Childhood Obesity, Epidemiology, and Bone Thickness
Keywords: Obesity; Children; Determinants; Prevention; Genetics; Physical activity; Dietary pattern

Introduction

Childhood obesity is one of the most serious public health challenges of the 21st century. The prevalence of obesity is increasing both in developed and in developing countries and has become a major public health problem. Up to the 1980s developing countries were with the lowest rates, but since then overweight and obesity prevalence have gradually increased in children. The global prevalence of overweight and obesity in children aged 5–17 years is 10% and this global average covers a wide range of prevalence levels in different regions and countries with above 30% in America and below 2% in Sub Saharan Africa [1]. Further, projections to the year 2010 for estimated prevalence of overweight and obesity in school age children (aged 5–17 years) are with 46% in America and below 5% in Africa [2].

For children, between 5–17 years in this regional prevalence data on overweight and obesity are currently unavailable [3]. However, data for overweight and obesity prevalence among children in different countries in South Asia are available: 25.0% among children from 2 to 15 years in Bangladesh and 22.0% among children from 5 to 19 years in India. Moreover, secular trends indicate increasing prevalence rates in these countries: for example, 9.8 to 11.7% among children from 5 to 19 years in India during 2006–2009 [4,5].

In recent years, the increase in the prevalence of obesity has led this condition to the forefront of the public health in Sri Lanka, especially among children. The overweight and obesity prevalence among children in Sri Lanka shows different ranges with provincial and gender variations; among boys and girls between 8 and 10 years this was 4.3% and 3.1% respectively and obesity prevalence among primary school children in Colombo district is 5.1% in 2008 [6–8].

Obesity is a condition of abnormal or excess fat accumulation in adipose tissue, which may adversely affect health of body and increases health problems. Although the mechanism of obesity development is not fully understood, it is confirmed that obesity occurs when energy intake exceeds energy expenditure [9,10].

The normal growth of adipose tissue takes account of several phases. During first year (early infancy) of the first period, adiposity increases and it reduces in next year and remains stable for several years. Adiposity rebound start at around 6 years during second period. From this stage, both size and number of adipocytes increases [11,12]. Therefore, childhood is the critical period to develop obesity because of the adiposity rebound.

Obesity negatively influences a child’s self-esteem and results in diminished quality of life. Moreover, children with high body mass index (BMI) often become obese adults, who are at increased risk of developing obesity-related diseases, such as type 2 diabetes, hypertension, dyslipidemia and certain types of cancer and place significant financial burden on healthcare systems [12,13].

For measuring healthy body weight, it is difficult to develop one simple index for overweight and obesity in children and adolescents because their bodies undergo a number of physiological changes as they grow.

Depending on the age, there are different methods to measure a body’s healthy weight:

- For children aged 0-5 years

The WHO Child Growth Standards, launched in April 2006, include measures for overweight and obesity for infants and young children up to age 5 [14].

- For individuals aged 5-19 years

World Health Organization (WHO) developed the Growth Reference Data for 5-19 years. It is a reconstruction of the 1977 National Center for Health Statistics (NCHS)/WHO reference and uses the original NCHS data set supplemented with data from the WHO child growth standards sample for young children up to age 5 [15].

Determinants of Childhood Overweight/Obesity

A number of studies indicate that several key determinants are widely acknowledged as the main drivers of the obesity epidemic among children.
Genetics

Obesity tracks in families, and one of the strongest predictors of child overweight is the BMI of the mother and father. In recent years, progress was made in identifying genes that may contribute to this effect. A recent study showed for association with the FTO (fat mass and obesity-associated) gene and found strong associations with BMI and weight among children. Moreover, a defect in the melanocortin 4 receptor gene (MC4R) is associated with a severe and early form of monogenic obesity in children [16].

Age and gender

Age and gender have been identified as key determinants for the development of obesity. A study conducted among 2-18 children in Nigeria and revealed that Males had higher BMI than females at age group 2-6 years, whereas females had higher BMI than males at age groups 11-14 years and 15-18 years [17].

