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Lower Bone Mineral Content in Hypertensive Compared with Normotensive Overweight Latino Children and Adolescents

Afrooz Afghani, PhD[a,b] and **Michael I. Goran, PhD**[a,c]
a*Department of Preventive Medicine, Keck School of Medicine, University of Southern California, Los Angeles, CA*

b*College of Health Sciences, Touro University International, Cypress, CA*

c*Department of Physiology & Biophysics, Keck School of Medicine, University of Southern California, Los Angeles, CA*

Abstract

Background—In adults, hypertension has been shown to be inversely correlated with bone mineral content (BMC); however, the association between blood pressure (BP) and BMC has not been studied in pediatrics.

Methods—Total body BMC of 187 overweight (mean BMI=28.7 kg/m^2) Latino children and adolescents (mean age=11.2 years) were measured using dual-energy x-ray absorptiometry. Seated systolic (SBP) and diastolic (DBP) blood pressure were measured using a standard mercury sphygmomanometer. Hypertension was defined by SBP or DBP above the 90th percentile for height, age, and gender.

Results—Partial correlations revealed an inverse association between SBP and BMC (*r*=- 0.24, p=0.02) in boys (n=105); results were non-significant (p=0.27) in girls (n=82). There were no significant correlations between DBP and BMC. When BMI and insulin sensitivity were adjusted for, hypertensive boys (n=21) had lower BMC (1435 versus 1636 g; p=0.03) than normotensive boys (n=84); similarly, hypertensive girls (n=25) had lower BMC (1438 versus 1618 g; p=0.02) than normotensive girls (n=57). In post-pubertal adolescents (Tanner stage 4-5; n= 48), inverse correlations were stronger (*r*=- 0.40, p=0.007); results were non-significant in pre-pubertal and pubertal children (Tanner stage 1-3; n=139, p=0.57). In post-pubertal girls (n=37), there were no significant correlations (p=0.14); inverse correlations in post-pubertal boys (n=11) became markedly stronger (*r*= -0.80, p=0.02).

Conclusion—SBP is inversely correlated with BMC in overweight adolescents; additionally, hypertensives have lower adjusted means of BMC than normotensives. These promising new findings suggest that hypertension may be a risk factor for osteopenia in overweight children and adolescents; this risk may be exacerbated in post-pubertal boys.

Address all correspondence to: Afrooz Afghani, PhD, MPH Associate Professor College of Health Sciences Touro University International 5665 Plaza Drive, Third Floor Cypress, CA 90630 Phone: (714) 226-9840 ext. 2009 Fax: (714) 226-9845 E-mail: aafghani@tourou.edu.

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Keywords

Pediatric; Latino; Bone Mineral Content; Blood Pressure; Osteopenia

INTRODUCTION

In 1980, McCarron et al. (1) published the first report of hypercalciuria in adult patients with hypertension. Since calcium is an important determinant of peak bone mass (2), it is logical to assume that urinary excretion of calcium due to hypertension would be detrimental to the growing skeleton of children and ultimately to adult bone mineral density (BMD). The link between hypertension and bone loss in adults has been previously studied but the existing evidence is inconclusive (3-5). Tsuda et al. (3) compared the BMD of 31 hypertensive Japanese women to 14 normotensives and reported inverse correlations between lumbar spine BMD and SBP. We (4) also reported significantly lower BMC in hypertensive compared with normotensive Hispanic women living in the United States. In contrast, in a study of bone mass and bone modeling markers in hypertensive postmenopausal Spanish women, Perez-Castrillon et al. (5) found no significant association between SBP or DBP and lumbar spine bone mass.

In normotensive children, urinary calcium loss as a function of blood pressure has been observed in the upper age distribution (6). It has also been reported that normotensive children of hypertensive adults have hypercalciuria (6-7). Although in hypertensive rats, metabolic studies (8) have shown that hypercalciuria and subsequent hyperparathyroidism lead to reduced growth and detectable deficits in BMC, such detriments have not been observed in human beings. To the best of our knowledge, the association between blood pressure and bone mass has not been studied in the pediatric population; a comparison of BMC in hypertensive versus normotensive children has not been reported either.

In this paper, we address the hypothesis that resting SBP and DBP will be inversely correlated with total body BMC in a large group of overweight Latino children and adolescents. In addition, examining whether hypertensive children and adolescents have lower BMC compared with normotensives is our second objective.

