Effect of Calcium Intake, Tennis Playing, and Body Composition on Bone-Mineral Density of Brazilian Male Adolescents

Claudia Ridel Juzwiak, Olga Maria Silverio Amancio, Maria Sylvia Souza Vitalle, Vera Lúcia Szejnfeld, and Marcelo Medeiros Pinheiro

In this prospective, cross-sectional study male adolescent tennis players (44) and nonathletic controls (32) were evaluated to determine the effects of physical activity, dietary nutrient intakes, sexual maturation, and body composition on bone-mineral density (BMD). Dietary nutrient intakes and physical activity expenditure were estimated by 4-d diaries. Total body composition, bone-mineral content (BMC), and BMD (L1–L4, femur, and nondominant forearm) were assessed by dual-energy X-ray absorptiometry. Tennis players had significantly greater lean body mass (mean [$SEM$] 50.6 [1.6] kg vs. 45.1 [1.7] kg, $p = .022$), trochanter BMD (1.0 [0.02] g/cm$^2$ vs. 0.9 [0.03] g/cm$^2$, $p = .032$), and dominant forearm BMC (173.7 [7.4] g vs. 146.5 [9.3] g) but lower BMD in the nondominant forearm (0.7 [0.02] g/cm$^2$ vs. 0.8 [0.03] g/cm$^2$, $p = .028$). Daily average calcium intake was below the recommendation in both groups. No correlation was found between BMD and calcium intake and exercise. Lean body mass was the best predictor of BMD and BMC for both tennis players and controls ($R^2 = .825$, .628, and .693 for L1–L4, total femur, and nondominant forearm, respectively). Based on these results the authors conclude that lean body mass is the best predictor of BMD and BMC for both tennis players and others. Tennis exerts a site-specific effect, and training should focus on ways minimize this effect. Although calcium intake showed no effect on BMD, nutrition education for young athletes should focus on promoting a balanced diet, providing energy and nutrients in adequate amounts.

Keywords: adolescence, lean body mass, exercise, dietary intake

Several studies have indicated that the peripubertal and pubertal periods represent “windows of opportunity” for bone-mass gain (MacKelvie, Khan, & McKay, 2002). It is estimated that an increase of 3–5% in bone-mineral density (BMD) might result in 30–50% reduction of fracture risk (Weaver, 2000; Wosje & Specker, 2000). Thus, bone-mass acquisition during the growth period and the reduction of bone loss related to aging are the main strategies for preventing osteoporosis.

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Osteoporosis is an important worldwide public health problem with rising incidence, prevalence, and socioeconomic impact because of the increase in life expectancy for both men and women (Silveira et al., 2005). In Brazil, although there are no consistent and representative data of national range, projections are similar to those of other nations (World Health Organization/Food and Agriculture Organization, 2003).

Nutrition and physical activity are the main environmental factors affecting bone acquisition. Not all studies have shown a correlation between dietary calcium intake and measures of bone health, however, and up to the moment, it is unclear how much each of these factors contributes to the skeletal growth (Lanou, Berkow, & Barnard, 2005; Vicente-Rodriguez, 2006; Weaver, 2000).

Taking these facts into consideration and selecting tennis, a unilateral model of physical loading, the aim of this study was to evaluate the effect of dietary calcium, exercise, and body composition on BMD in male adolescents.

**Methods**

Forty-four male adolescent tennis players (10–19 years old) from competition teams of clubs and fitness centers enrolled in this cross-sectional study. The study was approved by the ethics committee of the Federal University of São Paulo, and written consent from parents or guardians was obtained. All athletes were registered with the Brazilian Tennis Federation. In addition, these athletes had been training for at least 5 hr/week for the preceding 12 months and were participating in regional or national tournaments at least once a month. They presented no chronic or acute diseases and did not use any medication or nutritional supplements that could affect calcium or bone metabolism.

The control group consisted of 32 male adolescents matched by age and classified by the International Physical Activity Questionnaire in its validated version in Portuguese (Guedes, Lopes, & Guedes, 2005) as “insufficiently active.”

