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Exercise Before Puberty May Confer Residual Benefits in Bone Density in Adulthood: Studies in Active Prepubertal and Retired Female Gymnasts

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ABSTRACT

Exercise during growth may contribute to the prevention of osteoporosis by increasing peak bone mineral density (BMD). However, exercise during puberty may be associated with primary amenorrhea and low peak BMD, while exercise after puberty may be associated with secondary amenorrhea and bone loss. As growth before puberty is relatively sex hormone independent, are the prepubertal years the time during which exercise results in higher BMD? Are any benefits retained in adulthood? We measured areal BMD (g/cm^2) by dual-energy X-ray absorptiometry in 45 active prepubertal female gymnasts aged 10.4 ± 0.3 years (mean ± SEM), 36 retired female gymnasts aged 25.0 ± 0.9 years, and 50 controls. The results were expressed as a standardized deviation (SD) or Z score adjusted for bone age in prepubertal gymnasts and chronological age in retired gymnasts. In the cross-sectional analyses, areal BMD in the active prepubertal gymnasts was 0.7–1.9 SD higher at the weight-bearing sites than the predicted mean in controls ($p < 0.01$). The Z scores increased as the duration of training increased ($r = 0.32$–0.48, p ranging between <0.04 and <0.002). During 12 months, the increase in areal BMD (g/cm^2/year) of the total body, spine, and legs in the active prepubertal gymnasts was 30–85% greater than in prepubertal controls (all $p < 0.05$). In the retired gymnasts, the areal BMD was 0.5–1.5 SD higher than the predicted mean in controls at all sites, except the skull (p ranging between <0.06 and <0.0001). There was no diminution across the 20 years since retirement (mean 8 ± 1 years), despite the lower frequency and intensity of exercise. The prepubertal years are likely to be an opportune time for exercise to increase bone density. As residual benefits are maintained into adulthood, exercise before puberty may reduce fracture risk after menopause. (J Bone Miner Res 1998;13:500–507)

INTRODUCTION

Exercise may contribute to the prevention of osteoporosis and fractures by increasing the amount of bone accrued during growth, by reducing menopause-related and age-related bone loss, or by restoring bone already lost in the elderly. Of these periods of life, the available evidence suggests that the skeleton may be most responsive to exercise during growth.(1–4) However, during puberty, vigorous exercise may interrupt hormonal cyclicity, delay the progression of puberty, and result in the attainment of a lower peak bone mineral density (BMD).(5) After puberty, exercise may result in secondary amenorrhea, bone loss, and reduced BMD, particularly if body weight is reduced.(6) Thus, at what period during growth is exercise a valuable means of increasing peak BMD?

There are several reasons to suggest that the prepubertal years may be a uniquely opportune stage of growth during

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which the skeleton is responsive to exercise. First, growth is growth hormone and insulin-like growth factor I (IGF-I) dependent, and exercise is a potent stimulus for growth hormone secretion. Second, prepubertal growth is relatively sex hormone independent. This may be an advantage because the intensity of exercise needed to have an anabolic effect on BMD may be similar to the intensity associated with interference with sex hormone cyclicity.(7) Third, cross-sectional data comparing the playing and nonplaying arms in squash players who began training before puberty support this view.(1,2)

In the context of skeletal health, the purpose of exercise during growth is to increase peak BMD, and in doing so to reduce fracture risk in adulthood. Before recommendations can be made regarding the role of exercise before puberty in preventing fractures after menopause, the higher peak BMD must be shown to be sustained in adulthood and must be biologically significant—sufficiently large to reduce fracture risk. The aims of this study were to determine whether the prepubertal years are an opportune time for exercise to increase BMD and whether any benefits derived during this time were sustained in adulthood, despite the curtailment of vigorous exercise.

MATERIALS AND METHODS

Subjects

We studied 45 active prepubertal female gymnasts from the Victorian Institute of Sport and 35 prepubertal controls from Ivanhoe Girls Grammar School. All gymnasts and controls were Tanner breast stage 1 and had serum estradiol levels at or below the detection limit of 55 pmol/l at baseline and at 12 months follow-up. Gymnasts and controls were excluded if they were peri- or postpubertal, if they had anorexia nervosa, or exposure to oral contraceptives, anticonvulsants, or corticosteroids. Controls were excluded if they engaged in more than 6 h of weight-bearing exercise per week.

