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Age at Onset of Puberty Predicts Bone Mass in Young Adulthood

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Abstract

Objective—To determine whether the commencement and length of puberty influences dual x-ray absorptiometry (DXA) values of bone mineral content (BMC) and bone mineral density (BMD) in the axial and appendicular skeleton at skeletal maturity.

Study design—From the Bone Mineral Density in Childhood Study, we identified children who began puberty and completed sexual and skeletal development and examined whether the timing and length of puberty influence DXA values of BMC and BMD at skeletal maturity.

Results—A total of 78 girls and 85 boys began puberty and completed skeletal maturity; 4.4 ± 0.8 and 4.5 ± 0.8 years later, respectively. Multiple linear regression analyses indicated that the age of onset of puberty was a strong negative predictor of DXA bone measurements at skeletal maturity, independent of bone values at the beginning of puberty, and the length of puberty. This negative relation was observed for all BMC and BMD measurements at all skeletal sites, in both boys and girls (all $P < .0001$). In contrast, length of puberty had no relation to any measures of bone.

Conclusions—In healthy adolescent males and females, bone mass and bone density at skeletal maturity are inversely related to the timing of puberty.

Peak bone mass (PBM), a major determinant of the future risk of fractures in the elderly, is largely achieved by the end of sexual and skeletal maturity.[1,2] The greatest accretion of bone occurs during puberty, and low PBM may result from clinical states associated with abnormal pubertal development.[1–3] Idiopathic delayed puberty in females is a cause for reduced PBM,[4] and amenorrheic teenage girls have lower bone density than girls with normal menses.[5] Likewise, delayed puberty and constitutional delay in male teenagers results in decreased bone mineralization and lower PBM.[6–8] Genetic males with complete

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androgen insensitivity (testicular feminization) experience increased pubertal growth but achieve a bone mass less than expected of androgen-replete men.[9–11] Estrogens are also needed for the achievement of adequate PBM in males, and aromatase deficiency or estrogen receptor defects in men result in tall stature and severe osteoporosis.[12–15]

Although the amount of bone gained during adolescence is the main contributor to PBM, a greater understanding of the influence that normal variations in sexual development have on bone acquisition during growth would facilitate the planning of strategies to enhance PBM. Previously, we have shown that, in healthy girls and boys, bone mass in the axial and appendicular skeletons at early puberty is the strongest predictor of values at sexual maturity.[16] However, both age and duration of puberty vary greatly; whether the marked variability in pubertal development, even within the normal range, also influences PBM has yet to be defined. The purpose of this prospective longitudinal multicenter study was to determine whether the timing of commencement and length of puberty in contemporary children in the United States influence dual x-ray absorptiometry (DXA) values of bone mineral content (BMC) and bone mineral density (BMD) in the axial and appendicular skeletons at skeletal maturity.

Methods

The Bone Mineral Density in Childhood Study is an ongoing multicenter longitudinal study examining bone accretion in 1554 healthy children and teenagers of both sexes and different ethnic groups in the United States. From this unique subject pool, we identified children who began puberty and completed sexual and skeletal development during the duration of the study. Participants were recruited from July 2002 to November 2003 at 5 medical centers: Children's Hospital Los Angeles (Los Angeles, California), Cincinnati Children's Hospital Medical Center (Cincinnati, Ohio), Creighton University (Omaha, Nebraska), Children's Hospital of Philadelphia (Philadelphia, Pennsylvania), and Columbia University (New York, New York). Participants were seen annually for measurements. The Institutional Review Board at each clinical center approved the protocol, and consent was obtained from each participant's parent or guardian, and assent was obtained from the study participants.

From this cohort, we chose all 78 girls and 85 boys that started puberty and achieved sexual and skeletal maturity during the duration of the study. For the purpose of this study, baseline measures were obtained at Tanner II stage of sexual development, and follow-up examinations were taken when subjects reached sexual (Tanner V) and skeletal maturity; for this study skeletal maturation was defined as epiphyseal closure of the phalanges and metacarpals, corresponding to bone ages of 16 for females and 17 for males.

