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Association between dyslipidemia and anthropometric indicators in adolescents

S. C. Vieira Cunha Lima[1], C. Oliveira Lyra[1], L. Galvão Bacurau Pinheiro[1], P. R. Medeiros de Azevedo[2], R. F. Arrais[3] and L. F. Campos Pedrosa[4]

[1]Health Sciences Post Graduate Program, Federal University of Rio Grande do Norte, Rua Cordeiro de Farias, s/nº - Petrópolis, CEP: 59010-180, Natal, RN, Brazil. [2]Department of Statistics, Federal University of Rio Grande do Norte, Campus Universitário, Lagoa Nova, CEP 59078-970, Natal, RN, Brazil. [3]Department of Pediatrics, Pediatric Hospital, Federal University of Rio Grande do Norte. Rua Gal. Cordeiro de Farias, s/nº - Petrópolis, CEP 59012-570, Natal, RN Brazil. [4]Department of Nutrition, Federal University of Rio Grande do Norte, Rua Cordeiro de Farias, s/nº - Petrópolis, CEP: 59010-180, Natal, RN, Brazil.

Abstract

The dyslipidemia associated with excess weight is a risk profile global call for cardiovascular disease (CVD). The aim of this study was to investigate the association between dyslipidemias and other risk factors for cardiovascular diseases (CVD) in adolescents, considering sexual maturation. A cross-sectional study was carried out with 432 adolescents from public schools, aged 10-19 years. The correlations between the variables from the lipid profile and the Body Mass Index (BMI), waist circumference (WC), waist-to-height ratio (WHtR), sexual maturation, familial history and maternal education were evaluated using Pearson's correlation coefficient. Low high-density lipoprotein cholesterol (HDL-C) was the most prevalent dyslipidemia (50.5%), regardless of gender. There were significant correlations between triglycerides and BMI ($r = 0.30$, $p<0.01$), WC ($r = 0.32$, $p < 0.01$) and WHtR ($r = 0.33$, $p < 0.01$). The linear model, which took into consideration sexual maturation, age and BMI, explain about 1 to 10.4% of the lipid profile variation. The low HDL-c was the most prevalent dyslipidemia in all adolescents and hypertriglyceridemia was most prevalent in overweight adolescents. Associations between dyslipidemias and anthropometric indicators (BMI and RCA) detected in this study can generate the hypothesis of the risk factors for CVD in adolescents.

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Correspondence: Severina Carla Vieira Cunha Lima.
Health Sciences Post Graduate Program.
Federal University of Rio Grande do Norte.
Rua Cordeiro de Farias, s/n - Petrópolis.
CEP: 59010-180, Natal, RN, Brazil.
E-mail: scarla@ufrnet.br

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ASOCIACIÓN ENTRE LA DISLIPEMIA Y LOS INDICADORES ANTROPOMETRICOS EN ADOLESCENTES

Resumen

La dislipidemia asociada con el exceso de peso es un perfil de riesgo de alcance mundial para la enfermedad cardiovascular (ECV). El objetivo de este estudio fue investigar la asociación entre la dislipemia y otros factores de riesgo para enfermedad cardiovascular (ECV) en los adolescentes en edad púber. Se realizó un estudio transversal con 432 adolescentes de escuelas públicas, con edades entre 10-19 años. Estudiando las correlaciones entre las variables del perfil lipídico y el índice de masa corporal (IMC), la circunferencia de cintura (CC), la cintura/altura (RCA) y la maduración sexual. Los antecedentes familiares y la educación de la madre se evaluaron mediante el coeficiente de correlación de Pearson . La dislipidemia com bajos niveles de HDL-C fue más frecuente (50,5%), independientemente del género. Se observaron correlaciones significativas entre los triglicéridos y el IMC ($r = 0,30$, $p < 0,01$), CC ($r = 0,32$, $p < 0,01$) y RCA ($r = 0,33$, $p < 0, 01$). El modelo lineal, teniendo en cuenta la maduración sexual, la edad, y el IMC, explicó entre el 1 y el 10,4% de la variación del perfil lipídico. Los bajos niveles de HDL-C fueron la dislipidemia más frecuente en todos los adolescentes y la hipertrigliceridemia en adolescentes con sobrepeso. Las asociaciones entre las dislipidemias y los indicadores antropométricos (índice de masa corporal y RCA) detectado en este estudio, pueden generar hipótesis acerca de los factores de riesgo de ECV en los adolescentes.