**Anthropometric and Maturational Assessment**

Body mass and height were measured according to recommended techniques (Jelliffe, 1966). Body-mass index (BMI, kg/m²) was classified according to the percentiles proposed by the National Center of Health Statistics/Centers for Disease Control and Prevention as underweight (P < 5), normal weight (P5–<P85), risk for overweight (P85–P95), and overweight (P > 95; Kuczmarski et al., 2000).

The autoevaluation validated method was employed to assess maturational stage (Marshall & Tanner, 1970; Matsudo & Matsudo, 1994). Gonadal development was considered in classifying maturational stage.

**Evaluation of Body Composition and BMD**

Total body composition (fat mass [FM, kg], lean body mass [LBM, kg], bone-mineral content [BMC g]) and BMD (g/cm²) at the anteroposterior lumbar spine (L1–L4), right femur (total, femoral neck, and trochanter), and distal third of the nondominant radius (radius33%) were measured using dual-energy X-ray absorptiometry (DXA; GE-Lunar Radiation Corp., Model DPX MD Plus, Madison, WI).
Body FM was classified according to the criterion established for adolescents proposed by Lohman and adapted by Houtkooper (1996) and for the tennis players, the values (6–17%) suggested by Boileau and Horswill (2000).

The standard technical procedure was adopted for positioning the adolescents for the exam. The medium scan mode and the analyses were done using Lunar version 6.7. Quality assurance was performed daily according to the manufacturer’s instructions. The reference data of the manufacturer (pediatric software) were assumed for this population. The in vivo precision error of the DXA, expressed as the coefficient of variation, was 3% for the femur and total body and 2% for the lumbar spine and forearm.

Ten athletes did not attend the DXA evaluation for personal reasons.

Evaluation of Food Intake and Energy Expenditure

Forms were provided, and participants completed 4 nonconsecutive days of a dietary and physical activity diary (3 weekdays and 1 weekend day; Bouchard et al., 1983; Willet, 1998).

Energy and nutrient intakes were analyzed by Virtual Nutri software (version 1.0; University of São Paulo, Brazil: Phillipi, Szarfac, & Latterza, 1996). Dietary analyses included energy (kcal), protein (g · kg body mass⁻¹ · day and g/1,000 kcal), calcium (mg/day, g/1,000 kcal), phosphorus (mg), and the ratios of calcium to protein (Ca:Ptn) and calcium to phosphorus (Ca:P). Intakes were compared with the dietary reference intake (Institute of Medicine, Food and Nutrition Board [IOM], 1997, 2002).

Physical activities were recorded in 15-min intervals and quantified in metabolic equivalents on a scale from 1 to 9, sleeping being categorized as 1 and vigorous activities as 9. Total estimated energy expenditure in kcal/day was then calculated for each athlete using the metabolic equivalents of the recorded activities (Bouchard et al., 1983).

Statistical Analysis

Means (SEM) are given as descriptive statistics. Differences between groups were established using Student’s t test. For categorical variables Fisher’s exact test was used, and Pearson’s (r) linear correlation coefficient for the correlations between variables of bone mass, nutritional data, and body composition. For the correlation between bone mass and sexual maturation, Spearman’s correlation was used.

Three multivariate linear-regression models were established. The dependent variables were L1–L4, total femur, and radius33% BMD. The independent variables were tennis playing, age, body weight, sexual maturation, LBM, FM, calcium (mg/day), phosphorus (mg/day), and protein (g/1,000 kcal). The statistical package SPSS for Windows, version 11.0, was used for the analysis. We adopted an alpha level of p < .05 for all analyses.
Results

There were no significant differences between groups for age, body mass, height, or BMI. Nevertheless, in relation to body composition, tennis players presented lower mean FM ($p = .009$) and higher mean LBM ($p = .022$; Table 1).

The BMI was classified as normal for 88.6% of the tennis players and 87.5% of the controls, and 5 tennis players (11.4%) and 2 controls (6.4%) presented risk for overweight. Two controls were identified as being overweight (6.4%). Most controls (56%) presented FM percentage classified as moderately high, high, or very high, whereas most tennis players (56%) presented fat percentage classified as optimal. Of the athletes with risk for overweight, 2 were not evaluated (10 and 13 years old) by DXA, and the other 3 were classified as having optimal (14%), high (25%), and very high (35%) body-fat percentages. In comparison with the values suggested for male adolescent tennis players, 70.5% of the athletes were within this range.