Detailed information about the study participants, inclusion/exclusion criteria, and study procedures have been published previously.[17] Briefly, the sample was selected to reflect healthy, normally developing children in the United States. The following inclusion criteria were used: residence in United States for at least 3 years, school placement within 1 year of that expected for age, full-term birth (≥37 weeks gestation), birth weight > 2.3 kg, and no evidence of precocious or delayed puberty. For this study normal puberty was defined as

breast development beginning between 8 and 13 years for girls, and testes size of at least 4 mL between 9 and 14 years for boys.

Exclusion criteria were height, weight, or body mass index (BMI; kg/m^2) less than third or more than ninety-seventh percentile; current or previous medical condition known to affect growth, maturation, physical activity, or nutritional status, and medications known to affect growth, maturation, or bone mineral accrual such as steroids. Subjects with indwelling hardware; abnormality of the skeleton or spine such as scoliosis 20 degrees or more, kyphosis, or skeletal dysplasia by history; current or previous pregnancy; same-sex sibling enrolled in the Bone Mineral Density in Childhood Study; and participation in a diet or exercise intervention study in the previous year were also excluded from participation.

Height and weight measurements were obtained with participants dressed in examination gowns or lightweight clothing, without shoes. Body mass index (BMI) percentile was calculated with the Centers for Disease Control 2000 growth charts.[18] All subjects underwent a physical examination. The maturational stage of breast development in girls and testicular volume by orchidometer in boys was evaluated on the basis of standard endocrine practice and the criteria of Tanner.[19] Skeletal maturity was assessed on the basis of roentgenograms of the left hand and wrist obtained according to the method of Greulich and Pyle.[20]

Bone Densitometry

DXA scans were performed with Hologic, Inc. (Bedford, Massachusetts) bone densitometers (QDR4500A, QDR4500W, and Delphi A models). Scans were performed on a single densitometer at each center. The software versions used for acquisition varied from version 11.1 to 12.3. The following scans were performed according to manufacturer guidelines for subject positioning: whole body, posteroanterior lumbar spine (L1–L4, fast array), nondominant forearm, and left proximal femur (fast array). At study baseline and in year 3, the calibration of scanners was assessed by having all centers scan a single set of traveling phantoms that included the European Spine and Forearm Phantoms (QRM Inc, Mohrendorf, Germany) and the Hologic block, hip, and whole-body phantoms. The long-term calibration stability was monitored at each clinical site with two site-specific phantoms (Hologic anthropomorphic spine and whole-body phantoms) that were scanned weekly. All scans were analyzed centrally by the DXA Core Laboratory (University of California, San Francisco). The precision error for BMD and BMC were less than 1% for the spine phantom, and less than 2.5% for the whole-body phantom.

Statistical Analysis

Statistical analyses were conducted with Statview (version 5.0.1; SAS Institute Inc, Cary, North Carolina). Pearson correlations were used to examine associations between variables, and multiple regression analyses were used to determine the influence of the baseline bone measure, baseline chronological/bone age, and pubertal duration on values at PBM, corresponding to the time that both Tanner V and skeletal maturity were achieved.

To exclude the possibility of multicollinearity on the multiple regression models, postestimation procedures were used to calculate a condition number for the regression

models and comparing the condition number with the suggested cutoff value of 15.[21] Models with condition numbers less than 15 were judged to not have any substantial collinearity problems that would affect the results or the conclusions. The goodness of fit for the regression models was evaluated with the postestimation procedures of STATA (StataCorp, College Station, Texas). All models presented passed the following goodness of fit criteria: residuals appeared random and no strong influence or leverage points were present, on the basis of both a graphical and distribution evaluation.

Results

The age, anthropometric characteristics, and DXA bone measures at baseline and follow-up for boys and girls are described in Table I. As expected, the height, weight, and DXA measurements of BMC at all sites were significantly higher in males than in females at baseline and follow-up (all $P < .001$). Values for BMD for total body and the appendicular skeleton were also higher in males than in females (all $P < .001$), but there were no sex-related differences in BMD values for the axial skeleton ($P = .45$ and .57).