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Palabras clave: *Dislipidemias. Obesidad. Factores de riesgo.*

Introduction

There are several risk factors for coronary diseases, which can act independently or together. Among the most common are arterial hypertension, smoking, a sedentary lifestyle, diabetes, obesity, dyslipidemias, and a positive familial history of cardiovascular disease (CVD). The precocity of these factors signals the need to develop prevention and intervention strategies in pediatric populations.[4,11]

In the northeast region of Brazil, an increase in the prevalence of childhood and adolescence obesity has been observed. According to the Household Budget Survey 2002-2003, the prevalence of teenagers with excess body weight was of 11.8% for males and 11.6% for females. Epidemiological surveys performed in different states of this region have described a prevalence of excess body weight from 10.8% to 54.5% in children and adolescents.[3,6]

Studies of Brazilian children and adolescents have also demonstrated an elevated prevalence of dyslipidemias that is associated with being overweight or obese, both with and without a familial history of premature cardiovascular events. The modifications in lipid metabolism that trigger changes in plasma lipoprotein concentrations are due to genetic or ambient factors.[7,12,19]

Waist circumference (WC) and waist-to-height ratio (WHtR) during childhood are predictors of the development of risk factors for CVD. Visceral adiposity has a strong impact on CVD due to its association with dyslipidemias, arterial hypertension, insulin resistance and diabetes. High plasma triglycerides (TG) and low concentrations of high-density lipoprotein cholesterol (HDL-C) are among the alterations observed in the lipid profile that are primarily related to central fat distribution.[21]

Epidemiological investigations into dyslipidemias in adolescents, particularly considering the elevated concentration of total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C) and low concentrations of HDL-C, provide a basis for the prevention of atherosclerotic disease and a reduction of elevated mortality rates.[4,15]

The alterations in the lipid profile observed during puberty are more evident in males than in females due to hormonal influences. The lipid levels are elevated at about 9 to 10 years of age and lowered thereafter, with some differences between genders.[5]

The objective of this study was to verify the association between dyslipidemias and other risk factors for cardiovascular diseases in adolescents, considering sexual maturation.

Methods

Study design and sampling

A cross-sectional study was carried out with adolescents, aged 10 to 19 years, from 21 public schools in the city of Natal, Brazil, between 2007 and 2008.

The sampling plan was defined by stratified random sampling in two stages, based on a target population of 39,920 students, while considering the four districts of the city: N_{north} = 19,270, N_{south} = 4,128, N_{west} = 3,728 and N_{east} = 12,794. The estimated prevalence for the changes in the lipid profile in each district, according to a pilot study involving four schools was $_{north}$ = 35%, $_{south}$ = 33%, $_{west}$ = 9% and $_{east}$ = 7%. The maximum estimation error was 4%, and the prediction was 30% for sample losses, which resulted in a sample size of n = 483 students. Stratified sampling with *Neyman allocation* was used to define sample sizes by district, resulting in the following n_{north} = 285, n_{south} = 63, n_{west} = 34 and n_{east} = 101.

To determine the number of schools, the average number of students per school was considered, assuming that the variance of that number in the four districts was approximately equal. The school sample size obtained was n = 21, and according to proportional allocation, the following numbers of schools were obtained for each stratum: nNorth = 9, nSouth = 3, nEast = 3 and nWest = 6.

The inclusion criteria were that the adolescent be attending school regularly and be between the ages of 10 and 19 years. The exclusion criteria were that adolescents with genetic syndromes associated with obesity or other diseases, those who were pregnant, and those with disabilities or who were using medication that could change the results of biochemical tests.

The familial history of CVD and maternal education were included as descriptive variables. The presence of CVD was defined when the parents had diabetes, arterial hypertension, dyslipidemia or obesity. The education of the mother or caretaker was categorized as illiterate or as possessing a primary education, a secondary education, or a university education.

Anthropometric evaluation

The sampling of all of the anthropometric measurements was performed in duplicate by trained staff in accordance with standardized techniques. To measure weight, we used an electronic scale by Tanita Solar®, model HS301 with a 150 kg capacity and 100 g precision. Height was measured with a portable stadiometer by Altura Exata® (1 mm precision). The classification of BMI-by-age was performed according to cutoff points established by Cole et al.[8] (2000). The adolescents classified as having excess body weight were considered to be overweight or obese.