The distribution according to sexual maturation indicated that most of the adolescents, tennis players and controls, respectively, were in G4 (43.2% and 34.4%) and G5 (29.5% and 25%).

The mean BMD of the nondominant radius was significantly lower in tennis players than in controls ($p = .028$; Table 2).

Athletes presented a higher energy expenditure than controls ($p < .001$); however, mean energy intake was not different ($p = .064$). Protein intake was above recommended values in both groups, without significant difference between them ($p = .157$). Twenty-three percent of controls and 20.5% of tennis players had intakes $\geq 2.5 \text{ g} \cdot \text{kg}^{-1} \cdot \text{day}^{-1}$. Phosphorus intake was equal to or above the recommended daily values ($\geq 85\%$ probability that the intake was adequate, using the estimated average requirement of 1,055 mg for 9–18 years of age) for 43.2% of the tennis players and for 31.3% of the controls. Calcium intake was below the adequate intake of 1,300 mg/day (IOM, 1997), with the exception of 6 tennis

<table>
<thead>
<tr>
<th>Table 1</th>
<th>Characteristics of the Population, $M (SEM)$, Range</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Tennis players $n = 44$</td>
</tr>
<tr>
<td>Age (years)</td>
<td>15 (0.3), 10.3–18.0</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>58.5 (1.8), 36.5–93.0</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.68 (0.02), 1.44–1.89</td>
</tr>
<tr>
<td>Body-mass index (kg/m²)</td>
<td>20.5 (0.3), 16.2–27.2</td>
</tr>
<tr>
<td>Fat percentage</td>
<td>15 (0.01)$\dagger$, 7.0–35.0</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>9.2 (0.9)$\dagger$, 3.6–32.4</td>
</tr>
<tr>
<td>Lean body mass (kg)</td>
<td>50.6 (1.6)$\dagger$, 31.1–71.0</td>
</tr>
</tbody>
</table>

Note. $p$ is the descriptive level of Student’s $t$ test.

$\dagger$34 tennis players.
players (13.6%) and 2 controls (6.3%), without significant difference between groups ($p = .266$; Table 3).

Significant correlations were found between BMD and body mass, and BMD and LBM, in all evaluated bone sites for both groups and are shown for L1–L4, total femur, and nondominant radius33% in Table 4. With the exception of proteins, no correlations between BMD and nutritional variables were identified. In tennis players, protein intake (g/kg) presented an inverse significant correlation with BMD at all evaluated sites; however, this correlation was significant for the controls only in L1–L4 ($p = .048$). No correlation was found between BMD and training time (hr/week), type of training (technical/court or physical conditioning, hr/week), or years of training.

Sexual maturation was related to BMD at all evaluated sites. Figure 1 shows the results in L1–L4 (tennis players $r = .725$, $p < .001$; controls $r = .494$, $p < .004$). For other evaluated sites the results were similar.

In the regression model (Table 5) for L1–L4, the only statistically significant variable was LBM ($p < .001$). When LBM was excluded from the model, age, physical activity, and sexual maturation showed significance. Notwithstanding, the quality of the adjustment worsened ($R^2 = .825–.618$). In this new model other variables were significant: age ($p = .001$), exercise practice (tennis playing; $p = .057$), and sexual maturation (we excluded Stages 1 and 2 and included Stages 3, 4 [$p = .035$] and 5 [$p = .052$] separately). Stage 3 was not significant ($p = .967$). Even in this second model, the micronutrient variables did not show any significance.

In the total femur and radius33% models, LBM was also statistically significant ($R^2 = .628$, $p < .001$; $R^2 = .693$, $p = .001$, respectively), and age was also statistically significant ($p = .006$) for radius33%.

All models were recalculated using BMC as the dependent variable, and the results were similar.