The active prepubertal gymnasts were training in an elite squad in Melbourne at a sub-Olympic standard. They trained under supervision for 15–36 h/week. Most training sessions were ~4 h and consisted of a warm up, routine training, and strength and stretching exercises. The routine training involved practicing leaps, pivots, dance, acrobatic, and aerial elements singularly and in combination on each apparatus. Both the arms and legs were loaded. Body weight was used as the resistance in the strength component. Cross training (swimming and cycling) was only used if a gymnast was injured.

The prepubertal controls were currently exercising on average 1.7 ± 0.3 h/week and their activities included netball ($n = 11$), tennis ($n = 10$), gymnastics ($n = 6$), hockey ($n = 5$), ballet ($n = 2$), and other weight-bearing activities ($n = 10$). Six were not involved in any regular weight-bearing activities. Two controls were excluded because they were involved in 12 h of weight-bearing activity per week.

Skeletal development may be delayed in gymnasts.(8) Matching with controls was done by bone age rather than chronological age because the question was whether BMD in the gymnasts was higher than the predicted BMD of a bone of comparable maturity and size. Skeletal age was determined using the Greulich and Pyle method. Tanner breast staging was used to determine pubertal status. Written permission was obtained from all participants, the parents, the Victorian Institute of Sport, and the School principal.

The 36 retired elite gymnasts were compared with 15 controls matched for age, height, and weight. Inclusion criteria were as described above. The retired gymnasts exercised at a reduced intensity, frequency, and duration after retiring. They were exercising on average 1.8 ± 0.7 h/week, and their activities included aerobic exercise ($n = 13$), weight training ($n = 12$), netball ($n = 7$), swimming ($n = 8$), and jogging ($n = 9$). Two were sedentary.

The adult controls were exercising on average 1.8 ± 0.5 h/week, and their activities included walking ($n = 5$), aerobics ($n = 3$), swimming ($n = 1$), jogging ($n = 3$), and weight training ($n = 2$). Two controls were sedentary. Their weight-bearing activities during primary and secondary school were on average 1.0 ± 0.3 and 0.6 ± 0.3 h/week, respectively.

Bone densitometry, body composition, and anthropometry

BMD and body composition were measured by dual-energy X-ray absorptiometry (DPX-L, pediatric and adult software; Lunar Corp, Madison, WI, U.S.A.). All active prepubertal gymnasts and controls were measured using pediatric software, and all retired adult gymnasts and controls were measured on adult software. Bone density was expressed as an areal BMD (mass/projected area of the region, g/cm^2) and as a volumetric bone density (mass/vol, g/cm^3). Only the latter is independent of size, providing a measure of the amount of bone in the bone. Volumetric BMD within a projected area of 4 cm^2 was calculated at the femur midshaft, assuming the shaft to be cylindrical. The pediatric anteroposterior spine program and the ruler function were used to obtain these measurements. Vertebral volumetric BMD was calculated by dividing bone mass by the vertebral body volume.(9) According to geometric scaling, the vertebral body depth = (projected area)$^{1/2}$ so that volume = (projected area)$^{3/2}$. Bone mass and the projected area of L2-L4 were obtained during posteroanterior scanning. The precision was 0.9–0.5% for total and regional BMD, 0.7% for total body soft tissue, 2.9% for total fat mass, and 1.6% for total body lean mass.

Areal BMD of each subject was standarized by calculating the difference between the observed and predicted values (based on the fitted equation and the observed characteristic of that subject) divided by the square root of the estimated variance. This derived standard score, or Z score, is a measure of the deviation from the expected population mean, adjusted for the covariance on a scale with zero mean and unit standard deviation, so that about 95% of the normal population will have a Z score between -2 and 2.(10) Testing whether the mean Z score of the gymnasts differed from zero tests the study hypotheses. The Z score for areal BMD was adjusted for bone age using a linear regression in 35 bone age-matched controls. Height was measured using a Holtain stadiometer. Tibia, humerus, and ulna lengths were measured using a Harpenden anthropometer.(11) Femur length was measured as the distance from the inferior border of the lateral epicondyle to the superior border of the greater trochanter. The coefficient of variation ranged from 0.4–1.5%.

