9.6 (1.6)*	9.1 (1.6)	10.4 (2.4)*	9.5 (1.7)	10.3 (1.7)†
elbow flexion	7.6 (1.3)	7.6 (1.4)	9.1 (1.8)	9.8 (2.2)†	10.7 (1.6)	11.6 (1.8)†	11.9 (1.9)	12.9 (1.9)‡	13.2 (3.4)	14.8 (3.6)*
grip strength	10.2 (4.4)	11.7 (4.6)†	13.2 (6.4)	15.0 (7.0)‡	17.4 (9.1)	21.0 (9.7)‡	20.6 (6.2)	24.1 (7.2)‡	22.8 (6.9)	25.8 (7.0)*

Difference from nondominant arm: *$p < 0.05$, †$p < 0.01$, ‡$p < 0.001$.
Values are mean ± SD.

Interview

The participants received a mailed questionnaire, which they completed with parents at home. At the institute, the questionnaire was briefly reviewed by one of the investigators (H.H.) to confirm that all questions were answered and understood.

Information regarding past injuries, medication, known diseases, diet, possible vitamin or mineral supplementation, consumption of alcohol, use of cigarettes, onset of menses (age at the first period), menstrual status at the moment (cycle length, duration of bleeding), and menstrual history (any disturbances ever and the type and duration of these disturbances) was requested of every subject.

The training history of the players was collected in detail: the starting age of tennis playing, the starting age of regular (at least once a week) training, number of training sessions per week, average duration of the training sessions, training intensity, and physical activities other than tennis. Using the information from the players' exercise diaries and the interview, the total amount of training hours during the entire playing career was finally calculated for each player.

The daily dietary calcium intake was assessed using a prospective 7-day calcium intake diary[31] and analyzed with the Micro-nutrica software (Social Insurance Institution, Helsinki, Finland).

Height, weight, and pubertal status

The height and weight of each subject were measured in normal indoor clothing without shoes. The body mass index (BMI) was calculated according the formula BMI = weight (kg)/(height [m])^2. The pubertal status (Tanner stage) of each subject was determined by visual inspection of each subject with the help of self-assessment pictures illustrating the five Tanner stages of breast development and pubic hair distribution.[32]

Strength measurements

The maximal isometric strength of the forearm extensors and flexors and the grip strength were measured with strain cauge dynamometers (Digitest Inc., Muurame, Finland). One practice trial was performed before testing to become familiar with the equipment. Three maximal efforts were performed with both hands, and the median value of the three readings was used as the test score.

Bone mineral measurements

Areal BMD (g/cm^2) was measured from three different sites of both upper extremities (proximal humerus, humeral shaft, and distal radius) and lumbar spine using a Norland XR-26 dual-energy X-ray absorptiometer (DXA) scanner (Norland Inc., Fort Atkinson, WI, U.S.A.). All measurements were performed by the same experienced laboratory technician. In our laboratory, the in vivo precision of BMD measurements is between 0.8 and 1.0%, depending on the site of the measurement.[33] The measurements have been described in detail elsewhere.[33,34]