A study conducted among Sri Lankan children age between 5 and 14 found that fat free mass index (fat free mass/height$^2$) was decreased from age 5 to 6 and after that adipose tissue was increased without much increment in fat free mass index. After that fat mass index (fat mass/height$^2$) remained relatively stable and fat free mass index increased a little for girls. However, adiposity was increased until 10 years of age and fat free mass index was also increased a little for boys [18]. It clearly showed that weight gain in Sri Lankan children is due to increase in adiposity rather than increase in non-fat tissue (Figure 1).

Birth weight

Rapid weight gain which was traditionally considered as a healthy intervention for low birth weight infants is now recognized as a potential risk factor of increasing interest for obesity. In the geographically defined birth cohort of the Avon longitudinal study of pregnancy and childhood (ALSPAC), it showed that early postnatal catch-up growth, between birth and two years, is a risk factor for childhood obesity and childhood (ALSPAC), it showed that early postnatal catch-up growth, between birth and two years, is a risk factor for childhood obesity and childhood obesity. A study conducted among 2-18 children in Nigeria and revealed that Males had higher BMI than females at age group 2-6 years, whereas females had higher BMI than males at age groups 11-14 years and 15-18 years [17].

Dietary pattern

Eating fast foods and snacks: Fast foods play as a key contributor to the rising prevalence of obesity among children because of fast food’s poor nutritional quality, as fast foods have higher total energy, total fat, and saturated fat intakes; have refined carbohydrates and lower fiber intakes; and with higher energy density [20,21]. Further, fast food consumption is also associated with higher intake of sugar-sweetened beverages and French fries and lower intake of milk, fruit, and vegetables [22,23].

Skipping breakfast: Breakfast is a most important meal to start a day. Skipping breakfast leads to hunger and increases the amount of lunch. Therefore, it leads to play a key role in causing obesity in children [24]. Some studies showed the higher prevalence among students who skip breakfast than others [25,26]. Ortega et al., identified through 7 day food records that obese subject omit breakfast than normal person. It suggests that inadequate food choices contribute to poor food choices for rest of the day and it increases the risk of obesity in long term [27].

Behavioral Characteristics

Sedentary activities and screen viewing

Rapid increase in childhood obesity has also been attributed to a shift in the activity patterns from outdoor play to indoor entertainment: television viewing, internet, and computer games. A study suggested that decreasing any type of sedentary time is associated with lower health risk in youth aged 5-17 years. In particular, the evidence suggested that daily TV viewing in excess of 2 hours is associated upward in BMI [28,29]. Another study showed that overweight and obese children were more sedentary and higher screen time than normal weight children [30]. Further, a European youth heart study conducted among 9-10 year old boys and girls found the significant positive relationships between TV viewing and adiposity after adjusting for gender, age group, study location, sexual maturity and birth weight [31]. Mitchell et al. studied the association of hours of objectively measured sedentary behavior and odds of being obese and confirmed that sedentary behavior was positively associated with obesity.

Physical activity pattern

Physical activity plays an important role in protection from obesity. A study revealed that Prevalence of overweight and obese was higher among children who travel to school in motor vehicle than children who travel by cycle or walk [32]. Another study done in Iran reported significant difference in physical activity among obese and non-obese children. Non-obese children engaged with more physical activities like running, football, and travel to school by foot than obese children did [33]. A study conducted in Bangladesh reported that physical activity more than 30 minutes have protective effect on obesity in 10-15 year old school children [28].

Sleeping hours

In school-age children, several studies have consistently reported that short sleep duration was an independent risk factor for obesity [34]. A cross-sectional study conducted among 229 Mexican American 8-10-year-olds and concluded that children who slept less were more likely to have a higher BMI Z-Score [35]. A cohort study done in Japan observed positive relationship between decreased sleeping hours and obesity after adjusting for potential confounding factors [36].