All subjects achieved sexual maturity earlier than skeletal maturity; on average 1.3 ± 1.0 years earlier for girls and 1.7 ± 1.2 years earlier for boys. Boys overall commenced puberty 1 year later and achieved both sexual and skeletal maturity approximately 1 year later than girls. When divided by ethnic group, 33 were African American (19 male and 14 female), 10 subjects were Asian (6 male and 4 female), 90 subjects were Caucasian (46 male and 44 female), and 30 were Hispanic (14 male and 16 female). Regardless of sex, there were no differences in the duration of puberty between African Americans, Asians, Caucasians, and Hispanics. Whereas there was no ethnic or racial difference in the age at the onset of puberty (Tanner 2) or completion (Tanner 5) in males, African American and Hispanic females started and completed puberty approximately 6 months earlier than Caucasians.

Values for the simple correlations between DXA measurements and age and anthropometrics at baseline (Tanner II) and follow-up (skeletal maturity) are described in Table II (available at www.jpeds.com). These correlations were stronger at baseline than at follow-up, and tended to be stronger for height and weight than for age and bone age in females and males. There were also moderate correlations between baseline and follow-up BMC and BMD values at all skeletal sites (r values between 0.53 and 0.77, all P values < .001). Regardless of sex, significant correlations were present between the age of pubertal commencement (Tanner 2) and all baseline DXA measurements (r values between 0.29 and 0.55, all P values < .02). In contrast, pubertal length did not correlate with baseline DXA measurements in boys or girls (r values between 0.02 and −0.15, all P values > .05). Additionally, pubertal length and age of pubertal commencement (Tanner 2) did not correlate significantly (r = −0.17 and $P = .14$ for females and r = −0.18 and $P = .10$ for males).

Multiple linear regression analyses indicated that both baseline bone values and the age of the onset of puberty independently predicted DXA measurements at skeletal maturity. This was true for BMC and BMD measurements at all skeletal sites and for boys and girls, regardless of whether chronological age or bone age was used in the model (Tables III and

IV). However, although the baseline bone value had a positive predictive value with all DXA phenotypes, a negative effect was observed between the age of pubertal onset and all of these measures. The independent reciprocal relations between the timing of puberty commencement and all DXA measurements persisted even after adjusting for the possible confounding effect of height at all sites in both sexes (Tables V and VI, available at www.jpeds.com).

On the basis of the equations obtained from the multiple regression models, percent change in peak BMC and BMD values were calculated. Variations in predicted peak BMC and BMD measures for total body, spine, and upper and lower extremities for girls, 8 to 13 years of age, in relation to the mean pubertal age (10.7 ± 1.0 years) are shown in Figures 1, A, and 2, B (available at www.jpeds.com). On average, for every year, peak BMC values change 4.7% to 5.1%, and peak BMD values change 1.6% to 3.9% depending on skeletal site. Likewise, Figures 1, B, and 2, B, show the percent changes in BMC and BMD for boys starting puberty between ages 9 and 14 in reference to the mean age of pubertal commencement (11.7 ± 1.0 years) (Figure 2, B). On average, for every year, peak BMC values change between 2.5% to 3.9%, and peak BMD values change 1.9% to 3.1%. Similar findings for all bone phenotypes were observed when percent change in peak BMC and BMD in relation to the timing of pubertal commencement were calculated by use of the skeletal age (data not shown).

Discussion

The amount of bone gained during puberty is the main contributor to PBM, which, in turn, is a major determinant of osteoporosis and fracture risk in the elderly. The results of this longitudinal study provide strong evidence that the timing of puberty is a negative independent predictor of PBM. We found a strong reciprocal relation between all DXA values of bone mass and bone density at skeletal maturity and variations in the timing of puberty within the normal range. This negative relation was observed in both healthy young males and females, was present in the axial and appendicular skeleton, and was independent of the major known determinant of PBM: the bone value at the beginning of puberty. On average, healthy girls starting puberty a year earlier had approximately 5% greater BMC measures and 2.5% greater BMD values at skeletal maturity, but those starting a year later had 5% and 2.5% less. Similar findings of a slightly smaller magnitude were observed in healthy boys. Our findings that changes in the tempo of puberty within the normal range in males and females negatively effects PBM at all skeletal sites were also demonstrated when bone age rather than chronological age was used for the analyses.