METHODS

Study Cohort & Subject Description

A cohort of overweight Latino children with a positive family history of type 2 diabetes was established with the intention to follow longitudinally in the University of Southern California SOLAR (Study of Latino Adolescents at Risk) diabetes project. In a previous published article (9) on this cohort, we examined the hypothesis that children with impaired glucose tolerance will have lower BMC and BMD than those with normal glucose tolerance. The current article is examining a different hypothesis on the association between BP and BMC and is comparing BMC in hypertensive children versus normotensive children.

187 children (105 boys, 82 girls) were recruited to the SOLAR project through clinics, health fairs, newspaper announcements, and word of mouth. The children were required to meet the following inclusion criteria: 1) age 8-14 years; 2) Body mass index (BMI) $\geq 85^{th}$ percentile for age and gender based on the CDC standards (10), and an initial telephone pre-screening; 3) Latino ancestry (all 4 grandparents Latino by self-report); and, 4) family history of type 2 diabetes in at least one parent, sibling, or grandparent. Children were of Mexican-American (71%), Central American (16%), or mixed Mexican-Central American (13%) heritage. Children were excluded if they had prior major illness including type 1 or type 2 diabetes, or took medications or had a condition known to influence body composition, blood pressure, or

bone mass (e.g. pregnancy, glucocorticoid therapy, hyper- or hypothyroidism). The SOLAR study was approved by the Institutional Review Board, Health Science Campus of the University of Southern California. Informed consent and assent were obtained from all parents and children, respectively.

It is noteworthy that based on inverse correlation coefficients reported in adult studies by Larijani et al. (11) between femoral neck BMD and SBP (r=-0.17) and by Tsuda et al. (3) between lumbar spine BMD and SBP (r=-0.31), we expected an effect size in the range of 0.17-0.31 which would require 64 to 213 participants. With a sample size of 187 children and observed r values of -0.16 (for SBP and BMC) and -0.22 (for SBP and BMD), we had 70 and 91 % power, respectively.

Protocol Design

Outpatient Screening Visit—Children arrived at the USC General Clinical Research Center (GCRC) at approximately 8:00 am after an overnight fast. Weight and height were measured followed by a physical examination conducted by a board certified pediatric endocrinologist. Physical examinations included Tanner staging in girls (breast stage) and in boys (pubic hair stage). Children in Tanner stages 1 or 2 were the "pre-pubertal" group; Tanner stage 3 children were defined as "pubertal"; adolescents in Tanner stages 4 or 5 were the "post-pubertal" category. A medical history was conducted including parental interview detailing family history of diabetes. Children who met the following screening criteria were invited back for further testing during an in-patient GCRC visit: a) BMI $\geq 85^{th}$ percentile for age and gender based on the CDC standards (10) based on height and weight measures at the GCRC; and, b) absence of type 1 or type 2 diabetes using the ADA guidelines (12). Note that for the purposes of this study we used the CDC definitions for weight status in children (i.e. at risk of overweight/obesity is a BMI age/gender percentile greater than 85^{th}). The 187 children who met the inclusion/exclusion criteria constituted the study sample.

Inpatient Visit—Of the 187 children completing the out-patient visit, all returned to the GCRC within 15 ± 10 (SD) days. Children were admitted to the GCRC in the early afternoon and then completed tests for bone mass and body composition using dual energy x-ray absorptiometry (DXA). Children were served dinner and an evening snack, with only water permitted after 8 pm. The following morning insulin sensitivity was determined from an intravenous glucose tolerance test.

Detailed Methodologies

Weight, Height and Anthropometry—Height (by a wall mounted stadiometer) and weight (by a balance beam medical scale) were recorded at each visit to the nearest 0.1 cm and 0.1 kg respectively, and the average of the two measurements was used for analysis. BMI and BMI percentiles for age and gender were determined based upon established CDC normative curves using EpiInfo 2000, Version 1.1.

BMC, BMD, Bone Area, & Body Composition—A whole body DXA scan was performed to determine whole body BMC (units: grams), BMD (units: grams/cm^2), bone area (units: cm^2), and whole body composition (fat mass, lean mass) using a Hologic QDR 4500W (Hologic Inc., Bedford, Massachusetts). The whole body scan requires the subject to be placed supine with the arms and legs positioned according to the manufacturer's specifications. Quality control was performed daily using a phantom. The precision error (coefficient of variation for repeated measurements) was ≤1%.