### Table 2 Bone-Mineral Density and Bone-Mineral Content at Different Skeletal Sites, $M (SEM)$, Range

<table>
<thead>
<tr>
<th></th>
<th>Tennis players ($n = 44$)</th>
<th>Controls ($n = 32$)</th>
<th>$p$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Bone-mineral density, g/cm³</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>L1–L4</td>
<td>1.1 (0.03), 0.7–1.4</td>
<td>1.0 (0.04), 0.6–1.4</td>
<td>.198</td>
</tr>
<tr>
<td>femoral neck</td>
<td>1.1 (0.03), 0.9–1.6</td>
<td>1.1 (0.03), 0.7–1.3</td>
<td>.068</td>
</tr>
<tr>
<td>trochanter</td>
<td>1.0 (0.02), 0.7–1.2</td>
<td>0.9 (0.03), 0.1–1.2</td>
<td>.032</td>
</tr>
<tr>
<td>total femur</td>
<td>1.1 (0.02), 0.8–1.4</td>
<td>1.1 (0.03), 0.8–1.4</td>
<td>.118</td>
</tr>
<tr>
<td>nondominant radius33%</td>
<td>0.7 (0.02), 0.3–1.0</td>
<td>0.8 (0.03), 0.5–1.0</td>
<td>.028</td>
</tr>
<tr>
<td>total body</td>
<td>1.1 (0.02)a, 0.9–1.4</td>
<td>1.1 (0.02), 0.8–1.3</td>
<td>.602</td>
</tr>
<tr>
<td>Bone-mineral content, g</td>
<td></td>
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<td></td>
</tr>
<tr>
<td>dominant arm</td>
<td>173.7 (7.4)a, 93–292</td>
<td>146.5 (9.1), 56–237</td>
<td>.023</td>
</tr>
<tr>
<td>nondominant arm</td>
<td>143.0 (7.5)a, 78–247</td>
<td>140.4 (9.3), 49–233</td>
<td>.827</td>
</tr>
</tbody>
</table>

*Note. $p$ is the descriptive level of Student’s $t$ test.

a34 tennis players.
Tennis players had significant higher LBM content and lower FM body composition than controls. A longitudinal study evaluating 387 adolescents reported that in boys age 12–17 years, the yearly higher BMI resulted mainly from the increase in LBM (Maynard et al., 2001). The significant difference in body composition found between tennis players and controls might be a result of training (Boileau & Horswill, 2000; Malina, 2000), which might have optimized the pubertal body-composition distribution; however, a natural selection of adolescents with morphological characteristics that predispose them to better results might reflect our findings (Malina).

The relationships between BMC and BMD and the sexual maturational stage observed in this study are similar to others reported in the literature (Bradney et al., 1998; MacKelvie et al., 2002; Weaver, 2000; Wosje & Specker, 2000). A Brazilian study showed that from 14 to 15 years of age and between maturation levels of G4 and G5, adolescents experienced significant increase in vertebral and femoral bone mass (Silva, Goldberg, Teixeira, & Dalmas, 2004). Most of our adolescents, both tennis players and controls, were in G4 and G5, which means at their pubertal and bone mass peak.

Several studies have provided evidence that physical activity during adolescence positively affects bone-mass acquisition (Ginty et al., 2005; Vicente-Rodriguez, 2006; Vicente-Rodriguez, Ara, Perez-Gomez, Dorado, & Calbet, 2005; Weaver, 2000). Three hours of weekly high-impact or weight-bearing exercise seems to be sufficient for a positive osteogenic effect (Vicente-Rodriguez). As in other studies (Calbet, Moysi, & Rodriguez, 1998; Ducher, Jaffré, Arlettaz, Benhamou, & Courteix, 2005; Haapasalo et al., 1998; Kannis et al., 1995), our tennis