Waist circumference was measured at the midpoint between the last rib and the iliac crest, using anthropometric tape, and we referred to it as the waist measurement, conforming literature.[10] We evaluated the WHtR and the cutoff points were set into two categories, elevated and normal.[21]

Table I
Anthropometric and of lipid profile variables of adolescents, according to gender - Natal, Brazil, 2008

Variables	*Male (n = 223) Mean (SD)*	*Female (n = 209) Mean (SD)*	*Total (n = 432) Mean (SD)*
BMI (kg/m²)	18.0 (3.3)	18.6 (3.5)	18.3 (3.4)
Waist Circumference (cm)[a]	65.1 (9.0)	66.2 (8.6)	65.6 (8.8)
Waist/Height Ratio (cm)[b]	0.5 (0.1)	0.5 (0.1)	0.5 (0.1)
Total Cholesterol	149.1 (29.4)	148.0 (30.4)	148.5 (29.9)
LDL-cholesterol (mg/dL)	87.9 (27.2)	86.1 (28.8)	87.0 (28.0)
HDL-cholesterol (mg/dL)	44.2 (8.1)	44.7 (7.7)	44.4 (7.9)
Triglycerides (mg/dL)	83.5 (42.1)	84.5 (37.7)	84.0 (39.9)
Non-HDL Cholesterol (mg/dL)	104.9 (27.9)	103.3 (29.5)	104.1 (28.7)
TC:HDL Ratio (mg/dL)	3.5 (0.8)	3.4 (0.8)	3.4 (0. 8)
LDL:HDL Ratio (mg/dL)	2.2 (0.7)	2.0 (0.7)	2.0 (0.7)

[a,b]For these variables, the number of subjects does not add up to the total, and sampling equal to 191 and 187 for male and female genders , respectively.
Mean differences were tested by the bilateral t-test (not significant) for male and female genders.

Pubertal stage

An assessment of the pubertal stage was performed by the medical team according to the Tanner method, which considers breast development[13] and genitalia,[14] respectively for girls and boys. Classification criteria were defined as prepubescent: stage I, early pubescent: stages II and III and late pubescent: IV and V.

Lipid profile evaluation

After fasting for 10-12 hours, 5 mL of blood were collected by the technical staff at the private clinic laboratory "Centro de Patologia Clínica." To obtain LDL-C, the equation from Friedewald was used. TC, LDL-C, HDL-C and TG were classified according to Brazilian recommendations.[22] The Castelli indices I (TC/HDL) and II (LDL/HDL), were established considering Elcarte et al[9] (1993). For Non-HDL Cholesterol, the values set by Srinivasan et al.[23] (2002) were used. The cutoff points were TC < 150 mg/dL; LDL-C < 100 mg/dL; HDL-C ≥ 45 mg/dL; TG < 100 mg/dL; TC/HDL > 3.5 mg/dL; LDL/HDL > 2.2 mg/dL and Non-HDL Cholesterol < 123 mg/dL. When values of the lipid profile or the relationships between them were different from the recommended levels, they were classified as altered.

The study was approved by The Research and Ethics Committee of the UFRN, according to doc No. 112/06. All participants signed the consent form.

Statistical analysis

The database was built using EPI-INFO software, version 6.04, with pre-coding of variables and statistical analysis in Statistica, version 7.0. Descriptive analyses included mean and standard deviation for continuous variables, and proportion for categorical variables. Mean differences were tested by the bilateral t-test and, prevalence, by the chi-squared test. A prevalence ratio (PR) and the respective confidence interval of 95% (95% CI) were used to verify the associations between anthropometric variables according to gender. A 95% CI was considered significant when the value 1.0 was not found in the range. The correlation between variables of the lipid profile and risk factors for CVD was performed using Pearson's correlation coefficient. Sexual maturation variable was divided into two: sexual maturation 1 and 2, and considered for each one, two categories: sexual maturation 1 (SexMat1), prepubital, yes or no; and sexual maturation 2 (SexMat2), initial puberty, yes or no. The chi-squared test was used to test the independence between excess weight and variables of the lipid profile. Relations between plasma lipids and lipoproteins and other metabolic variables were determined using a multivariate linear regression model, with metabolic variables as dependent variables and sexual maturation, age and BMI as independent variables. All statistical analyses were considered significant when the p-value was less than 5%.

Results

Anthropometric, clinical and biochemical data we obtained from 432 adolescents, (51.6%) were male and (48.4%) were female, with average ages of 11.8 ± 1.4 and 11.9 ± 1.4 years, respectively. The values of the anthropometric parameters and the lipid profile were not different according to gender (table I).