Although the earlier the beginning of puberty, the higher the PBM at skeletal maturity, variations in pubertal length did not significantly influence bone accretion because both slow and fast sexually maturing male and female teenagers achieve similar PBM. This lack of association was observed for both BMC and BMD measurements at all skeletal sites.

Pubertal activation of sexual development accelerates skeletal growth and bone accretion, leading to epiphyseal fusion. In comparison with previous reports, our study is strengthened by highly detailed and standardized assessments of these physiological changes associated

with pubertal development. The degrees of sexual and skeletal development were assessed yearly by pediatric endocrinologists and pediatric radiologists, respectively, and all DXA bone measurements were analyzed at a central core facility following rigorous acquisition and analyses protocols. Hence, although the number of subjects examined in this study is relatively small, they represent a well-characterized longitudinally analyzed cohort of healthy, normally developing adolescents in the United States.

This study has some notable limitations. Although we did not account for known determinants of bone accretion during growth, such as dietary intake and physical activity, it is unlikely that this omission would affect our findings because it pertained to all subjects. Another limitation of this study is that the evaluation was restricted to adolescents with normally timed puberty, and replication of our findings in the extremes of the normal population is needed to further strengthen the claim that the age of pubertal commencement is a strong determinant of PBM. Nonetheless, even greater impairments in PBM are likely to occur in subjects with constitutional delay of puberty, a common clinical condition potentially affecting up to 3% of otherwise normal adolescents. If left untreated, these children will attain a full sexual maturity spontaneously, albeit at later chronological age and, as available data would suggest, with lower PBM than their peers.[4,7,8,11,14,15,22,23]

The effect of pubertal timing on PBM has become the center of considerable attention because of reports of adverse effects of treatments aimed at augmenting the height of adolescents with short stature. It was recently shown that prolonging the growth period of short children with normally timed puberty, by delaying sex hormone-induce growth-plate senescence, may increase final height but substantially decreases PBM.[24] Our findings indicate that minor delays in pubertal growth and maturation, even within the normal range, result in a deficit in PBM. This concurs with these foregoing observations, thus stressing the need for caution in the use of treatments aimed at prolonging the growth period, as they might result in reduced adult bone mass.

The care of patients with osteoporosis is difficult, and most interventions increase bone density by modest amounts despite long periods of treatment. In contrast, large increases in bone density occur over a relatively brief period during puberty.[25,26] Because the rate of decline in bone mass in adulthood is approximately 1% to 2% per year, a 10% to 20% difference in bone density because of the normal variations in the timing of puberty corresponds to an additional 10 to 20 years of protection against the normal age-related decline in skeletal mass.[27,28] The 2000 National Institutes of Health Consensus Development Conference on Osteoporosis Prevention, Diagnosis, and Therapy identified bone mineral accretion during adolescence as a critical determinant of osteoporosis risk later in life.[29] The results of this study provide further evidence of the importance of the timing of pubertal commencement as a strong independent predictor of bone mass and bone density in healthy young adults. They underscore the need for additional studies to establish whether the potential deficiency in PBM in adolescents with delays in pubertal commencement, even within the normal range, can be prevented as a result of simple nutritional, mechanical, or pharmacologic intervention.

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Glossary

BMC	Bone mineral content
BMD	Bone mineral density
BMI	Body mass index
DXA	Dual x-ray absorptiometry
PBM	Peak bone mass

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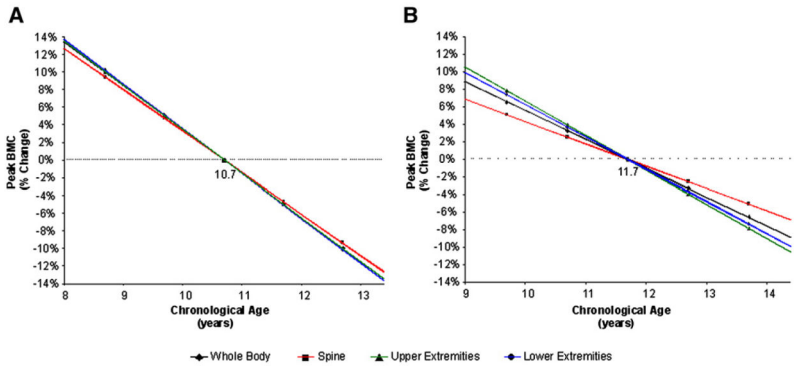

Figure 1.