Resting Blood Pressure—Blood pressure measurements were obtained from each seated subject on two separate days using a standard mercury sphygmomanometer with an appropriate

size cuff. The right arm was used for all blood pressure measurements after the subject had rested quietly for 5 minutes. The same trained technician made all measurements. SBP was measured at the first appearance of a pulse sound (Korotkoff phase 1) and DBP at the disappearance of the pulse sound (Korotkoff phase 5). On each occasion, three readings of blood pressure were obtained, and the average was recorded (13). Hypertension was defined by blood pressure above the 90th percentile for height, age, and gender (14).

Oral Glucose Tolerance Test—A topical anesthetic (EMLA cream, AstraZeneca LP, Wilmington, DE) was applied to the antecubital area of one arm, and a flexible intravenous catheter was placed in an antecubital vein. Subjects ingested 1.75 grams of oral glucose solution per kilogram body weight (to maximum 75 grams) at "Time 0". Blood was sampled and assayed for glucose and insulin at times -5' ("Fasting") and 120' ("2-Hour") relative to glucose ingestion.

Frequently Sampled Intravenous Glucose Tolerance Test—At 6:30 am EMLA was applied, followed approximately one hour later by flexible intravenous catheter placement in bilateral antecubital veins. Two fasting blood samples were drawn at -15 and -5 min for determination of basal glucose and insulin. At time 0, glucose (25% dextrose, 0.3 g/kg body weight) was administered intravenously. Blood samples were collected at time points 2, 4, 8, 19, 22, 30, 40, 50, 70, 100, and 180 minutes for determination of glucose and insulin concentrations. Insulin (0.02 U/kg body weight; Humulin® R (REGULAR insulin for human injection; Eli Lilly, Indianapolis, USA) was injected intravenously at 20 min. Values for glucose and insulin were entered into the MINMOD MILLENIUM 2002 computer program (Version 5.16, Richard N. Bergman) for determination of insulin sensitivity (15). The MINMOD program calculates an overall index of whole body insulin sensitivity based on the kinetics of insulin and glucose during this 3-hour test.

Statistical Analysis—All analyses were performed using SPSS version 14.0 (SPSS Inc, Chicago, IL), with a type I error set at $p<0.05$. Descriptive statistics were performed; partial correlations were used to determine the associations of BMC, BMD, and BA with SBP, DBP, and insulin sensitivity; independent t-tests and general linear models were used to compare BMC, BMD, and BA in two different groups (e.g. boys and girls or hypertensives and normotensives). A power analysis software program (Statistical Solutions, nQuery Advisor Version 3: Saugus, MA) was used for the power analysis results presented in the methods.

RESULTS

Mean and standard deviation of participant characteristics are shown in Table 1. The youngest child was 8 years old; the oldest was 14. Mean age was 11.2 years. SBP had a range of 85 to 152 mmHg. DBP had a range of 48 to 99 mmHg. Mean of total body BMC was 1583.2 grams. Mean of total body BMD was 0.929 g/cm^2. Bone area had a mean of 1676.1 cm^2. Girls were significantly more mature than boys and had significantly lower DBP. There were no other significant differences between boys and girls.

Table 2 shows partial correlations between blood pressure and BMC, BMD, and BA. In the entire cohort, SBP was partially and inversely correlated with BMC (r=-0.16, p=0.03). There were no significant associations between DBP and BMC (p=0.36). In boys, SBP and BMC were partially correlated (r=-0.24, p=0.02); correlations were not significant (p=0.27) in girls. SBP and DBP were not significantly related with BMC in pre-pubertal and pubertal children (Tanner stage 1-3). In post-pubertal adolescents (Tanner stage 4-5), SBP was inversely correlated with BMC (r=-0.40, p=0.007). The partial correlations between DBP and BMC in post-pubertal adolescents were not significant. Sub-division of the post-pubertal group based on gender revealed no significant correlations in girls (n=37); inverse correlations in boys

(n=11) became stronger (*r*= -0.80, p=0.02). Sub-division of the pre-pubertal and pubertal group based on gender did not change the non-significant findings.

Table 3 shows partial correlations between anthropometric indices (weight, BMI, fat mass, lean mass), insulin sensitivity (SI), and BMC, BMD, BA, SBP and DBP. Weight and lean mass were more strongly correlated with BMC and SBP as compared to BMI and fat mass. There were no significant correlations between anthropometric indices and DBP. SI was positively correlated with BMC (*r*=0.19, p=0.01) in all subjects. There were no significant correlations between SI and BMC (p=0.07) in boys; in girls, SI was correlated with BA only (*r*=0.26, p=0.03). While the relationships between SI and BMC (p=0.07) were non-significant in pre- and pubertal children, these relationships were quite strong (*r*=0.46, p=0.002) in post-pubertal adolescents. In these adolescents, SI and SBP (*r*=-0.39, p=0.01) and DBP (*r*=-0.35, p=0.02) were inversely correlated.