In the retired gymnasts, the results were expressed as areal BMD (g/cm^2) and as a Z score adjusted for chronological age and derived from the age regression using 177 (locally recruited) controls. Height and segment lengths were expressed in absolute terms.

Biochemical and dietary analyses

A 12-h overnight urine sample was collected before testing. A fasting blood sample was taken and the serum was stored at −40°C. Serum osteocalcin was measured with a human-specific immunoradiometric assay (ng/ml, ELISA-OSTEO; CIS BioInternational, Gifs/Yvette, France).(12) Serum bone-specific alkaline phosphatase (BSAP) was measured with an immunoradiometric assay (ng/ml, Ostase; Hybritech Inc., San Diego, CA, U.S.A.).(13) Serum collagen propeptide of type I collagen was measured with a two site enzyme-linked immunoassay (ELISA) (PICP, ng/ml, Procollagen-C, Metra Biosystems, Palo Alto, CA, U.S.A.).(14) Bone resorption was assessed by measuring urinary type I C-telopeptide breakdown products with an ELISA (μl/mmol, CrossLaps, Osteometer A/S, Rødovre, Denmark).(15)

Three-day diet records were analyzed by a research dietitian. Each subject was interviewed with their diet diary the day after recording had been completed. Total calories, protein, calcium, and fat content were calculated using the program Diet 3, NUTTAB database (Xyris Software, Australia Pty. Ltd.). Past and present weight-bearing exercise was determined by questionnaire and validated by interview.

Statistical analyses

The absolute values and Z scores were expressed as mean ± standard error of the mean (SEM). Two sample *t*-tests were used to determine whether there were differences in areal BMD, anthropometric and biochemical measurements between the active prepubertal gymnasts and controls, and differences between areal BMD in retired gymnasts and their controls. A one-sample *t*-test was used to determine whether the Z scores differed from zero. Linear regression was used to determine whether Z scores increased with increasing duration of training in the active prepubertal gymnasts and decreased as the duration since retirement increased in the retired gymnasts.

RESULTS

Active prepubertal gymnasts

In the cross-sectional analyses, areal BMD in the active gymnasts was higher than controls (Fig. 1 and Table 1). The Z scores (mean ± SEM) were higher at the legs (0.75 ± 0.20), spine (0.78 ± 0.20), and arms (1.9 ± 0.25) than the predicted mean (of zero) in controls (*p* ranging between < 0.01 to < 0.0001). The Z scores for the skull were not increased.

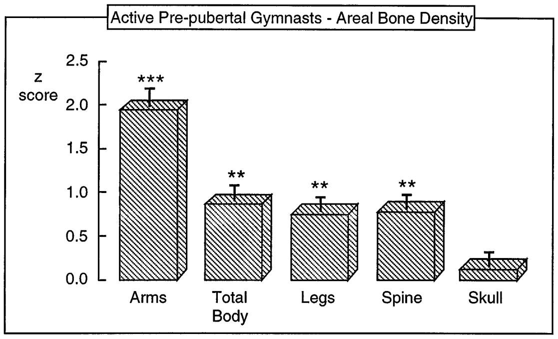

FIG. 1. Cross-sectional data showing regional areal bone density expressed as a Z score in all active female prepubertal gymnasts. The Z scores were higher than zero (the predicted mean value in controls) at each site, except at the skull. **p* < 0.05, ***p* < 0.01, ****p* < 0.001 compared with zero.

Lumbar spine bone mass, volume, and volumetric BMD were 24 ± 3, 12 ± 3, and 12 ± 2% higher than controls, respectively (all $p < 0.001$; Table 1). Femoral midshaft periosteal diameter was no greater in the gymnasts than controls, whereas endocortical (medullary) diameter was 19 ± 3% less, resulting in a greater cortical thickness, bone mass, areal, and volumetric BMD in the gymnasts (*p* ranging from < 0.05 to < 0.0001). Figure 2 shows that in the cross-sectional analysis, BMD Z scores increased at the weight-bearing sites with increased duration of gymnastic training, and the regression passed through the origin ($r = 0.32$–0.48, $p < 0.05$).