Due to technical difficulty of the DXA software used to

TABLE 3. DOMINANT VERSUS NONDOMINANT ARM BMD COMPARISONS, AND THE LUMBAR SPINE BMD

	Tanner I		*Tanner II*		*Tanner III*		*Tanner IV*		*Tanner V*	
BMD (g/cm²)	*Nondominant*	*Dominant*	*Nondominant*	*Dominant*	*Nondominant*	*Dominant*	*Nondominant*	*Dominant*	*Nondominant*	*Dominant*
Controls	$n = 11$		$n = 10$		$n = 10$		$n = 17$		$n = 10$	
proximal humerus	0.497 (0.073)	0.511 (0.072)	0.538 (0.055)	0.563 (0.059)†	0.635 (0.061)	0.635 (0.700)	0.662 (0.059)	0.674 (0.062)	0.713 (0.093)	0.729 (0.094)
humeral shaft	0.641 (0.076)	0.657 (0.083)*	0.714 (0.040)	0.722 (0.041)	0.790 (0.104)	0.803 (0.104)	0.848 (0.051)	0.872 (0.058)‡	0.885 (0.066)	0.910 (0.071)†
distal radius	—	—	0.297 (0.052)	0.296 (0.047)	0.328 (0.031)	0.335 (0.040)	0.317 (0.036)	0.320 (0.029)	0.343 (0.047)	0.348 (0.045)
Players	$n = 16$		$n = 18$		$n = 19$		$n = 20$		$n = 18$	
proximal humerus	0.503 (0.025)	0.531 (0.026)‡	0.565 (0.043)	0.592 (0.043)†	0.612 (0.069)	0.665 (0.083)‡	0.669 (0.093)	0.756 (0.110)‡	0.750 (0.111)	0.841 (0.110)‡
humeral shaft	0.640 (0.044)	0.658 (0.041)*	0.703 (0.061)	0.736 (0.056)‡	0.788 (0.059)	0.868 (0.070)‡	0.842 (0.079)	0.972 (0.098)‡	0.913 (0.073)	1.051 (0.082)‡
distal radius	—	—	0.310 (0.023)	0.315 (0.025)	0.304 (0.036)	0.324 (0.036)‡	0.326 (0.038)	0.368 (0.044)‡	0.339 (0.049)	0.377 (0.052)‡
Lumbar spine										
controls	0.558 (0.078)		0.683 (0.091)		0.806 (0.096)		0.893 (0.090)		0.963 (0.134)	
players	0.606 (0.047)		0.683 (0.054)		0.829 (0.059)		0.971 (0.126)§		1.082 (0.105)§	

Difference from the nondominant arm: $^{*}p < 0.05$, $^{\dagger}p < 0.01$, $^{\ddagger}p < 0.001$.
Lumbar spine BMD, difference from controls: $^{\S}p < 0.05$.
Values are mean ± SD.

reliably separate the immature bone ends of the radius from adjacent soft tissue in some of the youngest subjects, the BMD data of the distal radius could not be analyzed from the Tanner I individuals and seven of the Tanner II individuals.

Statistical analysis

In each Tanner stage, the intraindividual side-to-side arm differences were compared using the matched paired *t*-test. Student's nonpaired *t*-test was used to compare the relative side-to-side arm differences and the absolute BMD values of the lumbar spine and nondominant distal radius between the players and controls. Player versus control differences (players' relative difference minus controls' relative difference) among the five Tanner groups were analyzed by analysis of variance (ANOVA); the difference in each Tanner group was compared with the average difference of the subsequent Tanner groups with Helmert contrasts (i.e., player vs. control difference in the Tanner I group was compared with the average difference in the Tanner II–V groups; the difference in the Tanner II group was compared with the average difference in the Tanner III–V groups; the difference in the Tanner III group was compared with the average difference in the Tanner IV–V groups; and finally, the difference in the Tanner IV group was compared with that in the Tanner V group).

Within each Tanner stage of the players, the associations between the relative side-to-side BMD difference and its potentially predictive variables (anthropometric, muscle strength, and training history variables) was determined with the Spearman rank correlation coefficients. The rank correlation coefficients were used because of the relatively small sample sizes in the Tanner stages (n = 16–20) and the skewed data distribution in some of the used variables. A forward-stepping multiple regression analysis was also performed between relative side-to-side BMD differences and the predictive variables. In all tests, an α-level less than 5% ($p < 0.05$) was considered significant.

RESULTS

Background characteristics

The subject characteristics are listed in Table 1. There were no remarkable differences in the anthropometric, dietary, or menstrual variables between the controls and players in any of the five Tanner stages (Table 1). As expected, the number of training sessions per week, training years, and total amount of training increased from Tanner I to V (Table 1). None of the subjects smoked or used alcohol regularly.