Hypertensive individuals were compared with normotensives using general linear models and results are presented in table 4. When BMI and SI were adjusted for, hypertensive boys (n=21) had significantly lower BMC (1435 versus 1636 g; p=0.03) compared with normotensive boys (n=84); hypertensive girls (n=25) also had significantly lower BMC (1438 versus 1618 g; p=0.02) compared with normotensive girls (n=57). In the pre- and pubertal group, hypertensives (n=36) had significantly lower BMC (1319 versus 1458 g; p=0.007) compared with normotensives (n=103). When this analysis was repeated in pre-pubertal children (Tanner stage 1-2) with the exclusion of pubertal (Tanner stage 3) children, results did not materially change; hypertensives (n=33) had significantly lower BMC (1307 versus 1433 g; p=0.02) compared with normotensives (n=90). Additionally, in the post-pubertal group, hypertensives (n=10) had significantly lower BMC (1774 versus 2104 g; p=0.01) compared with normotensives (n=38).

DISCUSSION

The results of this study suggest that there is a strong inverse association between systolic BP and BMC in adolescent boys. In addition, regardless of gender, hypertensives had significantly lower BMC compared with normotensives. There are several explanations for the inverse relationship between SBP and BMC and we believe that they include the complex interactions between blood pressure, insulin resistance, sex hormones, and bone mass.

The inverse associations found between SBP and BMC in post-pubertal adolescents (table 2) may be explained by the observed differences in the relationship between insulin sensitivity and BMC of this population (table 3). In our previous study (16) of the metabolic profile of this sample, we found that there was a tendency for post-pubertal adolescents to have greater number of features of the metabolic syndrome than pre-pubertal and pubertal children; girls also tended to have a lower prevalence of features of the metabolic syndrome compared with boys. We have reported here (table 3) and in the previous article (16) that insulin sensitivity is correlated negatively with SBP and DBP; insulin resistance may alter blood pressure by its effects on the sympathetic nervous system and by directly influencing renal sodium reabsorption (17); high blood pressure may manifest due to lack of resistance to these secondary effects of insulin (18). Other underlying mechanisms related to high BP and abnormalities in calcium metabolism which may ultimately influence peak bone mass include evidence of secondary increase in parathyroid gland activity (1,19), increase in urinary cyclic AMP (1, 20), tendency to low serum ionized calcium (1,20-21), raised vitamin D levels (22), and increased intestinal calcium absorption (23).

Puberty is a critical time of flux and it raises a complex set of issues. The fact that we observed dramatic inverse correlations between SBP and BMC in post-pubertal boys but no such relationships in pre- and pubertal boys or in girls (table 2), may be explained by the profound

rise in testosterone during puberty in boys and its atherogenic effects (24). In adolescent boys, previous researchers have found a positive relationship between testosterone and SBP (25) and an inverse relationship between testosterone and HDL cholesterol (24). Morrison et al. (24) conclude that changes in sex steroid hormones during puberty has significant effects on changes in lipid parameters with increasing testosterone having atherogenic effects and increasing estradiol having antiatherogenic effects. The fact that the relationship between SBP and BMC was not significant in post-pubertal girls (table 2) may also be explained by the antihypertensive (26), antiatherosclerotic (24,26), and osteogenic (27) properties of estrogen as well as the lack of the testosterone surge that is seen in boys. It is noteworthy that when we compared body size and composition in pre-versus post-pubertal boys (data not shown), we observed that although post-pubertal boys weighed more (81 kg versus 65 kg, p=0.006), they had significantly lower percent fat (29% versus 39%, p=0.000) than pre- and pubertal boys. These findings (from DXA) are consistent with those of Leccia et al. (from skinfold measurements) who observed reductions in percent body fat in 98 Italian pubertal boys (28). Since obesity is known to increase estradiol in males by converting testosterone to estradiol and lowering testosterone (29), losing fat may have the opposite effect and may therefore be another plausible reason for why the adverse relationship between SBP and BMC is much more profound in post-pubertal boys. These tentative conclusions regarding the role of steroid hormones and their interaction with body composition, BP and BMC should be interpreted with caution until they are explored further in our future longitudinal investigations of this population and validated by other researchers.