As shown in Fig. 3, during 12 months of follow-up, the annual increase in total body, spine, and leg areal BMD (g/cm^2/year) was 30–85% more rapid in the 37 gymnasts than in the 17 bone-aged matched controls, all of whom remained prepubertal (confirmed clinically and by undetectable serum estradiol levels). Bone mass increased more in the gymnasts than in the controls (2.28 ± 0.12 vs. 2.09 ± 0.16 g/year, not significant [NS]). Bone volume increased less in the gymnasts than controls (11.1 ± 1.4 vs. 14.4 ± 1.8 cm^3/year, NS). Thus, volumetric BMD increased significantly in the gymnasts but not in the controls (0.0040 ± 0.0014 vs. –0.008 ± 0.0017 g/cm^3/year, $p < 0.05$).

Anthropometry, biochemistry, and diet

Body weight of the gymnasts was no different than controls; however, lean mass was 10% greater and fat mass was 57% less. In the cross-sectional analyses, the gymnasts were 0.4 ± 0.1 SD shorter than controls due to reduced sitting height and femur and tibial lengths. Humerus and radius-ulna lengths were not reduced (Table 1). Deficits in sitting height (not leg length) increased as the duration of training increased ($r = -0.25$, $p < 0.06$). In the 12-month longitu-

TABLE 1. CROSS-SECTIONAL DATA IN ACTIVE PREPUBERTAL GYMNASTS AND BONE AGE-MATCHED PREPUBERTAL CONTROLS

Variable	*Active gymnasts (n = 45)*	*Controls (n = 35)*
Chronological age (years)	10.4 ± 0.3*	9.3 ± 0.2
Bone age (years)	9.0 ± 0.2	9.2 ± 0.2
Total body and regional areal bone density		
total body (g/cm^2)	0.90 ± 0.01*	0.87 ± 0.01
spine (g/cm^2)	0.82 ± 0.01*	0.77 ± 0.01
leg (g/cm^2)	0.86 ± 0.02†	0.82 ± 0.01
arm (g/cm^2)	0.68 ± 0.01‡	0.61 ± 0.01
skull (g/cm^2)	1.61 ± 0.02	1.60 ± 0.02
Dimensions and volumetric bone density		
lumbar spine, L2–L4		
bone mass (g)	19.9 ± 0.5‡	16.0 ± 0.7
volume (cm^3)	125 ± 4†	112 ± 4
volumetric density (g/cm^3)	0.161 ± 0.003*	0.144 ± 0.005
femoral midshaft		
periosteal diameter (mm)	16.5 ± 0.3	16.7 ± 0.4
endocortical diameter (mm)	5.3 ± 0.2*	6.5 ± 0.5
bicortical thickness (mm)	11.2 ± 0.3†	10.1 ± 0.4
bone mass (g)	6.3 ± 0.1‡	5.5 ± 0.1
volumetric density (g/cm^3)	1.22 ± 0.03*	1.05 ± 0.03
Anthropometry and body composition		
total height (cm)	132.3 ± 1.1	135.0 ± 1.4
sitting height (cm)	69.7 ± 0.5†	71.4 ± 0.7
femur length (mm)	336 ± 4§	343 ± 5
tibial length (mm)	298 ± 3†	308 ± 4
humerus length (mm)	283 ± 3	284 ± 3
radius length (mm)	207 ± 2	209 ± 2
lean mass (kg)	23.2 ± 0.6†	21.1 ± 0.7
fat mass (kg)	2.7 ± 0.1‡	6.3 ± 0.7

Chronological age, bone age, total body and regional areal bone density, lumbar spine and femoral midshaft dimensions, bone mass and volumetric bone density, anthropometry, and body composition.

* $p < 0.01$, † $p < 0.05$, ‡ $p < 0.001$, § $p < 0.08$. Active prepubertal gymnasts versus controls.

Values are mean ± SE.

dinal analyses, the gymnasts grew more slowly than controls in sitting height (1.7 ± 0.1 vs. 2.4 ± 0.1 cm/year, respectively; $p < 0.001$), femur length (15.7 ± 1.1 vs. 19.1 ± 1.4 mm/year, respectively; $p < 0.06$), and tibial length (9.0 ± 0.7 vs. 12.7 ± 0.8 mm/year, respectively; $p < 0.01$).