Strategies for Childhood Obesity Prevention

Obesity prevention is not simply an issue of individual responsibility and prevention strategies seem to be more effective in children than in adults. Therefore, Successful strategies for obesity prevention among
children should be targeted and implemented in natural settings for influencing the diet and physical activities at home, in preschool institutions, schools, or after-school care services.

**School-Based Strategies**

School-based programs have great opportunity to promote healthy nutrition and physical activity because most children attend school and a child spends more than half of his/her waking hours at school on any given school day. Therefore, following school-based interventions should be implemented as standard practice in educational settings improvements in knowledge, attitudes, and behavior:

- Components on healthy eating, physical activity and body image should be integrated into the regular curriculum.
- Sessions for physical activity and the development of fundamental movement skills should be included throughout the school week.
- The nutritional quality of foods made available to students (e.g. in school canteens) should be closely monitored and improved.
- An environment and culture should be created that supports children eating nutritious foods and being active throughout each day.
- Parents should be engaged to support activities in the home setting to encourage children to become more active, eat more nutritious foods and spend less time in screen-based activities.

**Home-Based Strategies**

Parental initiative is necessary to succeed the home-based strategies influencing the diet and physical activity among children. Therefore, parents should be aware about following activities:

- Should be good role models.
- Should encourage the children to have regular meals including breakfast at home, because children who take part in family meals are also more likely to eat fruits, vegetables, and grains and less likely to snack on unhealthy foods.
- Should provide the children with healthy food choices.
- Should encourage young children to develop good eating habits and preferences for healthy foods because eating behaviors that develop during childhood tend to track into adulthood.
- Should accompany children to nearby parks and play with them. This would not only add to their health benefits but also let them supervise their children. Further, it helps to limit sedentary activities.
- Should involve the children in food purchasing by taking children to food shopping and allowing them to select healthy foods [37-39].

**Conclusion**

Childhood obesity leads to its related non-communicable diseases and psychosocial health problems in children. This is because Childhood obesity is a crucial issue that needs to be addressed urgently. Changing dietary practices and maintenance of regular physical activity starting as early as infancy through parental initiative and social support interventions are the most important strategies to tackle childhood obesity.
The relationship between visceral fat thickness and bone mineral density in sedentary obese children and adolescents

Abstract

Background: Among adults, obesity has been positively related to bone mineral density. However, recent findings have pointed out that abdominal obesity could be negatively related to bone density. The above mentioned relationship is not clear among pediatric populations. Therefore, this cross-sectional study analyzed the relationship between thickness of abdominal adipose tissue and bone mineral variables in sedentary obese children and adolescents.

Methods: One hundred and seventy five obese children and adolescents (83 male and 92 female) with ages ranging from 6 to 16 years-old were analyzed. Bone mineral content and density were estimated by dual-energy X-ray absorptiometry and ultrasound equipment which estimated the thickness of the abdominal adipose tissue. Pubertal stage was self-reported by the participants.

Results: The mean age was 11.1 (SD = 2.6). Thickness of the abdominal adipose tissue was negatively related to bone mineral density ($r = -0.17$ [95%CI: -0.03; -0.32]), independent of gender, pubertal stage and other confounders ($\beta = -0.134 \pm 0.042$ [95%CI: -0.217; -0.050]).

Conclusions: In sedentary obese children and adolescents abdominal obesity is negatively related to bone mineral density, suggesting a potential link between abdominal obesity and osteoporosis.

Keywords: Child, Adolescents, Obesity, Bone size, Bone density, Ultrasonography

Background

In modern society osteoporosis is a highly occurring disease and constitutes a public health concern due to its impact on public costs [1]. Early life has been pointed out as a crucial period in the development of osteoporosis. Childhood and adolescence are phases of the human development during which the adult bone mass density is determined and, therefore, problems during this period of life could compromise bone health in adulthood [2].