Strength measurements

In controls, significant side-by-side differences were found only in the grip strength in the Tanner III (8.4%) and IV (8.1%) players (Table 2). In players, significant side-to-side differences were found in every measured strength variable from Tanner stage III to V (the differences ranged

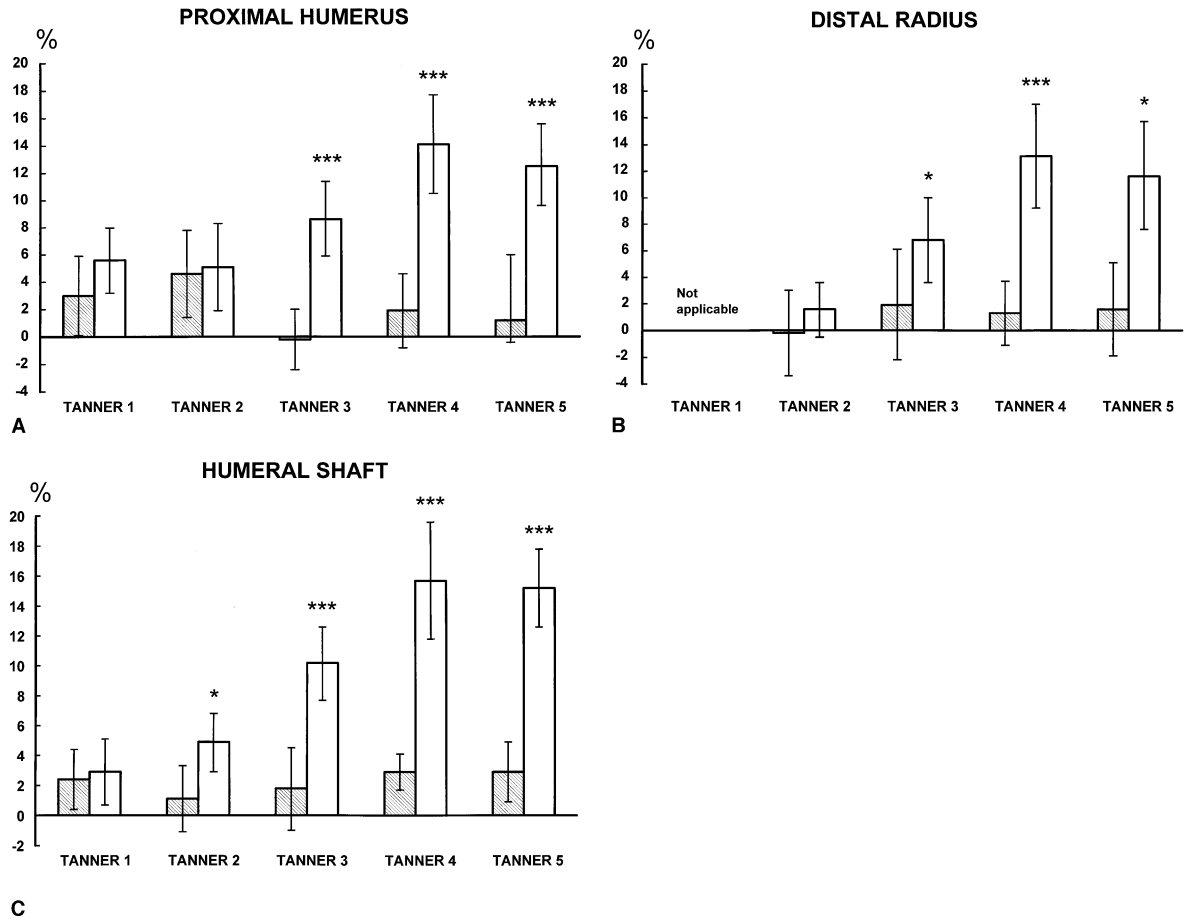

FIG. 1. The side-to-side BMD difference (%) in the upper extremities of controls (shaded bars) and players (open bars). The bars indicate the 95% confidence intervals. (Difference between controls and players $^{*}p < 0.05$, $^{***}p < 0.001$.) (A) Proximal humerus. In the ANOVA with Helmert contrasts, the player versus control difference in the Tanner I ($p = 0.041$) and Tanner II ($p < 0.001$) differed significantly from the average difference of the subsequent Tanner stages, whereas this was not the case in Tanner III ($p = 0.49$) and Tanner IV ($p = 0.72$). (B) Humeral shaft. In the ANOVA with Helmert contrasts, the player versus control difference in the Tanner I ($p < 0.001$) and Tanner II ($p = 0.001$) differed significantly from the average difference of the subsequent Tanner stages, whereas this was not the case in Tanner III ($p = 0.066$) and Tanner IV ($p = 0.84$). (C) Distal radius. The ANOVA with Helmert contrasts revealed that in none of the Tanner stages did the player versus control difference differ significantly from the average difference of the subsequent Tanner stages. The *p*-values were 0.071 for Tanner II, 0.067 for Tanner III, and 0.60 for Tanner IV.

from 7.1 to 26.6%) (Table 2). In Tanner I players, the side-to-side difference was significant in grip strength (17.3%), and in Tanner II players in grip strength (15.0%) and elbow flexion (7.7%) (Table 2).