The respective biochemical results (gymnasts vs. controls) were: BSAP (64 ± 5 vs. 80 ± 6 ng/ml, $p < 0.05$), osteocalcin (90 ± 7 vs. 109 ± 6 ng/ml, $p < 0.05$), procollagen I carboxy-terminal propeptide (244 ± 17 vs. 252 ± 16 ng/ml, NS), urinary type I collagen C-telopeptide (1206 ± 91 vs. 1306 ± 79 μl/mmol, NS), IGF-I (92 ± 6 vs. 114 ± 7 ng/ml, $p < 0.05$), and growth hormone (14 ± 3 vs. 11 ± 2 ng/ml, NS). IGF-I correlated with bone age ($r = 0.27$, $p < 0.02$), height ($r = 0.38$, $p < 0.001$) and bone mass ($r = 0.31$, $p < 0.03$).

The gymnasts had reduced daily intakes of energy (6106 ± 388 vs. 7949 ± 320 KJ), carbohydrates (185 ± 11 vs. 243 ± 11 g), and fat (43 ± 4 vs. 75 ± 3 g) (all $p < 0.001$), but not protein (67 ± 4 vs. 67 ± 4 g), or calcium (843 ± 58 vs. 743 ± 70 mg). Total daily energy and fat intake were independently associated with growth rate in height for the total sample ($p < 0.05$). However, the differences in tempo of growth in gymnasts and controls were not accounted for by differences in dietary and hormonal measurements.

Retired gymnasts

At the time of study, the former elite gymnasts were 18–35 years of age and had been retired for 8 years (range 1.5–20 years). They were 7.5 ± 0.4 years of age at the start of training (range 3–12 years), they trained for 9.5 ± 0.5 years (range 4–8 years), and retired at 16.7 ± 0.4 years of age (range 12–24 years). They trained for 14–19 h weekly between the ages of 12 and 17, and they had delayed menarche, higher lean mass, and lower fat mass. Areal BMD was 0.7–1.4 SD or 6–16% higher at all sites, except at the skull (p ranging < 0.05 to < 0.001, Table 2) and did not diminish with increasing duration since retirement (p ranging < 0.05 to < 0.001; Figs. 4 and 5).

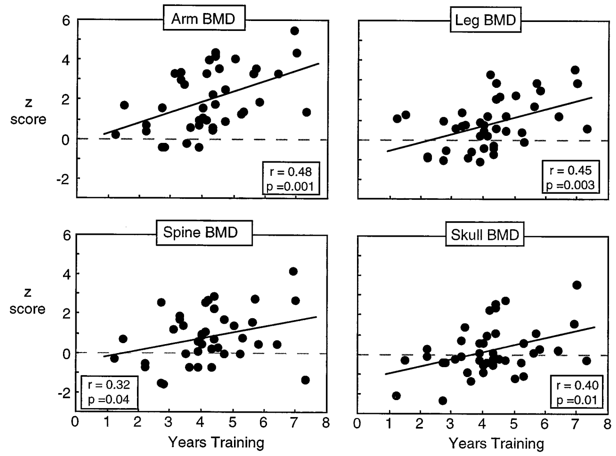

FIG. 2. Cross-sectional data showing regional areal bone density expressed as a Z score in active prepubertal gymnasts increased with increasing duration of gymnastic training.

DISCUSSION

The view that exercise in adulthood is a useful means of preventing osteoporosis is largely based on the following: athletes have higher bone and muscle mass than controls, there are positive associations between bone mass and lean mass in cross-sectional studies, and immobilization causes bone loss. The inference made is that exercise, or the lack of it, is responsible for these observations. However, genetic factors, rather than exercise, may account for the association between bone mass and lean mass,(16) while the occurrence of bone loss with immobilization is neither the test nor the proof of the hypothesis that exercise increases bone mass. This hypothesis is tested by exercise intervention studies.