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<tr>
<td></td>
<td>( n = 44 )</td>
<td>( n = 32 )</td>
</tr>
<tr>
<td>Energy expenditure, kcal</td>
<td>2,610 (86), 1,845–4,119</td>
<td>2,122 (97), 1,198–3,259</td>
</tr>
<tr>
<td>Energy intake, kcal</td>
<td>2,774 (110), 1,476–4,490</td>
<td>2,482 (99), 1,475–3,851</td>
</tr>
<tr>
<td>Proteins, g/kg body mass</td>
<td>2.0 (0.1), 0.9–3.7</td>
<td>1.8 (0.1), 1.0–3.6</td>
</tr>
<tr>
<td>Protein density, g/1,000 kcal</td>
<td>42.3 (1.2), 25.3–60.6</td>
<td>41.4 (1.6), 28.1–65.1</td>
</tr>
<tr>
<td>Phosphorus, mg</td>
<td>1,306 (76.4), 454–2,929</td>
<td>1,119 (69), 372–1,890</td>
</tr>
<tr>
<td>Calcium, mg</td>
<td>926 (63), 353–1,083</td>
<td>830 (52), 257–1,070</td>
</tr>
<tr>
<td>Calcium:protein, mg/g</td>
<td>8.1 (0.4), 3.4–16.2</td>
<td>8.5 (0.6), 3.4–17.7</td>
</tr>
<tr>
<td>Calcium:phosphorus, mg:mg</td>
<td>0.8 (0.1), 0.3–2.1</td>
<td>0.8 (0.1), 0.4–1.7</td>
</tr>
<tr>
<td>Calcium density, mg/1,000 kcal</td>
<td>330 (14), 144–600</td>
<td>339 (20), 123–604</td>
</tr>
</tbody>
</table>

*Note.* \( p \) is the descriptive level of Student’s \( t \) test.

### Discussion


tennis

Tennis players had significant higher LBM content and lower FM body composition than controls. A longitudinal study evaluating 387 adolescents reported that in boys age 12–17 years, the yearly higher BMI resulted mainly from the increase in LBM (Maynard et al., 2001). The significant difference in body composition found between tennis players and controls might be a result of training (Boileau & Horswill, 2000; Malina, 2000), which might have optimized the pubertal body-composition distribution; however, a natural selection of adolescents with morphological characteristics that predispose them to better results might reflect our findings (Malina).

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Table 4  Correlation (r) Between Body Composition, Nutritional Data and Lumbar, Femur, and Forearm Bone-Mineral Density (BMD)

<table>
<thead>
<tr>
<th></th>
<th>L1–L4 BMD, g/m²</th>
<th>Total femur BMD, g/m²</th>
<th>Radius33% BMD, g/m²</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Tennis players n = 44</td>
<td>Controls n = 32</td>
<td>Tennis players n = 44</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>.821 (&lt;.001)</td>
<td>.842 (&lt;.001)</td>
<td>.637 (&lt;.001)</td>
</tr>
<tr>
<td>Lean body mass (kg)</td>
<td>.849 (&lt;.001)</td>
<td>.929 (&lt;.001)</td>
<td>.605 (&lt;.001)</td>
</tr>
<tr>
<td>Fat mass (kg)</td>
<td>.285 (.103)</td>
<td>.341 (.056)</td>
<td>.241 (.170)</td>
</tr>
<tr>
<td>Calcium (mg)</td>
<td>.111 (.475)</td>
<td>−.072 (.697)</td>
<td>0.0 (.998)</td>
</tr>
<tr>
<td>Protein (g/kg)</td>
<td>−.365 (.015)</td>
<td>−.352 (.048)</td>
<td>−.365 (.015)</td>
</tr>
<tr>
<td>Calcium:protein, mg:g</td>
<td>−.025 (.226)</td>
<td>−.242 (.182)</td>
<td>−.070 (.651)</td>
</tr>
<tr>
<td>Calcium:phosphorus, g:mg</td>
<td>−.026 (.868)</td>
<td>−.158 (.387)</td>
<td>−.075 (.630)</td>
</tr>
</tbody>
</table>
Figure 1 — Box plot of sexual maturation and L1-L4 bone-mineral density (g/cm²) of tennis players and control.
players presented dominant-forearm BMC and trochanter BMD significantly greater than those of controls. Correlations between groups were very similar, however, suggesting that they were not affected by exercise practice. Thus, we believe that training presented a more important effect on LBM and FM than on bone-mass acquisition in adolescent male elite tennis players.

