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Resistin and visfatin concentrations are related to central obesity and inflammation in Brazilian children

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Abstract

Background: The evidence that cardiovascular disease begins in childhood and adolescence, especially in the presence of excess weight, is associated with dysfunction on adipokine pro-inflammatory secretion. These affect glucose metabolism and lead to other complications related to insulin resistance and cardiovascular disease. This study assessed the association of anthropometric and metabolic parameters related to obesity, cardiovascular risk, and insulin resistance with concentrations of resistin and visfatin, in children.

Methods: A cross-sectional study was developed with 178 children of 6–10 years old enrolled in public city schools. Anthropometric data, composition body, clinical, and biochemical were measured according to standard procedures. We used multiple regression models by stepwise method to evaluate the associations of resistin and visfatin with variables of interest.

Results: In healthy weight children, resistin was associated with LDL cholesterol, visfatin, atherogenic index, and waist-to-height ratio, whereas in obese children resistin was associated with visfatin and interaction between conicity index and HOMA-AD. Furthermore, in healthy weight children, visfatin was associated to resistin and triceps skinfold thickness and negatively associated to HOMA-AD, while in obese ones visfatin was associated with waist-to-height ratio, atherogenic index, resistin, and interaction between trunk adiposity index and adiponectin and was negatively associated with the HOMA-IR index.

Conclusions: Our study shows an association between anthropometric and biochemical variables related to visceral fat and inflammation. These results suggest the resistin and visfatin as good pro-inflammatory markers. In addition, both adipokines are strongly related to central obesity, in children.

Keywords: Adipokines, Adipose tissue, Insulin resistance, Waist-height ratio, Adiposity, Body composition, Metabolic diseases

Background

Obesity is a condition of chronic inflammation of low intensity resulted primarily from excess fat [1, 2]. Several anthropometric and body composition indexes have been used to measure obesity and to assess the risk of metabolic and cardiovascular diseases [3, 4].

Excess body fat is a risk factor for cardiovascular disease (CVD) and type 2 diabetes (DM2) [1, 4–6], especially the visceral fat. Thus, it is essential to investigate measures that not only reflect body proportions but also body fat distribution. Waist circumference, trunk adiposity index (TAI), and waist-to-height ratio (WHtR) have a good correlation with central adiposity and the low cost make these available and easily applicable indicators in clinical practice and research [6–9]. Some studies have shown that waist circumference in children tends to increase with growth, requiring specific references for sex and age [10]. Waist-to-height ratio has the advantage of not requiring such references or points of cut-offs, which facilitates its use [10–12].

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Studying anthropometric indicators that can predict accumulation of visceral fat as a risk factor is relevant. The visceral fat increased is related with macrophage population, which is majorly responsible for production of inflammatory cytokines. The inflammatory cytokines have a paracrine action that compromise vascular homeostasis and lead to systemic inflammation and endothelial dysfunction, such as changes in the concentration of resistin and visfatin [3, 13, 14].

Studies have shown that resistin has direct effect on endothelial cells of human coronary arteries, which contributes to arterial inflammation, accumulation of cholesterol, triglycerides, and insulin resistance [15, 16]. Other studies have shown the important role of resistin in controlling the cascade of inflammatory cytokines, which makes it a pro-inflammatory marker next to visfatin [2, 13, 17]. In addition to pro-inflammatory action, visfatin is essential for β-pancreatic cells in insulin secretion [18, 19] and also stands as a marker of visceral adipose tissue [4, 20].

Furthermore, accumulation of adipose tissue, dyslipidemia, and inflammation are risk factors for atherosclerosis and insulin resistance [21]. Studying these factors in obese children is an opportunity to examine the consequences of early stages of obesity, free of other pro-inflammatory conditions such as smoking, cardiovascular disease, and kidney and liver disease. Our study will help to determine the association between anthropometric and metabolic parameters related to obesity, cardiovascular risk, and insulin resistance with concentrations of resistin and visfatin in children.

Methods

Study design and population

A cross-sectional study was developed in 2009 with 178 children of 6–10 years old, 104 girls and 74 boys, enrolled in public city schools of Nova Era, in the state of Minas Gerais, Brazil.