When comparing the relative side-to-side strength differences of players with the corresponding values of the controls, significant differences (in favor of the players) were found in the grip strength of the Tanner I (controls 4.2% vs. players 17.3%, $p < 0.05$) and Tanner III (controls 8.4% vs. players 26.6%, $p < 0.05$) subjects. In the majority of the other strength comparisons, the side-to-side difference was higher in players than in controls, but the difference was not significant.

Bone measurements

In controls, the mean side-to-side BMD differences ranged from −0.2% to +4.6% and were significant at the humeral shaft (2.4%) in Tanner I; at the proximal humerus (4.6%) in Tanner II; and at the humeral shaft in Tanner IV (2.9%) and Tanner V (2.9%) (Table 3). In players, the side-to-side differences were clear and significant at all

measured sites and Tanner stages (the mean difference ranged from 1.6 to 15.7%) except at the distal radius in Tanner stage II (Table 3).

Compared with the controls, the Tanner stage III, IV, and V players had significantly higher relative side-to-side BMD differences at every measured site (Fig. 1). In Tanner II, there was a significant difference between controls and players in the humeral shaft only, ($p < 0.05$) and in Tanner I, there were no significant differences between players and controls (Fig. 1).

In the lumbar spine (a weightbearing bone site), significant BMD differences between the controls and players were found in Tanner stage IV (8.7%, $p < 0.05$) and V (12.4%, $p < 0.05$) (Table 3 and Fig. 2). In the nondominant distal radius (a nonloaded control site), no significant differences between controls and players were found in any Tanner stage (Table 3 and Fig. 2).

Prediction analysis

The total training hours and the number of current training sessions per week were the only predictive variables that showed a significant and systematic correlation with the relative side-to-side BMD differences in several measured sites; the duration of each training session and the number of training years did not show any significant correlation with the relative side-to-side BMD differences. The Spearman rank correlation coefficients (r_s) for these two variables are shown in Table 4. In the multiple stepwise regression analysis, addition of any of the other predictive variables into the model did not improve the prediction obtained by the strongest single predictor. This concerned every anatomic site and Tanner stage.

In the Tanner I players, there was no significant association between the total training hours or sessions per week and the side-to-side BMD difference in the proximal humerus and humeral shaft (Table 4) thus giving clear evidence that in these prepubertal girls not even strenuous tennis playing had resulted in BMD benefit. Neither in the Tanner II stage were the correlation coefficients for total training hours and sessions per week significant (r_s values were between 0.32 and 0.44 at the humeral sites). Instead, in Tanner III, both of the above-noted training parameters correlated significantly with the relative BMD difference at all measured bone sites, with the correlation coefficients ranging from 0.50 to 0.77. In Tanner IV, only one significant correlation was found (humeral shaft vs. total training, $r_s = 0.70$, $p < 0.01$), while in Tanner V the correlation coefficients ranged from 0.43 to 0.80 and were significant at all sites (except the BMD difference in the proximal humerus vs. total training, $r_s = 0.43$) (Table 4).