Seeman et al. (1996) suggested that lean and bone mass are genetically determined. Nonetheless, the use of a sports model with “unilateral” characteristics, such as tennis, allows an analysis that “excludes” the action of genetic, hormonal, and nutritional factors on forearm BMD. The effect and response to the tennis load on the dominant arm was widely demonstrated, with increments on the dominant arm varying from 4% to 20% (Calbet et al., 1998; Ducher et al., 2005; Haapasalo et al., 1998). Tennis players presented a 20% mean difference in BMC between dominant and nondominant forearms, and the control group only 5%. The greater mean BMD observed in the tennis players’ trochanters might also be a result of the movements executed in the sport. Some authors have encountered greater BMD in other bone sites in tennis players. For example, Calbet et al. found greater BMD in the femoral neck and L2–L4 in tennis players than in a sedentary group.

An interesting finding was that tennis players presented lower mean BMD in the radius33% of the nondominant arm than controls but had greater BMC. Ballard and Wallace (2004) also found lower BMD in the nondominant forearm of 21 tennis players (mean age of 20.9 years) than in sedentary controls. Contrary to that, other authors did not observe this difference (Calbet et al., 1998; Haapasalo et al., 1998). The explanation for this finding is not clear. There might be a compensatory mechanism caused by the lower mechanical stress on the nondominant forearm, which might function as an immobilized or resting arm and thus acquire lower bone mass or present greater bone loss. Notwithstanding,

### Table 5 Final Multivariate-Regression Model of Lumbar, Total Femur, and Radius33% Bone-Mineral Density After Adjustments for Age, Body Mass, Physical Activity, Sexual Maturation, Fat Mass, Phosphorus, Calcium, and Protein (mg/100 kcal) Intake in Tennis Players and Controls

<table>
<thead>
<tr>
<th>Model</th>
<th>Coefficient</th>
<th>SE</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>L1–L4&lt;sup&gt;a&lt;/sup&gt;</td>
<td>constant</td>
<td>0.242</td>
<td>0.116</td>
<td>2.074</td>
</tr>
<tr>
<td></td>
<td>lean body mass (kg)</td>
<td>1.652E−02</td>
<td>0.002</td>
<td>7.995</td>
</tr>
<tr>
<td>Total femur&lt;sup&gt;b&lt;/sup&gt;</td>
<td>constant</td>
<td>0.556</td>
<td>0.150</td>
<td>3.703</td>
</tr>
<tr>
<td></td>
<td>age (years)</td>
<td>−2.465E−02</td>
<td>0.014</td>
<td>−1.784</td>
</tr>
<tr>
<td></td>
<td>lean body mass (kg)</td>
<td>1.570E−03</td>
<td>0.003</td>
<td>5.599</td>
</tr>
<tr>
<td>Radius33%&lt;sup&gt;c&lt;/sup&gt;</td>
<td>constant</td>
<td>−1.050E−02</td>
<td>0.113</td>
<td>−0.093</td>
</tr>
<tr>
<td></td>
<td>age</td>
<td>2.845E−02</td>
<td>0.010</td>
<td>2.886</td>
</tr>
<tr>
<td></td>
<td>lean body mass (kg)</td>
<td>6.859E−02</td>
<td>0.002</td>
<td>3.494</td>
</tr>
</tbody>
</table>

*Note:* p is the descriptive level of Pearson’s linear coefficient.

Model adjustment: a $R^2 = .825$, b $R^2 = .628$, c $R^2 = .693$. 

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further data demonstrating the relevance of this finding, such as greater rate of forearm fracture in tennis players, is necessary. Another hypothesis is related to the greater projected forearm area and, consequently, lower BMD. We suggest that the increase of area at this skeletal site reflects greater bone remodeling, indicating that intense training affects only sites of greater impact, with no relevant systemic effect. In this case, tennis players’ training must focus on minimizing the observed specific local effect.