The city of Nova Era had 1024 children aged 6–10 years enrolled in public schools; of those, 6.4% ($n = 65$) are with obesity [22]. We included two healthy weight children ($n = 130$) for each obese child ($n = 65$), resulting in a 195 student sample. We excluded students with acute and chronic diseases, changes in the gastrointestinal tract, weight loss in the last 6 months, special diets, using medicines that alter metabolism and/or affect inflammation pathways, and C-reactive protein (CRP) levels above 10 mg/L. In the end, we included 57 obese and 121 healthy weight children.

The study followed the principles of the Declaration of Helsinki and was approved by the Research Ethics Committee of the Federal University of Ouro Preto under the number 2007/93. A written consent was signed in duplicate and obtained from those responsible.

Anthropometric, clinical, and body composition variables

The students were instructed to attend school with light clothes, fast for at least 4 h, and not to do vigorous exercise before the anthropometric and body composition assessment. Body mass index (BMI) was calculated by dividing weight (Tanita®BC554 Ironman, Illinois, USA) and the square of height (AlturExata®, Belo Horizonte, Brazil), which were measured according to the protocol [23]. These measures were also used for calculating the ponderal index (PI) = (weight, kg)/(height, m)^3 [24].

Circumferences were measured using a tape measure with 0.1 cm accuracy. Arm circumference (AC) was measured in duplicate at the midpoint between the acromion process of the scapula and the olecranon. Waist circumference (WC) was measured in triplicate at the midpoint between the anterior superior iliac crest and the last rib, then used to calculate the conicity index (C index), C index = (WC, cm)/(0.019 $\sqrt{}$((weight, kg)/(height, m))) [25]. All measurements were performed in the right side of the body.

Triceps skinfold (TSF) and subscapular skinfold (SSF) were also measured in triplicate and not consecutively using the caliper Cescorf® (Cescorf Equipment Ltd., Porto Alegre, Brazil). TSF was measured at the midpoint between the acromion and the olecranon on the back of the arm and SSF was measured in the marked point at the 45° diagonal, 2 cm below the lower angle of the scapula. Trunk adiposity index (TAI) was calculated based on these measurements, TAI = SSF (mm)/TSF (mm) [26].

Blood pressure (BP) was measured by vascular Doppler ultrasound using Doppler DV 610 after 5-min rest. The procedure was performed in triplicate with 2-min intervals between each measurement. All values were replaced by the corresponding arithmetic means.

Body fat percentage (BF%) was assessed through tetrapolar bioelectrical impedance (TBI) using Maltron® (Maltron® Bioscan BF-916, Maltron International Ltd., Essex, UK). We also used an equation to estimate body fat percentage by the sum of both triceps and subscapular skinfolds, as proposed by Slaughter et al. [27].

Biochemical variables

After 12 h fasting, a 10-mL blood sample was withdrawn by puncture of the cubital vein into 10-mL disposable tubes and then fractionated into vials containing sodium fluoride for glucose analysis and without anticoagulant for dosing cholesterol and ratios. After being centrifuged (Excelsa Baby® model 206–2; FANEM, São Paulo, Brazil), the serum was divided into three amber microtubes and stored at − 80 °C for further analysis.

Blood glucose was determined by the enzymatic-colorimetric method and insulin by chemiluminescence (Kit ultrasensitive insulin, Beckman Coulter). Then we

calculated homeostatic model assessment for insulin resistance (HOMA-IR) using the equation HOMA-IR = (fasting insulin (μUI/mL) × fasting glucose (mmol/mL)/22.5) [28] and homeostatic mode assessment-adiponectin (HOMA-AD) using the equation HOMA-AD = fasting insulin (mU/L) × fasting glucose (mg/dL)/adiponectin (μg/mL) [29]. The quantitative insulin sensitivity check index (QUICKI) was obtained by the equation QUICKI = 1/log (fasting insulin) + log (fasting glucose) [30].

Triacylglycerol concentrations and total cholesterol (TC) were determined using the CM 200 analyzer (WIENER LAB, Rosario, Argentina) by the enzymatic colorimetric method using Triglycerides Liquicolor mono kits and Cholesterol Liquicolor (Human of Brazil, Itabira, Brazil). The high-density lipoprotein cholesterol (HDL-C) fraction was measured by direct enzymatic colorimetric method HDL-PP (Analisa, Gold Analisa Diagnostics Ltd., Belo Horizonte) while the concentrations of low-density lipoprotein cholesterol (LDL-C) were calculated using the Friedewald equation [31]. Based on these concentrations, the atherogenic index (AI) was calculated by the ratio of total cholesterol/HDL-C [32].