Worldwide, children and adolescents are widely affected by obesity and its comorbidities [3-6]. Despite these related comorbidities, overweight/obesity has been associated with a lower occurrence of osteoporosis in adulthood. However, body weight is composed of lean and fat mass and the actual effect of the adipose tissue on bone mineral density (BMD) is not clear.

Moreover, the distribution of the adipose tissue could be a relevant confounder in this complex process that links obesity to osteoporosis. Recently, Bhupathiraju et al. [7] analyzing Porto Rican adults (47–79 years) observed that a higher abdominal fat mass (in kg) is related to a lower BMD, but the amount of visceral and subcutaneous abdominal adipose tissue were not assessed. Furthermore, there is an absence of data about this issue in pediatric populations. Understanding the relationship between pediatric obesity and bone health is relevant for health professionals [8-10], because childhood and adolescence are two critical periods in the prevention and development

* Correspondence: romulo@fct.unesp.br

1 Department of Physical Education, UNESP Univ Estadual Paulista, Presidente Prudente, Brazil
5 Group of Scientific Research Related to Physical Activity. Department of Physical Education, UNESP Univ Estadual Paulista, Presidente Prudente, Brazil

Full list of author information is available at the end of the article

© 2013 Júnior et al.; licensee BioMed Central Ltd. This is an Open Access article distributed under the terms of the Creative Commons Attribution License (http://creativecommons.org/licenses/by/2.0), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Júnior et al. BMC Pediatrics 2013, 13:37
http://www.biomedcentral.com/1471-2431/13/37
of diseases in adulthood (e.g. arterial hypertension, diabetes mellitus and dyslipidemias) [2,8-10]. So, the purpose of this study was to analyze the relationship between abdominal adipose tissue and BMD in obese children and adolescents.

Methods
Participants
The present study was approved by the Ethical Research Expert Committee of the Universidade Estadual Paulista in Presidente Prudente, Brazil (# 087/2008). The sample size was estimated through an equation for correlation’s coefficients, which took into consideration a power of 80% and significance of 5% ($z = 1.96$). A previous study analyzed the relationship between BMD and intra-abdominal adipose tissue (IAAT) and identified correlations ranging from $r = -0.31$ to $r = -0.65$ [11]. Thus, the lowest coefficient was inserted in the equation and the minimum sample size indicated was 80 subjects.

The subjects were invited, through television and newspaper advertising, to participate in this study. The participants contacted the researchers by phone and an appointment was made in order to take measurements at the Campus of the University. Initially, the diagnosis of obesity was based on body mass index cut-off points, adjusted by sex and age, developed by Cole et al. [12]. After a positive diagnosis of obesity, some inclusion criteria were also used to select the sample: i) aged between 6 and 17 years [chronological age computed taking into account their birthday and the measurement day]; ii) no engagement in regular physical activity within the three months prior to the study (established via a face-to-face interview with the child/adolescent and their parents); iii) a self-report of no diagnosis of either cardiovascular disease or regular medicine use and iv) a consent form signed by parents/guardians to participate in the study. Finally, one hundred and seventy five obese children and adolescents (83 male and 92 female) with ages ranging from 6 to 16 years-old were included in the study.

Bone mineral density
Body composition and BMD were estimated by Dual-energy X-ray absorptiometry (DEXA) (Lunar DPX-NT; General Electric Healthcare, Little Chalfont, Buckinghamshire [software version 4.7]). The method estimated the percentage of body fat (%BF) and trunk fat mass (TFM [kg]), as well as, whole-body bone mineral density (BMD [g/cm$^2$]). All data were collected by trained staff and all measurements were taken at the laboratory of the University, in a temperature controlled room. Each morning, before the first measurement, the DEXA equipment was calibrated by the same researcher according to the references provided by the manufacturer.