Predicted percent change in peak BMC over the normal range of pubertal commencement for **A,** girls and **B,** boys as compared with the mean peak BMC at mean age of pubertal commencement (10.7 for girls and 11.7 for boys). Data points represent one and two standard deviations from the mean age of pubertal onset.

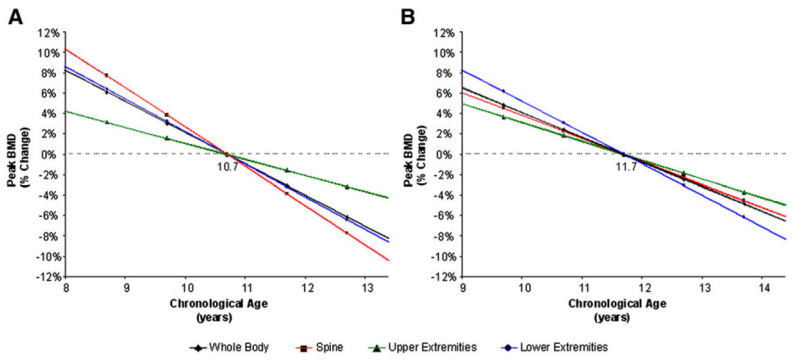

Figure 2.

Predicted percent change in peak BMD over the normal range of pubertal commencement for **A,** girls and **B,** boys as compared with the mean peak BMD at mean age of pubertal commencement (10.7 for girls and 11.7 for boys). Data points represent one and two standard deviations from the mean age of pubertal onset.

Table I

Age, anthropometric characteristics, and DXA measures for 78 females and 85 males at baseline (Tanner 2) and follow-up (sexual and skeletal maturity)

	Females		Males	
	Baseline	**Follow-up**	**Baseline**	**Follow-up**
Age (y)	10.7 ± 1.0	15.1 ± 1.0	11.7 ± 1.0	16.2 ± 0.9
Bone age (y)	11.2 ± 1.0	16.1 ± 0.4	11.3 ± 1.6	17.2 ± 0.4
Height (cm)	144.0 ± 7.3	163.5 ± 6.1	148.3 ± 6.8	174.6 ± 5.8
Weight (kg)	39.2 ± 8.1	59.3 ± 8.8	41.5 ± 7.5	67.3 ± 9.6
BMI (kg/m^2)	18.8 ± 2.9	22.1 ± 2.9	18.8 ± 2.6	22.1 ± 3.1
BMI percentile (%)	59.9 ± 28.4	65.5 ± 23.1	58.1 ± 28.2	58.1 ± 26.8
Whole body BMC (g)	938 ± 193	1572 ± 245	1015 ± 172	1973 ± 243
Whole body BMD (g/cm^2)	0.75 ± 0.07	0.94 ± 0.80	0.79 ± 0.06	1.03 ± 0.08
Spine BMC (g)	28.5 ± 6.4	54.6 ± 9.8	30.3 ± 5.6	62.8 ± 8.2
Spine BMD (g/cm^2)	0.67 ± 0.10	0.98 ± 0.12	0.65 ± 0.08	0.97 ± 0.10
Upper extremities BMC (g)	74.4 ± 15.0	133.6 ± 21.6	82.2 ± 16.8	178.3 ± 25.5
Upper extremities BMD (g/cm^2)	0.57 ± 0.05	0.70 ± 0.04	0.63 ± 0.05	0.80 ± 0.05
Lower extremities BMC (g)	246.5 ± 52.0	378.7 ± 62.3	266.4 ± 49.7	483.6 ± 63.9
Lower extremities BMD (g/cm^2)	0.90 ± 0.09	1.12 ± 0.10	0.94 ± 0.08	1.24 ± 0.11
Tanner 2 to sexual maturity (y)	-	3.1 ± 1.0	-	2.8 ± 1.0
Tanner 2 to skeletal maturity (y)	-	4.4 ± 0.8	-	4.5 ± 0.8

All measures Mean ± SD

Table II

Correlations between anthropometric measures and DXA bone values at baseline and follow-up