Concentrations of adiponectin (Linco Research Kit—St Charles, Missouri, USA), resistin (PeproTech® Kit, Rocky Hill, NJ, USA), and visfatin (USCN Kit Life Science Inc., Wuhan, China) were obtained by ELISA (enzyme-linked immunosorbent assay). C-reactive protein concentrations were processed in the IMMAGE® 800 analyzer (Beckman Coulter, Fullerton, California, USA) by nephelometry, with detection over 0.1 mg/dL.

Statistical analysis

Data were presented as mean and standard deviation for normally distributed variables or as mean and interquartile range for not-normal distributed, previously tested by Kolmogorov-Smirnov test. Comparing groups, samples with normal distribution were analyzed using a *t*-test of two independent samples, and non-parametric samples were tested by Wilcoxon-Mann-Whitney test.

To determine which variables were associated with adipokines and the magnitude of these associations, multiple regression models were constructed by a stepwise method with resistin and visfatin as dependent variables. Initially, all independent variables were included in the model, leaving only significant variables in the subsequent models. Some interactions were also included at this stage. If a significant interaction is present in the model, the effect of a variable x_1 on the average response depends on the second variable x_2 and vice versa. In this case, we considered a multiple regression model with two regression variables, as shown in the formula:

$$Y = \beta_0 + \beta_1 x_1 + \beta_2 x_2 + \beta_3 \underbrace{x_1 x_2}_{\text{Interaction}}$$

Analysis of residuals of each model was performed to assess validity of assumptions of normality, homoscedasticity, and independence between observations. The statistics Cook's distance and variance inflation factor (VIF) were used to identify outliers and to check for possible multicollinearity between independent variables [33, 34].

This study considered α of 0.05 as significant for all statistical tests. For analysis, we used the statistical software R.2.13.1.

Results

The characteristics of the students are shown in Table 1. The sample consisted of 104 girls (58.4%) and 74 boys (41.6%). The obese group showed mean/median anthropometric values higher than the healthy weight group. In addition, all variables analyzed were significantly different between groups, except age, HDL-C, adiponectin, and resistin.

Regression models in Tables 2 and 3 showed β-coefficients ± SE, R^2, and VIF. The VIF is responsible for quantifying multicollinearity between the independent variables, since it measures how much the variance of an estimated regression coefficient is greater because of collinearity [35]. For all adjusted models, VIF was lower than 2.0, indicating no multicollinearity between the independent variables, as all adjusted models were significant at the 0.05 significance level. Adjustments for age and sex were not performed, as the groups did not differ for these parameters.

Resistin was associated with LDL-C, visfatin, AI, and WHtR among healthy weight children (Table 2). While in obese ones, resistin was associated with visfatin with interaction between conicity index (CI) and HOMA-AD considering a significance level of 10% (Table 2). Visfatin levels were positively associated with concentration of resistin and triceps skinfold and negatively with HOMA-AD index in healthy weight children (Table 3), whereas in obese ones visfatin was positively associated with WHtR, AI, resistin, and interaction of TAI vs. adiponectin, and a negative association with HOMA-IR index (Table 3).

Discussion

In this study, obese children had higher concentrations of visfatin ($p < 0.05$) and similar concentrations of resistin ($p = 0.479$) compared to healthy weight children. These results confirm earlier studies [36–40]. Further, both adipokines were associated with central obesity markers, lipid metabolism, and insulin resistance.

In the case of central obesity, the strong association of waist-to-height ratio with both concentrations of resistin and visfatin along with absence of association

Table 1 Anthropometric, clinical, and metabolic characteristics and adipokine levels of schoolchildren 6–10 years of Nova Era, Minas Gerais, Brazil, 2009