Intervention studies do not substantiate the notion that exercise is an effective means of increasing areal BMD, at least in adulthood.(17–21) Few investigators report an increase in areal BMD of 8–10%,(22) amounts sufficient to halve fracture risk.(23) Most studies report increases of 1–3%, or less than 0.5 SD, amounts unlikely to reduce fracture risk.(17–21)

By contrast, areal BMD was 0.8–2 SD or 8–20% higher in this study, i.e., an order of magnitude higher than reported in exercise studies in adults. In the cross-sectional analysis, the areal BMD Z scores increased with increasing duration of training.

The longitudinal data support the cross-sectional observations; increases in areal BMD in the gymnasts were 30–85% greater than controls. Volumetric bone density—the amount of bone in the growing bone—increased in the gymnasts. In the controls, the increase in mass and size was commensurate so that volumetric BMD did not increase.(24)

Bias is unlikely to explain the higher areal BMD because (1) the Z score or deviation above the predicted mean increased with increasing duration of training, with the regression line passing through zero. If the sampling bias were present, this correlation would not be expected. (2) There was site specificity: areal BMD was increased in the arms and legs; weight-bearing sites in gymnasts, but not increased at the skull. (3) The longitudinal data support the cross-sectional data.

Evidence that the growing skeleton is more responsive to exercise than the fully grown skeleton is limited.(1,2) Studies in humans are confined to comparisons of the playing versus the nonplaying arm in racquet sports.(1,2,25,26) Squash or tennis players who began playing before puberty had 11–24% higher bone mass in their playing than nonplaying arm, two to four times the side-side difference of individuals who began the sport after puberty.(2) Jones et al.(26) showed that cortical thickness of the humerus in the playing arm was 35% higher than the nonplaying arm in 48 males aged 18–50 years and 28% higher in females aged 14–34 years involved in tennis. Huddleston et al.(25) found 13% higher radial bone mass of the playing arm relative to the nonplaying arm of 35 tennis players aged over 70 years. In these studies, greater cortical thickness was the result of both increased periosteal diameter and/or reduced endocortical (medullary) diameter. In this study, cortical thickness was greater at the femur because medullary diameter was re-

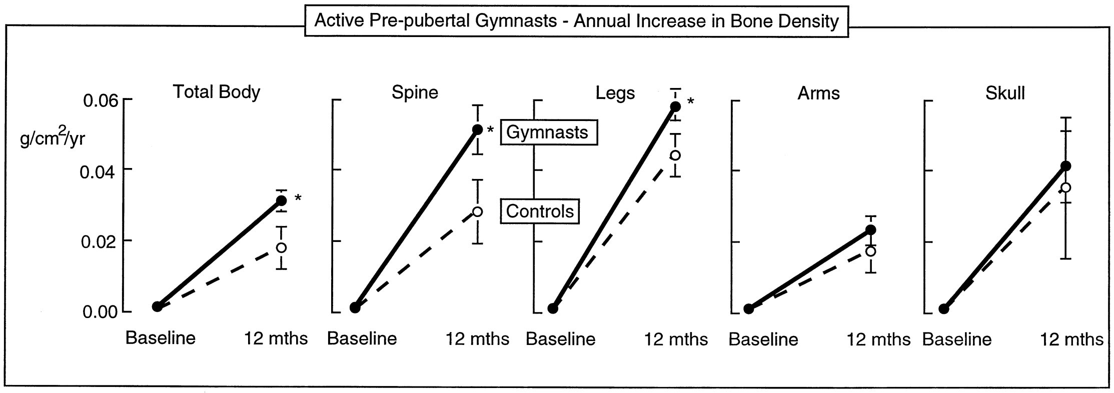

FIG. 3. Longitudinal data showing that during 12 months, total body, spine, and leg areal bone density increased 30–85% more rapidly in active prepubertal gymnasts than in bone age-matched prepubertal controls. *$p < 0.05$ compared with controls.