	Females					Males				
	Age	**Bone age**	**Height**	**Weight**	**BMI**	**Age**	**Bone age**	**Height**	**Weight**	**BMI**
Baseline										
Whole body BMC	0.55	0.50	0.79	0.74	0.44	0.52	0.68	0.67	0.63	0.38
Whole body BMD	0.47	0.49	0.69	0.66	0.41	0.35	0.48	0.49	0.42	0.23
Spine BMC	0.48	0.43	0.72	0.65	0.37	0.43	0.61	0.55	0.44	0.22
Spine BMD	0.33	0.36	0.59	0.63	0.43	0.27	0.36	0.32	0.28	0.16
Upper extremities BMC	0.55	0.43	0.72	0.72	0.47	0.47	0.65	0.63	0.67	0.46
Upper extremities BMD	0.40	0.50	0.59	0.58	0.39	0.29	0.38	0.48	0.40	0.23
Lower extremities BMC	0.54	0.53	0.81	0.73	0.42	0.50	0.68	0.67	0.63	0.38
Lower extremities BMD	0.48	0.53	0.68	0.69	0.46	0.38	0.53	0.50	0.46	0.28
Follow-up										
Whole body BMC	0.20	0.07	0.76	0.63	0.28	0.10	−0.06	0.49	0.52	0.32
Whole body BMD	0.05	0.02	0.43	0.35	0.16	−0.02	−0.09	0.20	0.25	0.17
Spine BMC	0.16	0.12	0.64	0.42	0.11	0.05	0.02	0.33	0.36	0.22
Spine BMD	0.01	0.09	0.44	0.41	0.21	0.04	−0.03	0.08	0.37	0.35
Upper extremities BMC	0.26	0.11	0.69	0.69	0.39	0.10	0.00	0.40	0.58	0.43
Upper extremities BMD	0.12	0.03	0.35	0.30	0.16	−0.01	−0.04	0.23	0.36	0.28
Lower extremities BMC	0.17	0.04	0.75	0.59	0.25	0.08	−0.08	0.49	0.44	0.24
Lower extremities BMD	0.02	0.01	0.40	0.33	0.17	−0.03	−0.08	0.16	0.16	0.09

Table III

Multiple regression models of DXA measures for 78 females with length of puberty, Tanner 2 chronological age, and Tanner 2 bone measure as independent variables

	B	σ	β	P
Whole body peak BMC (R^2 = 0.56)				
Tanner 2 chronological age	−78.234	23.473	−0.311	.001
Tanner 2 whole body BMC	1.093	0.117	0.860	.000
Length of puberty	35.906	19.690	0.142	.072
Whole body peak BMD (R^2 = 0.60)				
Tanner 2 chronological age	−0.029	0.007	−0.359	.000
Tanner 2 whole body BMD	0.963	0.093	0.868	.000
Length of puberty	0.009	0.006	0.116	.126
Spine peak BMC (R^2 = 0.48)				
Tanner 2 chronological age	−2.565	0.974	−0.254	.010
Tanner 2 spine BMC	1.160	0.146	0.758	.000
Length of puberty	1.473	0.857	0.146	.090
Spine peak BMD (R^2 = 0.69)				
Tanner 2 chronological age	−0.038	0.008	−0.316	.000
Tanner 2 spine BMD	1.020	0.080	0.880	.000
Length of puberty	0.008	0.008	0.067	.309
Upper extremities peak BMC (R^2 = 0.52)				
Tanner 2 chronological age	−6.671	2.152	−0.300	.003
Tanner 2 upper extremities BMC	1.209	0.138	0.838	.000
Length of puberty	2.234	1.808	0.101	.220
Upper extremities peak BMD (R^2 = 0.58)				
Tanner 2 chronological age	−0.011	0.004	−0.245	.018
Tanner 2 upper extremities BMD	0.533	0.082	0.652	.000
Length of puberty	0.007	0.004	0.170	.075
Lower extremities peak BMC (R^2 = 0.59)				
Tanner 2 chronological age	−19.264	5.721	−0.300	.001
Tanner 2 lower extremities BMC	1.055	0.106	0.879	.000
Length of puberty	9.445	4.829	0.147	.054
Lower Extremities Peak BMD (R^2 = 0.58)				
Tanner 2 chronological age	−0.036	0.009	−0.357	.000
Tanner 2 lower extremities BMD	0.949	0.095	0.866	.000
Length of puberty	0.007	0.008	0.070	.363

B, Unstandardized coefficients; *σ*, standard error of B; *β*, standardized coefficients.