This study, although the first to examine the relationship between BP and BMC in pediatrics, has a number of limitations that need to be addressed. First, this study was restricted to overweight Latino children of predominantly Mexican descent and is not necessarily generalizeable to children of other ethnic groups. Secondly, because body size is a powerful determinant of BMC and BP (30,14), the fact that our population was overweight may also influence generalizeability of our findings. However, in order to rule out the confounders of the relationship between BP and BMC, all statistical analyses of this study adjusted for body weight and height or BMI. In addition, the cross-sectional nature of this study makes it difficult to prove causality. Nevertheless, we were able to appropriately and accurately test the specific hypotheses of the study. Further, the strength of the association (ie. correlation) between BMC and SBP in the entire cohort, although significant, was not strong (r<0.30). However, sub-group correlations based on puberty and gender were as high as 0.80. Moreover, results from the general linear models comparing BMC in hypertensive versus normotensive children complemented all correlations and were significantly lower in hypertensives in every single sub-group analysis. In addition, the strength of the correlations observed in the entire cohort are similar to those seen in adults of other studies; therefore, the physiological mechanisms explaining hypertensive bone loss in adults could perhaps also explain the inverse relationships observed in adolescents and would therefore enhance the biological plausibility of our findings.

In conclusion, SBP is inversely correlated with BMC in overweight adolescents; additionally, hypertensives have lower adjusted means of BMC than normotensives. These promising new findings suggest that hypertension may be a risk factor for osteopenia in overweight children and adolescents; this risk may be exacerbated in post-pubertal boys.

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Table 1

CHARACTERISTICS OF PARTICIPANTS (MEAN ± SD)

	All Subjects (n=187)	Boys (n=105)	Girls (n=82)
Age (years)	11.2 ± 1.7	11.3 ± 1.5	11.1 ± 1.8
Tanner Stage	2.3 ± 1.4	1.7 ± 1.1	2.9 ± 1.4*
Tanner 1	79 (42%)	59 (56%)	20 (25%)
Tanner 2	44 (23%)	29 (27%)	15 (18%)
Tanner 3	16 (9%)	6 (6%)	10 (12%)
Tanner 4	30 (16%)	6 (6%)	24 (29%)
Tanner 5	18 (10%)	5 (5%)	13 (16%)
Weight (kg)	66.3 ± 19.7	66.5 ± 19.1	66.0 ± 20.5
Height (cm)	150.2 ± 11.4	151.1 ± 11.1	149.2 ± 11.8
BMI (kg/m^2)	28.7 ± 5.5	28.6 ± 5.5	28.9 ± 5.5
BMI Percentile (%)	96.0 ± 2.3	95.9 ± 2.5	96.0 ± 2.2
Fat Mass (kg)	25.8 ± 10.4	25.2 ± 10.4	26.5 ± 10.4
Lean Mass (kg)	38.0 ± 10.2	38.8 ± 10.0	37.0 ± 10.4
SI [$x10^{-4}$ min^{-1}/(μ U/ml)]	2.1 ± 1.5	2.2 ± 1.5	1.9 ± 1.5
Systolic Blood Pressure (mmHg)	110 ± 10.7	110 ± 10.1	110 ± 11.5
Diastolic Blood Pressure (mmHg)	64 ± 7.3	64 ± 7.6	62 ± 6.8†
Total Body BMC (g)	1583.2 ± 440.3	1598.6 ± 429.4	1563.6 ± 455.7
Total Body BMD (g/cm^2)	0.929 ± 0.1	0.925 ± 0.1	0.934 ± 0.1
Total Body BA (cm^2)	1676.1 ± 297.7	1703.1 ± 289.7	1641.6 ± 306.1

BMI: Body Mass Index

SI: Insulin Sensitivity

BMC: Bone Mineral Content

BMD: Bone Mineral Density

BA: Bone Area

* significantly greater than boys ($p<0.0001$)

† significantly lower than boys ($p<0.05$)