TABLE 2. CROSS-SECTIONAL DATA IN RETIRED ADULT GYMNASTS AND AGE-MATCHED CONTROLS

Variable	*Retired gymnasts* (n = *36)*	*Controls* (n = *15)*
Chronological age (years)	25.0 ± 0.9	25.3 ± 1.0
Age menarche (years)	14.4 ± 0.3*	13.3 ± 0.5
Total body and regional areal bone density		
total body (g/cm^2)	1.19 ± 0.01*	1.15 ± 0.02
femoral neck (g/cm^2)	1.16 ± 0.03†	1.00 ± 0.03
Ward's triangle (g/cm^2)	1.10 ± 0.03†	0.97 ± 0.03
trochanter (g/cm^2	0.94 ± 0.02‡	0.81 ± 0.03
lumbar spine (g/cm^2)	1.31 ± 0.02§	1.24 ± 0.03
arms (g/cm^2)	0.93 ± 0.01†	0.85 ± 0.02
legs (g/cm^2)	1.25 ± 0.01†	1.19 ± 0.02
skull (g/cm^2)	2.19 ± 0.03	2.24 ± 0.05
Anthropometry and body composition		
height (cm)	163.4 ± 0.8	165.5 ± 1.8
weight (kg)	58.9 ± 1.1	60.7 ± 1.9
lean mass (kg)	40.7 ± 0.6§	38.6 ± 1.0
fat mass (kg)	15.7 ± 0.7*	19.2 ± 1.4

Chronological age, age at menarche, total body and regional areal bone density, anthropometry, and body composition.
$^*p < 0.05$, $^{\dagger}p < 0.01$, $^{\ddagger}p < 0.001$. $^{\S}p < 0.06$. Retired gymnasts versus controls.
Values are mean ± SE.

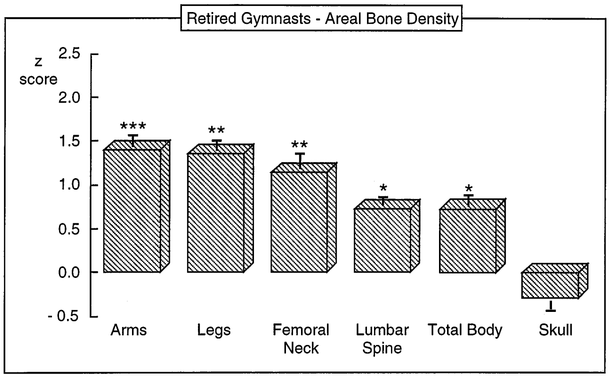

FIG. 4. Cross-sectional data showing regional areal bone density expressed as a Z score in retired gymnasts. The Z scores were higher than the predicted mean value in controls (represented by zero) at each site except the skull. $^*p < 0.05$, $^{**}p < 0.01$, $^{***}p < 0.001$ compared with zero.

duced as a result of reduced endocortical resorption or increased endocortical bone formation during growth.

Before recommendations can be made regarding the role of exercise before puberty in preventing fractures after menopause, the benefits must be shown to be maintained into adulthood. The evidence in retired gymnasts suggests that this is likely. Areal BMD was 0.5–1.5 SD higher at the weight-bearing sites but not at the skull. These are biologically worthwhile benefits because a 1 SD higher areal BMD is associated with a halving of fracture risk.(23) Moreover, there was no detectable diminution in areal BMD with advancing years since retirement, despite the lower activity level.

In support of the observation in retired gymnasts, Lindholm et al. reported that 19 retired gymnasts had 7% higher areal BMD of the arms than controls.(27) Kirchner et al. reported that 18 retired collegiate gymnasts who began training at 12 years had 9–22% higher areal BMD 18 years after retirement.(28) The retired gymnasts in these two studies, and in this study, were 21–36 years old. Fragility fractures usually occur later in life. Sustained biologically worthwhile increases in areal BMD have been found in other groups of retired athletes ranging in age from 18–50 years,(26) 50–64 years,(29) and over 70 years.(25) Karlsson et al. reported that 50- to 64-year-old retired weight lifters had 9–17% higher areal BMD than controls.(29) The authors concluded that the effects of exercise attenuated with time because the over 65-year-olds did not have higher areal BMD. However, there were only nine subjects over 70 years of age; a residual benefit may have been missed.