The alterations in the lipid profile were registered in up to 50.5% of the adolescents. Low HDL-c values were

Table II
Clinical, anthropometric and lipid profile characteristics of adolescents, according to gender - Natal, Brazil, 2008

Variables	Categories	Male		Female		Total		PR	C195%
		n	%	n	%	n	%		
Nutritional Status	Normal	194	(87.0)	174	(83.3)	368	(85.2)	1.00	–
	Overweight	19	(8.5)	26	(12.4)	45	(10.4)	0.69	0.39-1.20
	Obese	10	(4.5)	9	(4.3)	19	(4.4)	1.00	0.41-2.40
Central Obesity p90[a]	Normal	179	(93.7)	171	(91.4)	350	(92.6)	1.00	–
	Elevated	12	(6.3)	16	(8.6)	28	(7.4)	0.73	0.36-1.51
Waist/Height Ratio[b]	Normal	173	(90.6)	168	(89.8)	341	(90.2)	1.00	–
	Elevated	18	(9.4)	19	(10.2)	37	(9.8)	0.93	0.50-1.71
Sexual Maturation[c]	Prepubertal	97	(54.8)	20	(10.9)	117	(32.5)	1.00	–
	Initial Puberty	51	(28.8)	77	(42.1)	128	(35.6)	0.43	0.34-0.55
	Final Puberty	29	(16.4)	86	(47.0)	115	(31.9)	0.28	0.20-0.40
Family History[d]	Healthy	97	(58.4)	67	(43.8)	164	(51.4)	1.00	–
	History of CVD	69	(41.6)	86	(56.2)	155	(48.6)	0.74	0.59-0.93
Total Cholesterol	Acceptable	118	(52.9)	122	(58.4)	240	(55.6)	1.00	–
	Altered	105	(47.1)	87	(41.6)	192	(44.4)	0.91	0.77-1.07
LDL-cholesterol	Acceptable	156	(70.0)	149	(71.3)	305	(70.6)	1.00	–
	Altered	67	(30.0)	60	(28.7)	127	(29.4)	0.98	0.87-1.11
HDL-cholesterol	Acceptable	106	(47.5)	108	(51.7)	214	(49.5)	1.00	–
	Unsatisfactory	117	(52.5)	101	(48.3)	218	(50.5)	0.92	0.76-1.11
Triglycerides	Acceptable	168	(75.3)	151	(72.2)	319	(73.8)	1.00	–
	Altered	55	(24.7)	58	(27.8)	113	(26.2)	1.04	0.93-1.17
Non-HDL Cholesterol	Acceptable	165	(74.0)	158	(75.6)	323	(74.8)	1.00	–
	Altered	58	(26.0)	51	(24.4)	109	(25.2)	0.98	0.88-1.09
TC: HDL Ratio	Acceptable	124	(55.6)	123	(58.9)	247	(57.2)	1.00	–
	Altered	99	(44.4)	86	(41.1)	185	(42.8)	0.95	0.80-1.11
LDL: HDL Ratio	Acceptable	134	(60.1)	134	(64.1)	268	(62.0)	1.00	–
	Altered	89	(39.9)	75	(35.9)	164	(38.0)	0.94	0.81-1.09

PR = Prevalence Ratio. [a,b,c,d] For these variables, the number of subjects does not add up to the total due to a small number of subjects with missing data. TC/HDL: total cholesterol: HDL cholesterol ratio; LDL/HDL: LDL-cholesterol: HDL cholesterol ratio.

the most prevalent lipid abnormality, followed by hypercholesterolemia. Among males, there was a lower probability of being in the early pubescent stage or in the late pubescent stage. There was no association between gender and nutritional status, centralized obesity, elevated WHtR, or any of the dyslipidemias (table II).

Evaluating the influence of excess body weight in the alterations in the lipid profile, we found a higher prevalence of hypertriglyceridemia and an elevated TC/HDL ratio in overweight adolescents of both genders (table III).

In the univariate analysis, TC, LDL-C, TC/HDL ratio, LDL/HDL ratio ($p < 0.01$) and Non-HDL Cholesterol ($p < 0.05$) showed a positive correlation with familial history. The triglycerides showed a positive correlation with BMI ($p < 0.01$), WC ($p < 0.01$) and WHtR ($p < 0.01$). Non-HDL Cholesterol ($p < 0.05$) and TC/HDL ratio ($p < 0.01$) correlated with WHtR (table IV).

The multivariate linear regression analysis (table V) showed a significant inverse association between TC and sexual maturation (β-12.01 mg/dL, $p < 0.025$) and LDL-C (β-13.71 mg/dL, $p < 0.006$), adjusted by age and BMI. The model, which considered sexual maturation, age and BMI, explained about 2 to 10.4% of the variation in the alterations in the lipid profile in males and about 1 to 4.5% in females.