Table IV

Multiple regression models of DXA measures for 85 males with length of puberty, baseline chronological age, and baseline bone measure as independent variables

	B	σ	β	P
Whole body peak BMC (R^2 = 0.31)				
Tanner 2 age	−64.625	26.130	−0.270	.016
Tanner 2 whole body BMC	0.910	0.152	0.646	.000
Length of puberty	0.086	22.717	0.000	.997
Whole body peak BMD (R^2 = 0.57)				
Tanner 2 age	−0.025	0.006	−0.313	.000
Tanner 2 whole body BMD	1.015	0.099	0.804	.000
Length of puberty	0.005	0.006	0.057	.446
Spine peak BMC (R^2 = 0.40)				
Tanner 2 age	−1.591	0.782	−0.197	.045
Tanner 2 spine BMC	1.011	0.141	0.688	.000
Length of puberty	−0.132	0.717	−0.016	.854
Spine peak BMD (R^2 = 0.57)				
Tanner 2 age	−0.022	0.007	−0.232	.003
Tanner 2 spine BMD	0.900	0.088	0.776	.000
Length of puberty	−0.004	0.007	−0.040	.590
Upper extremities peak BMC (R^2 = 0.34)				
Tanner 2 age	−7.000	2.649	−0.278	.010
Tanner 2 upper extremities BMC	0.996	0.157	0.657	.000
Length of puberty	0.018	2.357	0.001	.994
Upper extremities peak BMD (R^2 = 0.45)				
Tanner 2 age	−0.015	0.004	−0.302	.001
Tanner 2 upper extremities BMD	0.728	0.090	0.695	.000
Length of puberty	0.000	0.004	0.007	.937
Lower extremities peak BMC (R^2 = 0.36)				
Tanner 2 age	−17.763	6.565	−0.282	.008
Tanner 2 lower extremities BMD	0.882	0.132	0.686	.000
Length of puberty	1.721	5.769	0.027	.766
Lower extremities peak BMD (R^2 = 0.56)				
Tanner 2 age	−0.038	0.008	−0.363	.000
Tanner 2 lower extremities BMD	1.015	0.100	0.809	.000
Length of puberty	0.003	0.008	0.032	.673

B, Unstandardized coefficients; *σ*, standard error of B; *β*, standardized coefficients.

Table V

Multiple regression models of DXA measures for 78 females with length of puberty, Tanner 2 chronological age, Tanner 2 height, and Tanner 2 bone measure as independent variables

	B	σ	β	P
Whole body peak BMC (R^2 = 0.61)				
Tanner 2 chronological age	−113.554	25.370	−0.451	.000
Tanner 2 whole body BMC	0.782	0.154	0.615	.000
Tanner 2 height	13.704	4.661	0.407	.004
Length of puberty	40.790	18.819	0.162	.034
Whole body peak BMD (R^2 = 0.60)				
Tanner 2 chronological age	−0.029	0.008	−0.357	.001
Tanner 2 whole body BMD	0.966	0.114	0.870	.000
Tanner 2 height	0.000	0.001	−0.004	.973
Length of puberty	0.009	0.006	0.116	.129
Spine peak BMC (R^2 = 0.52)				
Tanner 2 chronological age	−4.031	1.122	−0.400	.001
Tanner 2 spine BMC	0.889	0.180	0.581	.000
Tanner 2 height	0.461	0.191	0.342	.018
Length of puberty	1.682	0.834	0.167	.048
Spine peak BMD (R^2 = 0.69)				
Tanner 2 chronological age	−0.042	0.011	−0.347	.000
Tanner 2 spine BMD	0.993	0.094	0.856	.000
Tanner 2 height	0.001	0.002	0.058	.579
Length of puberty	0.008	0.008	0.069	.302
Upper extremities peak BMC (R^2 = 0.55)				
Tanner 2 chronological age	−8.879	2.421	−0.400	.001
Tanner 2 upper extremities BMC	1.032	0.166	0.715	.000
Tanner 2 height	0.733	0.391	0.247	.064
Length of puberty	2.466	1.782	0.111	.171
Upper extremities peak BMD (R^2 = 0.37)				
Tanner 2 chronological age	−0.009	0.006	−0.213	.099
Tanner 2 upper extremities BMD	0.551	0.094	0.675	.000
Tanner 2 height	0.000	0.001	−0.060	.680
Length of puberty	0.007	0.004	0.170	.077
Lower extremities peak BMC (R^2 = 0.63)				
Tanner 2 chronological age	−27.279	6.288	−0.425	.000
Tanner 2 lower extremities BMC	0.783	0.145	0.652	.000
Tanner 2 height	3.145	1.193	0.367	.010
Length of puberty	10.411	4.660	0.162	.029
Lower extremities peak BMD (R^2 = 0.58)				
Tanner 2 chronological age	−0.038	0.011	−0.378	.001
Tanner 2 lower extremities BMD	0.926	0.114	0.846	.000