The active gymnasts had reduced leg length, sitting height, and growth velocity during 12 months of observation. Reduced growth in gymnasts is associated with a negative energy balance and low IGF-I levels.(30–32) Inaccuracies in dietary questionnaires and biological variation in biochemical measurements may explain why the lower daily intake of energy, carbohydrate, and fat did not account for the slower growth. Compression of the epiphysial growth plates may also contribute to reduced growth in the legs.(33) Reduced bone turnover reflected in the lower osteocalcin and alkaline phosphatase was found in this study and is likely to be due to physical activity and low dietary kilojoules.(34,35)

Thus, this study and the published literature in animals and humans provides consistent evidence that the prepubertal years are likely an opportune time when exercise

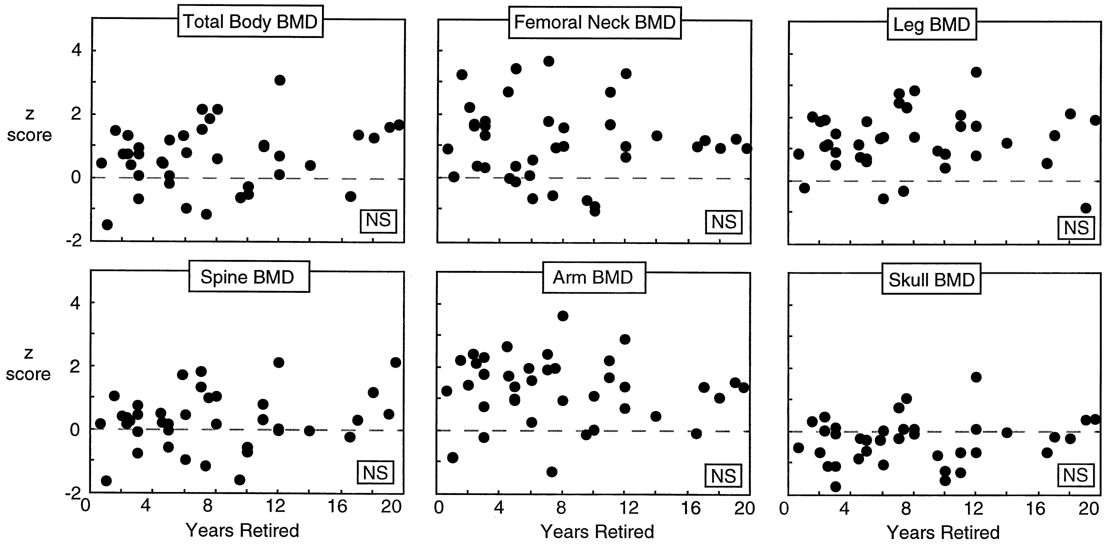

FIG. 5. Cross-sectional data showing total body and regional area bone density expressed as a Z score in retired gymnasts does not diminish with increased number of years since retirement.

increases volumetric BMD. The increments achieved by vigorous exercise are large and are likely to reduce fracture risk 2- to 4-fold, whereas exercise in adulthood has effects on areal BMD that are of little biological value in terms of fracture risk reduction. The greater responsiveness of the growing skeleton is likely to provide a lasting residual benefit in adulthood despite the lower frequency and intensity of exercise.

Despite having residual benefits, the retired gymnasts had a relative lower areal BMD compared with the active prepubertal gymnasts. The reasons for this are uncertain: exercise training one to two decades ago may have been less intense so the retired gymnasts may have attained a lower peak areal BMD compared with the current elite gymnasts; the gymnasts may experience some bone loss early in retirement; alternatively, if the athletes trained during the peripubertal years and had primary amenorrhea, the estrogen-dependent increase in bone mass at puberty may not have occurred.

The effects of gymnastics and weight lifting are extreme models of what is possible. However, there are several studies that suggest that there is a positive association between more moderate exercise in childhood and bone density.(3,4,36,37) Studies are needed to determine the mechanisms responsible for the greater responsiveness of the growing (modeling) skeleton to exercise than the adult (remodeling) skeleton, and to determine the mechanisms responsible for the maintenance or partial maintenance of the benefits in adulthood, despite less intensive exercise.

In conclusion, the prevention of fractures requires recognition that BMD in old age is determined by factors operating during growth. Of these, exercise may be an important and modifiable protective factor. Urbanization and electronic forms of home entertainment may result in reduced physical activity in children at a time when most benefits are to be gained. A reduction in the numbers of fractures in old age may be achieved by attention to exercise during the prepubertal years of growth.

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