Discussion

An elevated prevalence of dyslipidemias was found in our study in adolescents with and without excess weight, independent of gender, similar to those in the findings of Gama et al.[11] (2007) and Vieira et al.[24] (2009); and higher than those observed in other studies carried in Brazil.[12,18]

The prevalence of excess body weight among the adolescents studied was similar to that seen in other epidemiological studies from the region, which did not show significant differences between genders. During childhood, this prevalence has a tendency vary as a result of the population analyzed, the cutoff point used for diagnosis, and the socio-economical status and age group studied.[3,6]

Hypercholesterolemia and elevated concentrations of LDL-C in adolescents suggest a genetic susceptibility. The elevation of plasma TC has been reported in

Table III
Prevalence (%) of lipid profile alteration by BMI of adolescents, according to gender - Natal, Brazil, 2008

	Male (n = 223)			*Female (n = 209)*		
	Overweight (n = 29)	*Normal Weight (n = 194)*	*p*	*Overweight (n = 35)*	*Normal Weight (n = 174)*	*p*
Hyper TC	55.2	45.9	ns	54.3	39.1	ns
Hyper LDL-C	27.6	30.4	ns	25.7	29.3	ns
Low HDL-C	51.7	52.6	ns	62.9	45.4	ns
Hyper TG	58.6	19.6	< 0.01	57.1	21.8	< 0.01
Non-HDL Cholesterol elevated	34.5	24.7	ns	34.3	22.4	ns
TC/HDL elevated	62.1	41.8	< 0.05	60.0	37.4	0.01
LDL/HDL elevated	44.8	39.2	ns	42.9	34.5	ns

p values for the chi-squared test. ns: not significant.
TC: total cholesterol; LDL-C: LDL-cholesterol; HDL-C: HDL-cholesterol; TG: triglycerides; TC/HDL: total cholesterol: HDL cholesterol ratio; LDL/HDL:LDL-cholesterol: HDL cholesterol ratio.

Table IV
Correlation coefficient between lipid profile and clinical and anthropometric variables of adolescents (n = 262) - Natal, Brazil, 2008

Variables	*TC*	*LDL-C*	*HDL-C*	*TG*	*Non-HDL Cholesterol*	*TC/HDL*	*LDL/HDL*
SexMat1	0.04	0.11	-0.13[a]	-0.11	0.08	0.14[a]	0.16[b]
SexMat2	-0.08	-0.12	0.01	0.12[a]	-0.09	-0.08	-0.12
BMI	0.13[a]	0.05	-0.01	0.30[b]	0.13[a]	0.13[a]	0.06
WC	0.09	0.01	-0.033	0.32[b]	0.10	0.12	0.04
WCHtR	0.10	0.05	-0.12[a]	0.33[b]	0.14[a]	0.20[b]	0.12
FamH	0.19[b]	0.18[b]	-0.01	0.11	0.20[a]	0.16[b]	0.15[b]
Mat. Edu 1	-0.10	-0.13[a]	0.20[a]	-0.12[a]	-0.16[b]	-0.23[b]	-0.20[b]
Mat. Edu 2	0.13[a]	0.15[a]	-0.17	0.15[a]	0.18[b]	0.24[b]	0.21[b]

[a]p < 0.05; [b]p < 0.01.
TC: total cholesterol; LDL-C: LDL-cholesterol; HDL-C: HDL-cholesterol; TG: triglycerides; TC/HDL: total cholesterol: HDL cholesterol ratio; LDL/HDL:LDL-cholesterol:HDL cholesterol; SexMat1: sexual maturation 1 (prepubital, yes or no); SexMat2: sexual maturation2 (initial puberty, yes or no); BMI: Body Mass Index; WC: Waist circumference; WHtR: Waist/Height Ratio; FamH: family history; Mat. Edu 1: maternal primary education and Mat. Edu 2: maternal secondary or university education.

children and adolescents with[16] and without a familial history of premature cardiovascular events.[19]

The level of plasma TC during childhood can be explained by the *tracking* phenomenon seen during growth and development. Moreover, it has been observed that when certain risk factors are seen together *(clustering effect)* in this age group, those children are at an increased risk of an early occurrence of atherosclerosis.[4]