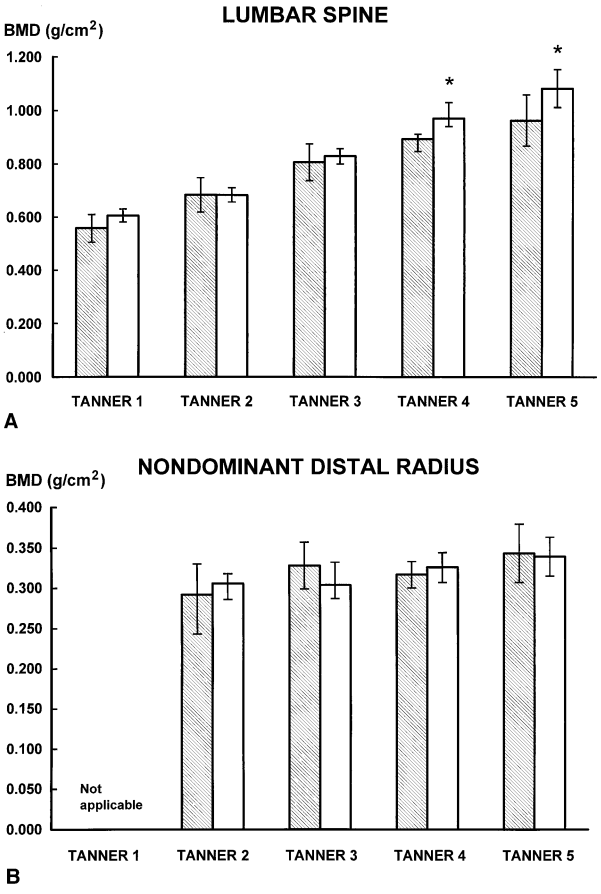

FIG. 2. The BMD (g/cm^2) of the lumbar spine and nondominant distal radius in the controls (shaded bars) and players (open bars). The bars indicate the 95% confidence intervals. (Difference between players and controls: $^*p < 0.05$, $^{***}p < 0.001$.) (A) Lumbar spine. The ANOVA with Helmert contrasts revealed that in none of the Tanner stages did the player versus control difference differ significantly from the average difference of the subsequent Tanner stages. The *p*-values were 0.86 for Tanner I, 0.079 for Tanner II, 0.079 for Tanner III, and 0.39 for Tanner IV. (B) Nondominant distal radius. The ANOVA with Helmert contrasts revealed that in none of the Tanner stages did the player versus control difference differ significantly from the average difference of the subsequent Tanner stages. The *p* values were 0.32 for Tanner II, 0.19 for Tanner III, and 0.52 for Tanner IV.

DISCUSSION

No previous study has clarified at which developmental stage the effect of physical activity on bone becomes obvious. In this study, highly significant BMD differences between the playing and nonplaying arms were found in players from all Tanner stages (Table 3), but the players' relative BMD differences did not start to differ clearly and significantly from the corresponding values of the controls until Tanner stage III (Fig. 1). This suggests that to some extent loading may increase bone density already in Tanner stage II, but the activity-induced bone gain becomes especially accentuated in stage III (i.e., during the rapid growth and natural bone accumulation period just before menarche). This finding can be explained by the fact that the adolescent growth spurt is the only time in life when bone is added in substantial amounts to the inner and outer sides of the bone cortex by endosteal and periosteal apposi-

Table 4. Spearman Rank Correlation Coefficients Between the Relative Side-to-Side BMD Differences and the Training Variables of the Players

Relative side-to-side BMD difference	*Training variable*	*Tanner I (n = 16)*	*Tanner II (n = 18)*	*Tanner III (n = 19)*	*Tanner IV (n = 20)*	*Tanner V (n = 18)*
Proximal humerus	total training hours‡	0.23	0.37	0.50*	0.03	0.43
	sessions per week	−0.11	0.44	0.55*	0.14	0.80†
Humeral shaft	total training hours	0.31	0.32	0.63*	0.70†	0.52*
	sessions per week	0.03	0.44	0.77†	0.47	0.61*
Distal Radius	total training hours	—	($n = 11$) −0.22	0.61*	0.11	0.61*
	sessions per week		−0.23	0.66†	0.13	0.67†

*$p < 0.05$, †$p < 0.01$.
‡ The total number of training hours during the entire playing career.

tion,[26] and quickly thereafter, the endosteal apposition fades away. It seems logical that when the rising levels of estrogens during puberty inhibit the bone resorption and enhance bone formation, addition of a stimulus of mechanical loading will further enhance bone formation in a synergistic fashion with the estrogens. It has to be kept in mind, however, that too vigorous exercise may lead to estrogen deprivation and menstrual dysfunction, thus disturbing the normal bone acquisition.[35]

In the lumbar spine, a site which was included in the study as an appropriate representative of a weight-bearing bone, the BMD differences between the players and controls did not become significant until Tanner stage IV (Table 3 and Fig. 2). In the nondominant distal radius (a nonloaded skeletal site), no difference between controls and players was found at any of the Tanner stages (Table 3 and Fig. 2). These findings clearly indicate the site specificity of the effects of mechanical loading on growing bone.