	B	σ	β	*P*
Tanner 2 height	0.001	0.002	0.045	.722
Length of puberty	0.007	0.008	0.071	.362

B, Unstandardized coefficients; σ, standard error of B; β, standardized coefficients.

Table VI

Multiple regression models of DXA measures for 85 males with length of puberty, Tanner 2 chronological age, Tanner 2 height, and Tanner 2 bone measure as independent variables

	B	σ	B	P
Whole body peak BMC (R^2 = 0.37)				
Tanner 2 chronological age	−111.352	30.129	−0.465	.000
Tanner 2 whole body BMC	0.670	0.169	0.476	.000
Tanner 2 height	14.393	5.138	0.406	.006
Length of puberty	4.493	21.870	0.019	.838
Whole body peak BMD (R^2 = 0.57)				
Tanner 2 chronological age	−0.024	0.008	−0.307	.004
Tanner 2 whole body BMD	1.018	0.107	0.807	.000
Tanner 2 height	0.000	0.001	−0.009	.936
Length of puberty	0.005	0.006	0.057	.451
Spine peak BMC (R^2 = 0.41)				
Tanner 2 chronological age	−2.308	0.987	−0.285	.022
Tanner 2 spine BMC	0.943	0.151	0.642	.000
Tanner 2 height	0.186	0.157	0.156	.239
Length of puberty	−0.086	0.716	−0.010	.905
Spine peak BMD (R^2 = 0.57)				
Tanner 2 chronological age	−0.020	0.010	−0.211	.046
Tanner 2 spine BMD	0.905	0.090	0.781	.000
Tanner 2 height	0.000	0.002	−0.032	.764
Length of puberty	−0.004	0.007	−0.041	.584
Upper extremities peak BMC (R^2 = 0.37)				
Tanner 2 chronological age	−10.932	3.157	−0.435	.001
Tanner 2 upper extremities BMC	0.815	0.175	0.538	.000
Tanner 2 height	1.150	0.528	0.309	.032
Length of puberty	0.690	2.325	0.027	.767
Upper extremities peak BMD (R^2 = 0.46)				
Tanner 2 chronological age	−0.018	0.006	−0.345	.004
Tanner 2 upper extremities BMD	0.706	0.098	0.674	.000
Tanner 2 height	0.001	0.001	0.070	.582
Length of puberty	0.001	0.004	0.010	.904
Lower extremities peak BMC (R^2 = 0.42)				
Tanner 2 chronological age	−30.204	7.612	−0.479	.000
Tanner 2 lower extremities BMC	0.662	0.147	0.515	.000
Tanner 2 height	3.786	1.307	0.406	.005
Length of puberty	3.015	5.541	0.047	.588
Lower extremities peak BMD (R^2 = 0.56)				
Tanner 2 chronological age	−0.034	0.011	−0.322	.003
Tanner 2 lower extremities BMD	1.038	0.107	0.828	.000

	B	σ	B	*P*
Tanner 2 height	−0.001	0.002	−0.069	.536
Length of puberty	0.003	0.008	0.030	.688

B, Unstandardized coefficients; σ, standard error of B; β, standardized coefficients.