	Normal weight	Obese	*p* value
	n = 121	*n* = 57	
Sex (*n* M/F)	49/72	25/32	–
Age (years)	8.0 (7.0–9.0)	8.0 (7.0–9.0)	0.618
Weight (kg)	27.7 (24.0–30.6)	40.9 (36.1–48.3)	*< 0.001**
Height (cm)	130.83 ± 9.52[a]	135.11 ± 9.45[a]	*0.006**
BMI (kg/m^2)	15.7 (15.1–16.53)	22.4 (20.7–25.13)	*< 0.001**
Arm circumference (cm)	19.3 (18.0–20.2)	25.7 (24.05–27.65)	*< 0.001**
Waist circumference (cm)	58.5 (55.7–61.0)	76.5 (70.87–83.0)	*< 0.001**
Triceps skinfold (mm)	8.66 (7.18–10.60)	21.13 (16.23–24.11)	*< 0.001**
Subscapular skinfold (mm)	6.03 (5.23–7.6)	19.46 (12.83–24.86)	*< 0.001**
BF from skinfolds (%)	14.22 (11.96–17.33)	32.08 (27.11–36.94)	*< 0.001**
BF tetrapolar (%)	15.78 ± 5.50[a]	27.66 ± 5.83[a]	*< 0.001**
Systolic BP Doppler (mmHg)	92.6 (87.3–98.3)	103.3 (94.8–111.6)	*< 0.001**
Fasting glucose (mg/dL)	83.48 ± 7.45[a]	86.14 ± 7.08[a]	*0.023**
Fasting insulin (mg/dL)	4.72 (3.25–6.42)	7.61 (5.34–13.13)	*< 0.001**
Total cholesterol (mg/dL)	147.75 ± 27.08[a]	161.84 ± 30.68[a]	*0.004**
HDL cholesterol (mg/dL)	57.0 (50.0–66.0)	54.0 (48.0–64.0)	0.160
LDL cholesterol (mg/dL)	74.82 ± 23.00[a]	87.38 ± 23.86[a]	*0.001**
VLDL cholesterol (mg/dL)	12.59 (9.68–19.79)	18.82 (13.05–23.68)	*< 0.001**
Triacylglycerols (mg/dL)	63.0 (48.5–99.0)	94.0 (65.0–118.5)	*< 0.001**
Waist/height ratio	0.44 (0.43–0.46)	0.56 (0.53–0.60)	*< 0.001**
Atherogenic index	2.58 ± 0.50[a]	2.94 ± 0.49[a]	*< 0.001**
Conicity index	1.17 (1.14–1.20)	1.26 (1.22–1.29)	*< 0.001**
Trunk adiposity index	0.76 (0.64–0.84)	0.92 (0.77–1.06)	*< 0.001**
HOMA-AD index	14.01 (8.42–24.21)	25.21 (16.37–51.0)	*< 0.001**
HOMA-IR index	0.96 (0.69–1.32)	1.64 (1.13–2.76)	*< 0.001**
QUICKI	0.38 (0.36–0.40)	0.35 (0.32–0.37)	*< 0.001**
Adiponectin (μg/mL)	26.89 (18.23–39.66)	26.30 (19.74–35.11)	0.473
C-reactive protein (mg/mL)	ND (ND–0.051)	0.148 (ND–0.449)	*< 0.001**
Resistin (ng/mL)	4.02 ± 1.86[a]	4.27 ± 2.00[a]	0.479
Visfatin (ng/mL)	2.19 (1.40–3.26)	2.66 (2.10–3.71)	*0.037**

Abbreviations: *M* male, *F* female, *BMI* body mass index, *BF* body fat, *BP* blood pressure, *HDL-C* high-density lipoprotein cholesterol, *LDL-C* low-density lipoprotein cholesterol, *VLDL-C* very-low-density lipoprotein cholesterol, *HOMA-AD* homeostatic model assessment-adiponectin, *HOMA-IR* homeostatic model assessment for insulin resistance, *QUICKI* quantitative insulin-sensitivity check index[a]: mean ± SD. Significant values are in italic. *p < 0.05

with BMI and body fat percentage. This suggests that these adipokine concentrations are related not only with total fat but also with fat distribution. Scientific evidence shows that WHtR was strongly associated with abdominal fat measured by advanced imaging techniques such as computed tomography and magnetic resonance imaging [18]. Several studies of sensitivity and specificity demonstrated the WHtR as a method to use for identifying childhood overweight, obesity, and tracking of inflammatory and cardiometabolic risk in 6- to 16-year-old children [4, 6, 7, 10–12].