The mean lumbar spine BMD differences between the players and controls were 9% (Tanner IV) and 12% (Tanner V). These percentages are very close to those found in the cross-sectional studies of elite junior weight lifters (13%),[36] female figure skaters (6–14%),[29] and gymnasts (8%).[37] The magnitude of the BMD differences between adult athletes and their sedentary controls have also been reported to be about 9–15%.[16,17,35,38] Thus, the majority of the additional bone mineral gained with exercise seems to be acquired during adolescence.

As already noted, our study design allowed us to analyze the bone-gain predicting factors *within* each Tanner stage only (not across them), and it can be seen from Table 1 that our players in Tanner stages III, IV, and V had trained more than the players in Tanner stages I and II. Basically this could be the reason for the side-to-side differences not appearing until Tanner stage III. However, there was a rather large range in the training variables within every Tanner stage (including stages I and II; see Table 1), but despite this the correlation analysis did not reveal any significant associations between the training history variables and the outcome variable (BMD side-to-side difference) in Tanner stages I and II. The correlation analysis showed especially clearly that in stage I not even very strenuous training had resulted in BMD gain (Table 4). Additionally, it is also noteworthy that these Tanner I and II girls had trained on average two times a week for over 2 years before the measurements, and with similar background there were many Tanner III, IV, and V girls with a clear bone response. Thus, we feel that our findings support the concept that before puberty bone's responsiveness to loading is, in general, rather poor and that beyond the mechanical loading itself there must be many, still largely unknown (genetic, hormonal) factors that modulate the response of a growing bone to loading.

In the correlation analysis, the highest associations between training variables and the relative side-to-side BMD differences were found at Tanner stages III and V, while in Tanner stages I, II, and IV these associations were weaker (Table 4). In Tanner stages I and II, this may partly be due to the smaller range in the training and bone variables (Table 1). In Tanner stage III, a period of rapid natural skeletal growth and high bone turnover, the threshold for physical activity to add new bone might be relatively low (the effects of activity became obvious already with a relatively moderate training),[39] but, in this stage, intense training could lead to remarkable side-to-side differences also (Fig. 1). In Tanner IV, the only significant correlation was found between the total training hours and the side-to-side BMD difference in the humeral shaft (which mainly consists of cortical bone), while the other sites (which mainly consist of trabecular bone) showed no association. In this stage, all the mean side-to-side differences were, however, even higher than in Tanner V (Fig. 1), and the largest side-to-side differences in proximal humerus were seen in those Tanner IV girls who had trained with average frequency and intensity only. It may be that during this period the loading-induced bone mineral accumulation in the proximal humerus and distal radius became saturated already at moderate levels of activity, and thus more intense training gave no clear additional benefit (except in the humeral shaft). Near the skeletal maturity in Tanner stage V, a stage where the natural bone growth and turnover is slowing down, the threshold for physical activity to add new bone might again increase, and the only way to gain bone is

to train with higher frequency and intensity, and maybe that is why significant correlations between the training and bone parameters were found in Tanner V (Table 4).

Overall, combining the findings of this and our previous study,[28] it seems possible that the effect of unilateral activity on bone is greatest during a relatively short period in puberty, a period when rapid natural bone mineral accumulation and rapid longitudinal growth occur. Before or after this time period, the loading effect seems to be less clear, and most probably the loading has then to be more frequent and intense to become obvious. For these reasons, we recommend regular exercise during the pubescent years for maximizing the peak bone mass and thus preventing osteoporosis and related fractures in later life, but longitudinal studies are needed to confirm our cross-sectional observations.

ACKNOWLEDGMENTS

The authors thank the Finnish Tennis Federation and Finnish Tennis Clubs for their cooperation, Virpi Koskue for expert bone measurements, and Taru Malminen for help in recruiting the study subjects. This work was supported in part by grants from the Ministry of Education, Helsinki, Finland, and the Medical Research Fund of Tampere University Hospital.