Ultrasound measures of the abdominal adipose tissue
Ultrasound equipment (Toshiba Aplio Model Tochigi-ken, Japan) was used to measure the thickness of adipose tissue in the abdominal region. IAAT was defined as the distance between the internal face of the rectus abdominal muscle and the anterior wall of the aorta. Thus, the thickness (in cm) of IAAT was estimated. For statistical analysis, the values of IAAT were stratified into tertile (Tertile-1 [bottom]; middle Tertile-2 [middle]; Tertile-3 [top]). All measurements were taken by a trained physician, in a Hospital, in a room with a constantly controlled temperature. Each morning, before any measurements were taken, the device was calibrated and, according to the reference values provided by the manufacturer, the tests presented high reliability.

Pubertal stage
Pubertal stage was self-assessed by the participants. The subjects received a standardized series of drawings to assess their own pubertal development (Girls: drawings with five stages of breast and female pubic hair development; Boys: drawings with five stages of genitalia and male pubic hair development) [13,14]. These scales have been previously validated in Brazilian pediatric populations [15,16]. The drawing had appropriate descriptions accompanying it. The results were placed by each subject in a locked box to guarantee the integrity and anonymity of the subjects, and only the main researcher had access to them. All pubertal measurements were performed at the University laboratory.

Statistical analysis
The Kolmogorov-Smirnov (K-S) test, used to test the distribution of the numerical variables and logarithm transformation, was used in variables of non-parametric distribution. Mean, median, 95% confidence interval (95% CI), standard deviation (SD) and interquartile range ($P_{25}$-$P_{75}$) were used as descriptive statistics. For analysis of variance (ANOVA), homogeneity assumption was assessed in advance. If the assumption was in accordance, test F was performed and, in cases of statistical differences, the tertiles of IAAT were tested through Tukey’s multiple comparisons. Pearson’s correlation was used to analyze the relationship between the numerical variables. In cases of moderate-high correlations, multivariable models were elaborated using linear regression and, therefore, the correlations between BMD and IAAT were adjusted by potential confounders (sex, age, total body fatness, trunk fatness, height and pubertal stage). Chi-square test analyzed associations between categorical variables and Yates correction was applied in 2x2 contingence tables. Statistical significance was set at 5% and statistical software BioEstat version 5.0 was used for all analyses.
Results

General characteristics of the analyzed sample are presented in Table 1. The K-S test indicated that most of the numerical variables were under non-parametric distribution, except for weight, height and %BF. Male gender made up 47.4% of the sample and there was a similarity between the numbers of boys and girls (P = 0.496). The mean age was 11.1 (SD = 2.6), and ranged from 6 to 16 years-old and the proportion of children and adolescents was similar (P = 0.112).

There were more children and adolescents at initial pubertal stage (40%) than final pubertal stage (24.6%) (P = 0.037). Children and adolescents at a higher pubertal stage had a higher BMD (Stage-1 = 0.96 ± 0.07 g/cm²; Stages 2-3 = 1.06 ± 0.10 g/cm²; Stages 4-5 = 1.16 ± 0.10 g/cm²; ANOVA P = 0.001). Boys and girls had similar values of BMD (1.05 ± 0.11 g/cm² and 1.04 ± 0.12 g/cm² [P = 0.630], respectively). Adolescents presented higher BMD than children (6-10 years = 0.95 ± 0.05 g/cm² and 11-17 years = 1.12 ± 0.10 g/cm² [P = 0.001], respectively).

Age (r = 0.80), weight (r = 0.78), height (r = 0.70), TFM (r = 0.63) and pubertal stage (r = 0.66) were significantly related to BMD, except for %BF values (r = 0.09). In the multivariable model, IAAT was negatively related to BMD, independent of other confounders (Table 2). IAAT was stratified into tertiles and the values of BMD (Figure 1) were compared between them. Obese adolescents in the highest IAAT tertile presented lower BMD than those in the lowest IAAT tertile.