Although there was a statistically significant difference between the BMD values of tennis players and controls, the difference was small and could also be related to the precision of the DXA measurements for BMD. We do not believe this was the case, however, because the BMD coefficient of variation is more relevant when repeated measurements are performed in the same individual. For single measurements, the coefficient of variation is not the most appropriate tool to evaluate statistical significance.

In accordance with several studies (Arabi et al., 2004; Högler, Briody, Woodhead, Chan, & Cowell, 2003; Seeman et al., 1996), LBM was the variable most correlated with bone mass and was also the main bone-mass predictor in all analyzed sites. The predictive ability of LBM on L1–L4, total femur, and radius was 33%, 33%, and 69%, respectively. The positive effect of LBM on BMD seems to be a result of the direct action of the “mechanical load” on the bone, performed by strength and muscle mass. Schoenau and Frost (2002) suggest that bone and muscle form a “bone–muscle unit” that undergoes the influence and modifications of muscle strength. Another study (Rauch, Bailey, Baxter-Jones, Murwald, & Faulkner, 2004) demonstrated that during pubertal growth, the muscle-mass acquisition peak precedes bone development, corroborating the mechanostatic theory, which suggests that the increase of muscle mass (and consequently muscle strength) during development stimulates the formation and resistance of the bone.

In this study we observed that, although presenting greater LBM, athletes did not present greater bone mass than controls at other sites than the BMC of the dominant forearm and trochanter BMD. Besides the greater remodeling in this group, another hypothesis that might explain the absence of an exercise effect on bone mass is that intense or excessive exercise (overtraining) stimulates the production of proinflammatory cytokines such as interleukin 6 and tumor necrosis factor and, thus, greater osteoclastogenesis, as well as negatively affecting bone formation by suppressing the GH/IGF-1 axis and increasing serum cortisol (Lee & Lorenzo, 2006; Nemet, Youngman, Kim, Hill, & Cooper, 2002; Scheett et al., 2002). Scheett et al. observed that 90 min of daily aerobic exercise for 5 weeks was enough to induce an increase of the proinflammatory cytokines and suppression of the GH/IGF-I axis in prepubertal boys. The tennis players evaluated in the current study trained, on average, 13.9 hr/week (5–27.3 hr/week distributed into 3–5 weekly sessions of technical training and physical conditioning) and participated in tournaments on weekends. Further studies evaluating proinflammatory cytokines and bone markers are necessary to elucidate this hypothesis.

The relationship between dietary calcium intake in childhood and adolescence and bone-mass acquisition has presented conflicting results in the literature (IOM, 1997; Heaney, 2000a; Lanou et al., 2005). In our study, calcium did not correlate with bone mass. This result might be explained by the lack of a gold
standard for dietary data collection and because short periods of dietary evaluation might not yield valid calcium intake information (Wilson & Horwath, 1996). Studies suggest that for a reliable calcium intake evaluation, estimating the intake within a period of up to 10% of the real mean for groups or individuals 7–88 days would be necessary (Palaniappan, Cue, Payette, & Gray-Donald, 2003; Wilson & Horwath). Such long periods of food registry decrease data precision because of lower adherence to the method, especially among adolescents, and potential changes in food intake (Palaniappan et al.). For more accurate dietary intake assessment in free-living participants the duplicate-plate method could be used (Yang & Boushey, 2007). Another limitation of analyzing dietary information is the incompleteness of food-composition databases.

Calcium intake was greater than observed in other studies (Petrie, Stover, & Horswill, 2004; Silva et al., 2004), even though the mean intake was still below the current recommended values. Only 6 tennis players and 2 controls had dietary calcium intake over the adequate intake of 1,300 mg/day. Perhaps lack of numbers over the adequate intake contributed to a lack of predictive ability of calcium intake on BMD and BMC. Furthermore, the adjustment of calcium intake in relation to energy (calcium in mg/1,000 kcal) showed that it was below the recommended 550 mg/1,000 kcal (tennis players 330.33 mg vs. controls 339.42 mg). In athletes, calcium loss in sweat should also be considered. Studies report sweat losses up to 3 L/hr in adult and older adolescent tennis players, which could lead to extensive loss of electrolytes, including calcium (Bergeron, 2003; Klesges et al., 1996).