REFERENCES

1. Johnston CC Jr, Slemenda CW 1994 Peak bone mass, bone loss and risk of fracture. Osteoporos Int **1:**S43–S45.
2. Krall EA, Dawson-Hughes B 1993 Heritable and lifestyle determinants of bone mineral density. J Bone Miner Res **8:**1–9.
3. Välimäki MJ, Kärkkäinen M, Lamberg-Allardt C, Laitinen K, Alhava E, Heikkinen J, Impivaara O, Mäkelä P, Palmgren J, Seppänen R, Vuori I 1994 Exercise, smoking, and calcium intake during adolescence and early adulthood as determinants of peak bone mass. Br Med J **309:**230–235.
4. Eisman J 1995 Vitamin D receptor gene alleles and osteoporosis: An affirmative view. J Bone Miner Res **10:**1289–1293.
5. Henderson NK, Price RI, Cole JH, Gutteridge DH, Bhagat CI 1995 Bone density in young women is associated with body weight and muscle strength but not dietary intakes. J Bone Miner Res **10:**384–393.
6. Bonjour J-P, Theinz G, Buchs B, Slosman D, Rizzoli R 1991 Critical years and stages of puberty for spinal and femoral bone mass accumulation during adolescence. J Clin Endocrinol Metab **73:**555–563.
7. Theinz G, Buchs B, Rizzoli R, Slosman D, Clavien H, Sizonenko PC, Bonjour J-P 1992 Longitudinal monitoring of bone mass accumulation in healthy adolescents: Evidence for a marked reduction after 16 years of age at the levels of lumbar spine and femoral neck in female subjects. J Clin Endocrinol Metab **75:**1060–1065.
8. Matkovic V, Jelic T, Wardlaw GT, Ilich JZ, Goel PK, Wright JK, Andon MB, Smith KT, Heaney RP 1994 Timing of peak bone mass in Caucasian females and its implication for the prevention of osteoporosis. J Clin Invest **93:**799–808.
9. Haapasalo H, Kannus P, Sievänen H, Pasanen M, Uusi-Rasi K, Heinonen A, Oja P, Vuori I 1996 Development of mass, density, and estimated mechanical characteristics of bones in Caucasian females. J Bone Miner Res **11:**1751–1760.
10. Seeman E, Tsalamandris C, Formica C, Hopper JL, McKay J 1994 Reduced femoral neck bone density in the daughters of women with hip fractures: The role of low peak bone density in the pathogenesis of osteoporosis. J Bone Miner Res **9:**739–743.
11. Ribot C, Tremollieres F, Pouilles JM 1995 Late consequences of a low peak bone mass. Acta Paediatr Suppl **411:**31–35.
12. Riis BJ, Hansen MA, Jensen AM, Overgaard K, Christiansen C 1996 Low peak bone mass and fast rate of bone loss at menopause. Equal risk factors for future fracture: A 15-year follow up study. Bone **19:**9–12.
13. Adami S 1994 Optimizing peak bone mass: What are the therapeutic possibilities? Osteoporos Int **1:**S27–S30.
14. Nilsson BE, Westlin NE 1971 Bone density in athletes. Clin Orthop **77:**179–182.
15. Jacobson PC, Beaver W, Grubbs SA, Taft TN, Talmage RV 1984 Bone density in women: College athletes and older athletic women. J Orthop Res **2:**328–332.
16. Heinonen A, Oja P, Kannus P, Sievänen H, Haapasalo H, Mänttäri A, Vuori I 1995 Bone mineral density in female athletes representing sports with different loading characteristics of the skeleton. Bone **17:**197–203.
17. Margulies J, Simkin A, Leichter I, Bivas A, Stinberg R, Giladi M 1986 Effects of intense physical activity on the bone mineral content in the lower limbs of young adults. J Bone Joint Surg **68A:**1090–1093.
18. Dalsky GP, Stocke KS, Eshani AA, Slatopolsky E, Lee WC, Birge SJ 1988 Weight-bearing exercise training and lumbar bone mineral content in postmenopausal women. Ann Intern Med **108:**824–828.
19. Heinonen A, Kannus P, Sievänen H, Oja P, Pasanen M, Rinne M, Uusi-Rasi K, Vuori I 1996 Randomised controlled trial of effect of high-impact exercise on selected risk-factors for osteoporotic fractures. Lancet **348:**1343–1347.
20. Jones HH, Priest JD, Hayes WC, Tichenor CC, Nagel DA 1977 Humeral hypertrophy in response to exercise. J Bone Joint Surg **59A:**204–208.
21. Huddleston AL, Rockwell D, Kulund DN, Harrison RB 1980 Bone mass in lifetime tennis athletes. JAMA **244:**1107–1109.
22. Kannus P, Haapasalo H, Sievänen H, Oja P, Vuori I 1994 The site-specific effects of long-term unilateral activity on bone mineral density and content. Bone **15:**279–284.
23. Raab DM, Smith EL, Crenshaw TD, Thomas DP 1990 Bone mechanical properties after exercise training in young and old rats. J Appl Physiol **68:**130–134.
24. Rubin CT, Bain SD, McLeod KJ 1992 Suppression of the osteogenic response in the aging skeleton. Calcif Tissue Int **50:**306–313.
25. Forwood MR, Burr DB 1993 Physical activity and bone mass: Exercises in futility? Bone Miner **21:**89–112.
26. Parfitt AM 1994 The two faces of growth: Benefits and risks to bone integrity. Osteoporos Int **4:**382–398.
27. Haapasalo H, Kannus P, Sievänen H, Heinonen A, Oja P, Vuori I 1994 Long-term unilateral loading and bone mineral density and content in female squash players. Calcif Tissue Int **54:**249–255.
28. Kannus P, Haapasalo H, Sankelo M, Sievänen H, Pasanen M, Heinonen A, Oja P, Vuori I 1995 Effect of starting age of physical activity on bone mass in the dominant arm of tennis and squash players. Ann Intern Med **123:**27–31.
29. Slemenda CW, Johnston CC 1993 High intensity activities in young women: Site specific bone mass effects among female figure skaters. Bone Miner **20:**125–132.
30. VandenBergh MFQ, DeMan SA, Witteman CM, Hofman A, Trouerbach WT, Grobbee DE 1995 Physical activity, calcium intake, and bone mineral content in children in the Netherlands. J Epidemiol Community Health **49:**299–304.
31. Uusi-Rasi K, Salmi H-M, Fogelholm M 1994 Estimation of calcium and riboflavin intake by a short diary. Scand J Nutr **38:**122–124.
32. Morris NM, Udry JR 1980 Validation of a self-administered instrument to assess stage of adolescent development. J Youth Adolesc **9:**271–280.
33. Sievänen H, Kannus P, Nieminen V, Heinonen A, Oja P, Vuori I 1996 Estimation of various mechanical characteristics of hu-