Discussion

A cross-sectional study, which identified that IAAT is inversely related to BMD. In this pediatric sample, the mean of IAAT was 4.35 cm which seems a normal value, because it was similar to another group of sedentary obese children and adolescents without non-alcoholic fat liver disease (mean value of 4.1 cm) [17]. On the other hand, the same group of obese children and adolescents had a significant decrease of 1.6 cm after one year of exercise intervention [17], supporting the relevance of the sedentary lifestyle as an inclusion criteria and evidencing the potential of prolonged engagement of physical exercise in combating comorbidities related to abdominal obesity [8,9]. The inclusion of the sedentary lifestyle was also important because there is a positive relationship between increased habitual physical activity and BMD [18].

---

Table 1 General characteristics of obese children and adolescents (n = 175)

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean (SD)</th>
<th>(95% CI)</th>
<th>Median (P25-P75)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>11.1 (2.6)</td>
<td>(10.7; 11.5)</td>
<td>11 (4)</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>66.8 (19.2)</td>
<td>(63.9; 69.7)</td>
<td>64.1 (25.3)</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.50 (0.12)</td>
<td>(1.48; 1.52)</td>
<td>1.50 (0.20)</td>
</tr>
<tr>
<td>%BF</td>
<td>45.4 (5.2)</td>
<td>(44.6; 46.2)</td>
<td>45 (7.2)</td>
</tr>
<tr>
<td>TFM (kg)</td>
<td>13.8 (5.1)</td>
<td>(13.1; 14.5)</td>
<td>13.4 (6.3)</td>
</tr>
<tr>
<td>IAAT (cm)</td>
<td>4.35 (1.5)</td>
<td>(4.13; 4.58)</td>
<td>4.1 (1.8)</td>
</tr>
<tr>
<td>BMD (g/cm²)</td>
<td>1.04 (0.12)</td>
<td>(1.03; 1.06)</td>
<td>1.03 (0.19)</td>
</tr>
<tr>
<td>Gender</td>
<td>n (%)</td>
<td></td>
<td>P</td>
</tr>
<tr>
<td>Male</td>
<td>83 (47.4)</td>
<td></td>
<td>0.496</td>
</tr>
<tr>
<td>Female</td>
<td>92 (52.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pubertal stage</td>
<td></td>
<td></td>
<td>0.037</td>
</tr>
<tr>
<td>Stage 1</td>
<td>70 (40)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stages 2-3</td>
<td>62 (35.4)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stages 4-5</td>
<td>43 (24.6)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td></td>
<td></td>
<td>0.112</td>
</tr>
<tr>
<td>6-10 years</td>
<td>77 (44)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>11-17 years</td>
<td>98 (56)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

SD = standard-deviation; 95%CI = 95% confidence interval; %BF = percentage of body fatness; Trunk-BF = trunk body fatness; IAAT = intra-abdominal adipose tissue; BMD = bone mineral density; TFM = trunk fat mass.

Table 2 Relationship between intra-abdominal adipose tissue and variables related to the bones of children and adolescents

<table>
<thead>
<tr>
<th></th>
<th>Univariate</th>
<th>Multivariable model</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>r (95% CI)</td>
<td>βadjusted ± SEM (95% CI)</td>
</tr>
<tr>
<td>BMD*</td>
<td>−0.17 (−0.03; −0.32)</td>
<td>−0.134 ± 0.042 (−0.217; −0.050)</td>
</tr>
</tbody>
</table>

IAAT* = numerical variable under logarithm transformation; βadjusted = model adjusted by sex, age, height, total body fatness, trunk fatness and pubertal stage; BMD = bone mineral density; IAAT = intra-abdominal adipose tissue; SEM = standard error mean; 95%CI = 95% confidence interval.

---

Figure 1 Bone mineral density according to intra-abdominal adipose tissue tertiles.
Gender is an important variable related to skeletal formation. Male adolescents, more than girls, are affected by biological processes that accelerate bone development [2]. In the final stages of adolescence, boys are taller and have a higher bone mineral density [2]. Through childhood and adolescence, when compared to the female gender, boys have an increased likelihood of practicing sports [8,9], which are related to bone development. Moreover, during adulthood, hormonal characteristics of male obesity may exert deleterious effects on bone microarchitecture [19]. Therefore, the inclusion of gender as a potential confounder constitutes a methodological strength, because it indicates that IAAT is inversely related to BMD independent of this important confounder.