Another relevant factor in central obesity is the evidence that increase in body fat, especially visceral, results in cellular hypoxia process, which lead to adipocyte death by necrosis or apoptosis, and increased macrophage population. This inflammatory process is primarily responsible for production of adipokines, resistin and visfatin [13, 41]. A study by Li et al. showed that pubescent girls with acute inflammation showed no association between resistin and anthropometric variables, implying that pro-inflammatory effects of this adipokine were not dependent on obesity only. This can be seen in

Table 2 Multiple regression analyses between resistin level with anthropometric and biochemical variables in schoolchildren 6–10 years of Nova Era, Minas Gerais, Brazil, 2009

	β-coefficient ± SE	*p* value	Model R^2	VIF
Constant	−3.054 ± 1.623	0.063	20,70%	
Visfatin (ng/dL)	0.507 ± 0.146	*0.001**		1.0
LDL-C (mg/dL)	0.016 ± 0.007	*0.038**		1.0
Atherogenic index	1.940 ± 0.864	*0.028**		1.0
Waist/height ratio	6.487 ± 2.651	*0.016**		1.0
Constant	2.537 ± 0.728	*0.001**	10,60%	
Visfatin (ng/dL)	0.042 ± 0.1812	*0.023**		1.1
CI* HOMA-AD	0.011 ± 0.006	*0.069*†		1.1

Model normal weight: resistin = visfatin + LDL-C + atherogenic index + waist/height ratio. Model obese: resistin = β_0 + visfatin + conicity index × HOMA-AD
Abbreviations: *CI* conicity index, *HOMA-AD* homeostatic model assessment-adiponectin
*$p < 0.05$; †$p < 0.10$. Significant values are in italic

our study, since there were significant associations in healthy weight children opposing a low explanatory power in the model for resistin in obese children (R = 10.6%).

In the same context, high concentrations of visfatin are associated with the area of visceral adipose tissue, obesity, and metabolic syndrome in children [18, 40, 42–44]. In this study, analysis confirmed the relationship between visfatin concentrations and central obesity through association with WHtR and TAI in obese children. This model reveals a significant interaction between trunk adiposity index and adiponectin in the variation of average visfatin concentration. Currently, it is recognized that adiponectin concentrations are inversely proportional to body fat [45, 46]. A possible explanation for this interaction may be attributed to reduction of adiponectin levels by increasing trunk fat in obese children, causing the variation of visfatin concentrations.

Table 3 Multiple regression analyses between visfatin level with anthropometric and biochemical variables in schoolchildren 6–10 years of Nova Era, Minas Gerais, Brazil, 2009

	β-coefficient ± SE	*p* value	Model R^2	VIF
Constant	0.871 ± 0.409	*0.036**	20,00%	
Resistin (ng/mL)	0.186 ± 0.060	*0.003**		1.0
Triceps skinfold (mm)	0.116 ± 0.037	*0.002**		1.1
HOMA-AD	− 0.018 ± 0.006	*0.008**		1.1
Constant	− 2.691 ± 1.356	0.054	37,70%	
Resistin (ng/mL)	0.241 ± 0.075	*0.003**		1.1
Waist/height ratio	4.078 ± 1.913	*0.039**		1.1
Atherogenic index	0.834 ± 0.2854	*0.006**		1.1
HOMA-IR	− 0.334 ± 0.112	*0.005**		1.1
TAI*adiponectin (μg/mL)	0.029 ± 0.014	*0.046**		1.1

Model normal weight: visfatin = β_0 + resistin + triceps skinfold − HOMA-AD. Model obese: resistin + waist/height ratio + atherogenic index − HOMA-IR + trunk adiposity index × adiponectin
Abbreviations: *HOMA-AD* homeostatic model assessment-adiponectin, *HOMA-IR* homeostatic model assessment of insulin resistance, *TAI* trunk adiposity index
*$p < 0.05$. Significant values are in italic

In addition to reducing adiponectin concentration, childhood obesity is associated with increased C-reactive protein concentration and visceral fat. The C-reactive protein is a non-specific marker of inflammation, and visceral fat has been increasingly demonstrated as an important endocrine organ involved in the relationship between obesity and inflammation through accumulation of macrophages, which in turn promotes synthesis of these pro-inflammatory cytokines [47]. In our sample, previously published data showed important relationships between C-reactive protein (CRP) and adiponectin with anthropometric variables, and metabolic and body composition which highlighted the role of adipose tissue in the inflammatory process [48].

Authors have shown that resistin controls the pro-inflammatory cytokine cascade [2, 17] and that increased visfatin concentration was related to inflammation in overweight individuals [40, 44, 49]. Such inflammatory signaling significantly alters lipid metabolism in adipose tissue, liver, and skeletal muscle, and the interaction between dyslipidemia and inflammation accelerates the development of atherosclerosis and insulin resistance [50, 51].