We observed that HDL-C was inversely associated with body fat distribution and that higher TC was related to body fat distribution. The association between increased body weight and hypertriglyceridemia and low HDL-C has been described in children.[12] The PROCAM study proposed a score to calculate the risk of acute cardiovascular events. Traditional factors are considered, ranked in order of importance: age, LDL-C, smoking, low HDL-c, systemic arterial hypertension (SAH), family history (FH), diabetes mellitus, and hypertriglyceridemia.[1]

The association between hypertriglyceridemia and centralized obesity can be explained by the increased number and size of adipocytes in the abdominal region, which promotes insulin resistance and intensifies the release of free fatty acids (FFA) into the plasma, which provide a substrate for triacylglycerol synthesis in the liver, leading to increased hepatic release of TG rich very low density lipoprotein (VLDL) particles into the plasma.[20]

In this study, the average concentration of Non-HDL Cholesterol was similar to that reported by Giuliano et al.[12] (2005) and was greater than that observed by the Bogalusa Study.[23] Data from the Bogalusa Heart Study indicated that the altered levels of Non-HDL Cholesterol and LDL-C persist with time and are indicators for dyslipidemia in adulthood.[23]

The negative association between sexual maturation and concentrations of TC and LDL-C, independent of age and BMI, demonstrate that gender and pubertal development influence the lipid profile, as described in

Table V
Estimated coefficientes ($\hat{\beta}$), p value and R^2 of regressions between lipid profile (dependent variables) and sexual maturation, adjusted for age and BMI, according to gender - Natal, Brazil, 2008

Variables	Male (n = 177)			Female (n = 183)		
	$\hat{\beta}$ (CI95%)	p	R^2 (%)	$\hat{\beta}$ (CI95%)	p	R^2 (%)
TC						
SexMC	-12.01 (-22.38--1.63)	0.025	4.2	-4.36 (-18.90-10.18)	0.557	0.8
Age	0.33 (-3.12-3.79)	0.850		-0.34 (-3.74-3.04)	0.842	
BMI	0.93 (-0.43-2.28)	0.181		0.78 (-0.55-2.11)	0.251	
LDL-C						
SexMC	-13.71 (-23.38--4.04)	0.006	5.8	-9.03 (-22.75-4.69)	0.199	1.3
Age	0.35 (-2.87-3.56)	0.833		0.16 (-3.04-3.36)	0.924	
BMI	0.17 (-1.09-1.43)	0.795		0.66 (-0.59-1.91)	0.304	
HDL-C						
SexMC	-0.51 (-3.44-2.42)	0.734	2.4	2.37 (-1.41-6.14)	0.221	2.2
Age	0.92 (-0.06-1.90)	0.066		0.12 (-0.76-1.00)	0.782	
BMI	-0.03 (-0.42-0.35)	0.859		-0.30 (-0.64-0.05)	0.092	
TG						
SexMC	11.54 (-3.42-26.50)	0.132	10.4	10.74 (-7.57-29.04)	0.252	4.5
Age	-4.57 (-9.54-0.41)	0.074		-2.86 (-7.13-1.41)	0.190	
BMI	3.96 (2.01-5.91)	0.000		2.14 (0.47-3.81)	0.013	

TC: total cholesterol; LDL-C: LDL-cholesterol; HDL-C: HDL-cholesterol; TGL: triglycerides; SexMC:– sexual maturation category, BMI: Body Mass Index.

other studies.[5,22] The findings regarding the positive effects that BMI has on the level of TG, independent of sexual maturation and age for both genders, are already evidenced.[17]

Lipid and lipoprotein concentrations changes during growth and development, showing two phases of expressive increase: up to 2 years old and during pubertal development.[5] In our study, which considers sexual maturation as a control variable, we did not find an influence in the HDL-C ratio, probably due to the age group of the adolescents. Since it was the most prevalent dyslipidemia from the study, we highlight its important role, in combination with hypertriglyceridemia, in the pathogenesis of the metabolic syndrome and as an important risk factor for CVD during an adult's life.[2]

Some limitations deserve mention. First, other risk factors for CVD that were not described in this study, such as smoking, alcoholic beverages, physical activity and hypertension. Second, the results of cross-sectional studies should be viewed with caution, since they are subject to reverse causality. However, these considerations do not invalidate the findings of this study from contributing to the important investigation of nutritional status and dyslipidemias in adolescents.

Conclusions

Low HDL-C and hypertriglyceridemia associated with excess body weight and centralized obesity represent a higher risk for CVD in these adolescents. These findings demonstrate the importance of establishing an early diagnosis of the dyslipidemias, mainly if it is already associated to another risk factor for CVD, such as obesity.

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