Agreeing with previous studies, in this sample there was a positive relationship between body weight/adiposity and BMD [11,20]. Indeed, obese subjects, from an early age, have increased bone density, mainly due to the stress occasioned by the increased weight on bone tissue that causes deformation and, hence, leads to bone remodeling [2]. Similarly, the same mechanical/biochemical process offers support to the idea that sports with impact are important tools in the promotion of adequate bone health in adolescents [2]. It is noteworthy that although obesity is positively related to BMD, more recent findings suggest that bone quality is compromised in obese subjects [21].

On the other hand, our findings point out that body fatness distribution should be considered as a potential confounder in this relationship, because increased IAAT was inversely related to BMD, independent of general obesity. Previous studies involving anthropometric (waist-to-hip ratio) [20] and DXA variables (abdominal fatness in kg) [7] identified similar relationship patterns in children and adults, respectively.

The inverse relationship between IAAT and BMD could be based on the action of adipokines produced by adipose tissue over growth mediators related to bone development. Nemet et al. [22], identified, in a longitudinal design, that the practice of very high intensity physical exercise simultaneously caused a significant increase in pro-inflammatory markers (tumor necrosis factor - alpha) and a subsequent decrease in growth mediators, such as insulin-like-growth-factor-I. Similarly, the adipose tissue located in the abdominal region (mainly the visceral one) has a special role in the release of adipokines into the bloodstream [23]. Therefore, it is possible to believe that IAAT could be a risk factor related to a harmful effect in bone remodeling and in turn to a risk factor of osteoporosis in adulthood.

Visceral adipose tissue is related to insulin resistance and insulin plays a role in the proliferation of osteoblasts. Thus, decreased insulin action may be one of the possible mechanisms by which obesity affects bone mass. In agreement, in a recent study, increased insulin concentration and HOMA-IR were considered negative predictors of bone mineral density in adolescents [24]. However, this is a recent finding that needs further top research focusing on the understanding of the physiological common mechanisms behind this association.

Our study has positive points, such as: (i) the sample size calculation; (ii) the use of adequate techniques to measure body composition/IAAT. On the other hand, limitations should be recognized: (i) the cross-sectional design (absence of causality statements), (ii) an absence of measurements relating to the intake of calcium and vitamin D and (iii) an absence of pro-inflammatory adipokines. Moreover, there were correlations of low magnitude between BMD and IAAT, indicating that other variables are important in this relationship and, therefore, further studies are necessary to identify them.

**Conclusions**

In summary, our findings indicate that abdominal obesity negatively affects the bone density of obese children and adolescents, indicating that abdominal obesity could be a determinate in the development of osteoporosis in adulthood. Further studies should analyze whether this negative effect also occurs in non-obese youth.

**Abbreviations**

BMD: Bone mineral density; IAAT: Intra-abdominal adipose tissue; BMI: Body mass index; TFM: Trunk fat mass; DEXA: Dual-energy X-ray absorptiometry; BF: Body fat; TFM: Trunk fat mass; K-S: Kolmogorov-Smirnov; SD: Standard deviation; ANOVA: Analysis of variance; Cm: Centimeters; SEM: Standard-error mean.
“This course was developed and edited from the open access article: Mohamed SM (2015) Childhood Obesity: Epidemiology, Determinants, and Prevention. J Nutr Disorders Ther 5:156 (doi:10.4172/2161-0509.1000156), used under the Creative Commons Attribution License.”

“This course was developed and edited from the open access article: Júnior et al.: The relationship between visceral fat thickness and bone mineral density in sedentary obese children and adolescents. BMC Pediatrics 2013 13:37. (doi:10.1186/1471-2431-13-37), used under the Creative Commons Attribution License.”