Analysis of the adjusted models strengthened the role of adipokines in lipid metabolism in our study. Both resistin and visfatin were associated with atherogenic index and resistin also was associated with cholesterol concentration LDL-C. In contrast with results obtained in children, some studies have shown visfatin positively associated with HOMA-IR [43, 52] but in our study showed negative association between these two variables. Araki et al. found no difference in visfatin concentrations in a group of obese children stratified by HOMA-IR and hyperinsulinemia, suggesting that visfatin is a marker for visceral adipose tissue (VAT) accumulation but not a marker for insulin metabolism [18].

Although some studies have shown an insulin-mimetic property of visfatin, other reports consider visfatin to be a molecule homologous to nicotinamide phosphoribosyl transferase (NAMPT). The NAMPT is an enzyme that triggers the synthesis of nicotinamide adenine dinucleotide (NAD), which is essential for the functioning of cells β-pancreaticin insulin secretion [2]. As in both cases visfatin may promote reduction of blood glucose, a negative association is suggested between visfatin and insulin resistance [53, 54], as observed in our results.

In the case of resistin, it was observed that our model presents an adipokine association with the interaction between conicity and HOMA-AD indexes. This result demonstrates the importance of resistin in insulin resistance and this is obesity-dependent, since the conicity index estimates central

obesity [55, 56] and the HOMA-AD index was positively associated with levels of resistin in obese children.

However, the fact that both resistin and visfatin are associated with insulin resistance should be analyzed with caution, since no association was found between concentrations of resistin and visfatin with fasting glucose or QUICKI. According to a study by Keskin et al., HOMA-IR index is more reliable than QUICKI to measure insulin resistance in children [57], and this is a likely explanation for the absence of association with QUICKI in our sample. In addition, several discrepancies between theories described in various studies maybe due to different characteristics of the studied populations or confounding factors such as sex and age, and use of different laboratory methods in the assessment of adipokines.

Thus, these findings add evidence to underscore the importance of early identification of risk factors for cardiometabolic diseases and insulin resistance in order to promote early intervention and improved quality of life in this population. In addition, these results reinforce the importance of conducting studies that infer causality to endorse the use of anthropometric parameters and body composition in the estimation of resistin and visfatin concentrations, thus allowing a more applicable diagnosis in clinical practice.

Conclusion

Our study shows a significant association of anthropometric and metabolic variables with resistin and visfatin concentrations in children. These results suggesting that both adipokines are pro-inflammatory markers and strongly related to central obesity, especially waist-height ratio.

Furthermore, studies are needed to investigate the adipokine receptors, especially their regulation, whereas this could help in understanding the metabolic pathways linking obesity with atherosclerosis and other cardiovascular diseases, and it could determine causal assumptions.

Abbreviations

AC: Arm circumference; AI: Atherogenic index; BF: Body fat; BMI: Body mass index; BMI/A: Body mass index for age; BP: Blood pressure; CI: Conicity index; CRP: C-reactive protein; CVD: Cardiovascular disease; DM2: Type 2 diabetes; HDL-C: High-density lipoprotein cholesterol; HOMA-AD: Homeostatic model assessment-adiponectin; HOMA-IR: Homeostatic model assessment for insulin resistance; LDL-C: Low-density lipoprotein cholesterol; NAD: Nicotinamide adenine dinucleotide; NAMPT: Nicotinamide phosphoribosyl transferase; PI: Ponderal index; QUICKI: Quantitative insulin sensitivity check index; SSF: Subscapular skinfold; TAI: Trunk adiposity index; TBI: Tetrapolar bioelectrical impedance; TC: Total cholesterol; TSF: Triceps skinfold; VAT: Visceral adipose tissue; VIF: Variance inflation factor; VLDL-C: Very-low-density lipoprotein cholesterol; WC: Waist circumference; WHtR: Waist-to-height ratio

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Availability of data and materials

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

Authors' contributions

NFS wrote and edited the manuscript and is responsible for the data analysis and interpretation; ALGD wrote and edited the manuscript and is responsible for the interpretation; MRG and VCF wrote and edited the manuscript; FLPO wrote and edited the manuscript and is responsible for the statistical analysis and data interpretation; SNF wrote and edited the manuscript and the conducted field work and data analysis and interpretation. All authors read and approved the final manuscript.

Ethics approval and consent to participate

The study followed the principles of the Declaration of Helsinki and was approved by the Research Ethics Committee of the Federal University of Ouro Preto under the number 2007/93. A written consent was signed in duplicate and obtained from those responsible.

Consent for publication

Not applicable

Competing interests

The authors declare that they have no competing interests.

Publisher's Note

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