Insufficient energy or protein intake might compromise bone formation by inhibiting the GH/IGF-I axis (Ballard, Clapper, Specker, Binkley, & Vukovich, 2005). Most tennis players and controls did not present energy deficit. Tennis players and controls presented protein intake above the recommendation for sedentary (0.95 g · kg\(^{-1}\) · day\(^{-1}\) for 9–13 years and 0.85 g · kg\(^{-1}\) · day\(^{-1}\) for 14–18 years; IOM, 2002) and young athletes (1.5–2 g · kg\(^{-1}\) · day\(^{-1}\); Petrie et al., 2004), and this intake was inversely correlated with the BMD at all evaluated sites for tennis players, with the exception of the femoral neck, and in L1–L4 for controls. When the adjusted energy intake was used in the multivariate regression model (protein g/1,000 kcal), however, no negative effect of protein on bone mass was observed.

Sufficient dietary protein is a key to bone health (Alexy, Remer, Manz, Neu, & Schoenau, 2005; Bonjour, 2005). Chevalley, Bonjour, Ferrari, and Rizzolli (2008) reported that high protein intake (mean intake of 2 g · kg\(^{-1}\) · day\(^{-1}\)) was associated with a higher BMC in physically active prepubertal boys. There is evidence, however, that excessive protein might negatively affect bone health by increasing the renal acid load and generating greater renal calcium loss (Alexy et al.; Massey, 2003). This effect, however, seems to be more related to an imbalance between protein and calcium intake and other alkalizing minerals than to the protein intake alone (Alexy et al.; Dawson-Hughes & Haris, 2002). Thus, the use of the ratio Ca:Ptn has been suggested as a calcium urinary excretion predictor. A ratio of 20 (mg) to 1 (g) is recommended, based on the protein and calcium recommendations proposed by the Institute of Medicine for middle-aged women (IOM, 2002). A review (Massey) that evaluated the effect of protein on bone mass, however, showed conflicting results, with Ca:Ptn values ranging from 9:1 to 17:1. In our study, some of the boys from both groups had protein intakes above values...
that showed beneficial effects, and the mean Ca:Ptn ratio was lower than the suggested (<9:1). Therefore we believe that more attention must be paid to balancing these two nutrients.

Balance studies conducted on adults showed that the Ca:P ratio’s varying from 0.08:1 to 2.4:1 does not affect calcium absorption or balance (Heaney, 2000b; IOM, 1997). Our tennis players and controls presented intake within this range.

Nutrition, exercise practice, genetic factors, and pubertal hormonal modifications affect bone mass synergistically; the isolated evaluation of each factor does not answer all the questions regarding bone mass acquisition, which is a limitation of the current study. Although calcium is the most important nutrient related to bone health, other nutritional factors determinant for its uptake and body utilization (i.e., vitamins D, K, and C; phosphorus; magnesium; and potassium) and that might negatively affect calcium bioavailability and excretion (i.e., phytates, oxalates, caffeine, and vitamin A) should be evaluated (Ilich & Kerstetter, 2000).

Data interpretation of bone mass in the adolescent population is complex because of the intense modifications that occur in BMD, bone dimension, and body composition (IOM, 1997). To better understand the interaction between nutrition and exercise, longitudinal studies and analyses of other nutritional variables related are needed, as well as biochemical markers of bone remodeling. Studies with larger samples and other methodologies might better explain the question proposed in this study. Studies evaluating long-term fracture risk and rate in this population are necessary to better understand the role of tennis in bone mass.

In conclusion, our data enable us to assert that LBM is the main BMD and BMC predictor for both controls and tennis players. Moreover, male adolescent tennis players have site-specific effects on bone mass, and training should focus on ways to minimize this negative effect on the nondominant arm. No correlation between BMD and calcium was observed. Nonetheless, calcium intake should be analyzed in the context of other nutritional variables that affects its utilization. Nutrition education for young athletes should focus on promoting a balanced diet, providing energy and nutrients in adequate amounts.

Acknowledgments

Competing interests: None declared.

References