man bones using dual energy x-ray absorptiometry: Methodology and precision. Bone **18:**S17–S27.
34. Sievänen H, Oja P, Vuori I 1993 Precision of dual-energy x-ray absorptiometry in the upper extremities. Bone Miner **20:**235–243.
35. Drinkwater B, Nilsson K, Chesnut CH III, Bremmer W, Shainholtz S, Southworth M 1984 Bone mineral content of amennorrheic and eumenorrheic athletes. N Engl J Med **311:** 277–281.
36. Conroy BP, Kraemer WJ, Maresh CM, Fleck SJ, Stone MH, Fry AC 1993 Bone mineral density in elite junior olympic weightlifters. Med Sci Sports Exerc **25:**1103–1109.
37. Nichols DL, Sanborn CF, Bonnik SL, Ben-Ezra V, Gench B, DiMarco NM 1994 The effects of gymnastics training on bone mineral density. Med Sci Sports Exerc **26:**1220–1225.
38. Etherington J, Harris PA, Nandra D, Hart DJ, Wolman RL, Doyle DV, Spector TD 1996 The effect of weight-bearing exercise on bone mineral density: A study of female ex-elite athletes and the general population. J Bone Miner Res **11:** 1333–1338.
39. Meade JB 1991 The adaptation of bone to mechanical stress: Experimentation and current concepts. In: Cowin SC (ed.) Bone Mechanics. CRC Press, Boca Raton, FL, U.S.A., pp. 245–246.

Address reprint requests to:
Heidi Haapasalo, M.D.
The Bone Research Group
UKK Institute
Kaupinpuistonkatu 1
FIN-33500 Tampere, Finland

Received in original form April 28, 1997; in revised form August 25, 1997; accepted September 10, 1997.