Table 2

PARTIAL CORRELATIONS BETWEEN BLOOD PRESSURE AND BMC, BMD, AND BA

	Variables		
SBP	**BMC**	**BMD**	**BA**
All Subjects (n=187) ‡	−0.16†	−0.22*	−0.06
Boys (n=105)§	−0.24†	−0.25†	−0.15
Girls (n=82)§	−0.13	−0.18	0.01
Pre- and Pubertal (n=139) (Tanner Stage 1-3) ‖	−0.04	−0.12	0.09
Boys (n=94)¶	−0.06	−0.10	0.05
Girls (n=45)¶	−0.02	−0.11	0.13
Post-Pubertal (n=48) (Tanner Stage 4-5) //	−0.40†	−0.39†	−0.36†
Boys (n=11)¶	−0.80†	−0.72†	−0.92*
Girls (n=37)¶	−0.26	−0.27	−0.18
DBP			
All Subjects (n=187) ‡	−0.07	−0.10	−0.02
Boys (n=105)§	−0.15	−0.18	−0.13
Girls (n=82)§	0.12	0.07	0.21
Pre- and Pubertal (n=139) (Tanner Stage 1-3) //	−0.05	−0.11	0.05
Post-Pubertal (n=48) (Tanner Stage 4-5) //	−0.15	−0.10	−0.21

BMC: Bone Mineral Content

BMD: Bone Mineral Density

BA: Bone Area

SBP: Systolic Blood Pressure

DBP: Diastolic Blood Pressure

* p<0.005

† p<0.05

‡ Adjusted for gender, age, weight, height, Tanner stage

§ Adjusted for age, weight, height, Tanner stage

// Adjusted for gender, age, weight, height

¶ Adjusted for age, weight, height

Table 3

PARTIAL CORRELATIONS BETWEEN ANTHROPOMETRIC INDICES AND BMC, BMD, BA, SBP, DBP

Variables	BMC	BMD	BA	SBP	DBP
WEIGHT					
All Subjects*	0.72	0.42	0.81	0.24	0.08
Boys	0.72	0.42	0.81	0.35	0.02
Girls	0.74	0.42	0.82	0.14	0.17
Pre- and Pubertal	0.78	0.45	0.84	0.28	0.06
Post-Pubertal	0.62	0.41	0.71	0.21	0.13
BMI					
All Subjects*	0.54	0.31	0.62	0.25	0.09
Boys	0.55	0.32	0.63	0.36	0.03
Girls	0.54	0.28	0.62	0.19	0.21
Pre- and Pubertal	0.60	0.34	0.65	0.29	0.08
Post-Pubertal	0.35	0.19	0.44	0.26	0.16
FAT MASS					
All Subjects*	0.55	0.29	0.66	0.23	0.09
Boys	0.56	0.29	0.67	0.37	0.06
Girls	0.59	0.30	0.69	0.16	0.15
Pre- and Pubertal	0.65	0.33	0.73	0.29	0.08
Post-Pubertal	0.35	0.19	0.45	0.29	0.16
LEAN MASS					
All Subjects*	0.81	0.50	0.88	0.21	0.04
Boys	0.81	0.52	0.88	0.28	−0.05
Girls	0.81	0.48	0.88	0.10	0.18
Pre- and Pubertal	0.85	0.53	0.90	0.23	0.01
Post-Pubertal	0.78	0.56	0.87	0.07	0.08
SI					
All Subjects†	0.19	0.17	0.15	−0.14	−0.01
Boys	0.18	0.20	0.08	−0.16	0.03
Girls	0.20	0.12	0.26	−0.15	−0.04
Pre- and Pubertal	0.16	0.13	0.10	−0.07	0.06
Post-Pubertal	0.46	0.42	0.45	−0.39	−0.35

Bold: significant at $p<0.05$

BMC: Bone Mineral Content

BMD: Bone Mineral Density

BA: Bone Area

SBP: Systolic Blood Pressure

DBP: Diastolic Blood Pressure

BMI: Body Mass Index

SI: Insulin Sensitivity

*Adjusted forgender, age, Tanner stage

†Additionally adjusted for fat mass, lean mass, height

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Table 4

BMC, BMD, BA MEANS OF HYPERTENSIVES VERSUS NORMOTENSIVES (ADJUSTING FOR BMI AND SI)

	BMC (g)	BMD (g/cm^2)	BA (cm^2)
BOYS			
Hypertensives n=21	1435*	0.888*	1594*
Normotensives n=84	1636	0.936	1724
GIRLS			
Hypertensives n=25	1438*	0.895*	1576*
Normotensives n=57	1618	0.951	1669
PRE- AND PUBERTAL			
Hypertensives n=36	1319*	0.866*	1501*
Normotensives n=103	1458	0.900	1605
POST-PUBERTAL			
Hypertensives n=10	1774*	0.967*	1824*
Normotensives n=38	2104	1.059	1976

BMC: Bone Mineral Content

BMD: Bone Mineral Density

BA: Bone Area

BMI: Body Mass Index

SI: Insulin Sensitivity

* significantly lower than normotensives ($p<0.05$)