Artículo Original / Original Article

Influence of weight gain on the lipid profile of adolescents

Influencia de la ganancia de peso sobre el perfil lipídico en adolescentes

ABSTRACT

The objective of this study was to assess the influence of weight gain on the lipid profile of 135 adolescents between 10 - 14 years at baseline and 15 - 19 years at follow-up, enrolled in public schools in Recife, Brazil. The results showed that a BMI z-score correlated with triglycerides (TG) and with high density triglyceride lipoprotein ratio (TG/HDL-c) in males. In females, high z-score correlated with total cholesterol (TC) and low density lipoprotein (LDL-c). In males, for each unit increase in z-score, TG increased by 14.7 mg/dL and the TG/HDL-c ratio increased by 0.4. Among females, TC increased by 9.4 mg/dL, LDL-c increased by 11.6 mg/dL, non-HDL cholesterol increased by 11.8 mg/dL, and HDL-c decreased by 2.3 mg/dL. In males, excessive weight gain was associated with an increase in TG and TG/HDL-c; in females, it was associated with a higher increase in TG/HDL-c and non-HDL cholesterol. However, z-score variation can be a good predictor of lipid profile changes, even in those that are within the normal range.

Keywords: Adolescent; Body mass index; Cardiovascular diseases; Dyslipidemia; Weight gain.

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RESUMEN

El objetivo de este estudio fue evaluar la influencia del aumento de peso en el perfil lipídico de 135 adolescentes de edades entre 10 y 14 años de edad al inicio del estudio y de 15 a 19 años en el seguimiento. Los adolescentes pertenecían a escuelas públicas de Recife, Brasil. Los resultados mostraron que el alto puntaje z de índice de masa corporal (IMC) correlacionaba con triglicéridos (TG) y con relación de triglicéridos con lipoproteínas de alta densidad (TG/HDL-c) en los hombres. En las mujeres, puntaje z de IMC se correlacionó con CT y lipoproteína de baja densidad (LDL-c). En los hombres, por cada unidad de aumento en el puntaje z, los TG aumentaron en 14,7 mg/dL y la relación TG / HDL-c aumentó en 0,4; en las mujeres, el CT aumentó en 9,4 mg/dL, el LDL-c aumentó en 11,6 mg/dL, el colesterol no HDL aumentó en 11,8 mg / dL y el HDL-c disminuyó en 2,3 mg/dL. En los hombres, el aumento de peso excesivo se asoció con un aumento de TG y TG/HDL-c; en las mujeres, con un aumento mayor en

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TG/HDL-c y colesterol no HDL. Sin embargo, la variación z-score puede ser un buen predictor de cambios en el perfil lipídico, incluso en aquellos que se encuentran dentro del rango normal.

Palabras clave: Adolescente; Aumento de peso; Dislipidemia; Enfermedades cardiovasculares; Índice de masa corporal.

INTRODUCTION

Weight gain during adolescence may result from the intrinsic pattern of human growth, but, on the other hand, may reflect differential access to food or physical activity. Some individuals gain excess weight, consisting mainly of fat mass¹.

Excessive weight gain during the first two decades of

life seems to have an adverse effect on lipid profile, making it more atherogenic. A high body mass index (BMI) has been positively associated with high low-density lipoprotein cholesterol (LDL-c) and triglycerides (TG), and low high-density lipoprotein cholesterol (HDL-c)².

However, proper BMI during this phase does not ensure protection against obesity and its comorbidities. A high prevalence of excess body fat has been found in normal weight adolescents, as well as metabolic changes, such as dyslipidemia, demonstrating that nutritional status does not reflect one's health status³. There is evidence that BMI gain during childhood and adolescence may be associated with the onset of cardiovascular risk factors⁴, and even a small increase in BMI-for-age z-score (BMIz) may increase the risk of cardiovascular diseases (CVD)⁵.

In this context studies have suggested that the proportion of weight gain during childhood and adolescence may be one of the most significant factors to prevent cardiovascular risk compared with a one-time weight measurement⁶. Thus, the present study aimed to assess the influence of weight gain on the lipid profile of a cohort of adolescents from Recife, Brazil.

METHODS

This study stemmed from a cohort designed to study excess weight and dyslipidemia in students from the city of Recife⁷. The sample consisted of 135 male and female students aged 10 to 14 years recruited from October to December 2007 and reassessed in 2012. The following variables were assessed at baseline and follow-up: anthropometry, biochemical markers of health, food intake, lifestyles, socioeconomic status, and demographics.

The sample size was calculated considering an α error of 5.0%, a β error of 10%, a 95% confidence level (95%CI), and a standard deviation (SD) of 10 based on the smallest variation of the lipid fraction of 4 mg/dL for HDL-c found by Weiss et al⁶. using the formula $(Z_{\alpha/2} + Z_{\beta/2})^2 \times 2 \times S^2/d^2$, resulting in 132 adolescents. The sample size was increased by 20% to compensate for losses, totaling 159 adolescents. The initial sample consisted of 142 adolescents and the follow-up, of 135 adolescents. Students who had baseline and follow-up anthropometric and biochemical data were included. The variables used different sample sizes in the statistical analyses because the number of respondents varied and some biochemical tests were lost.

Five mL of blood were collected in dry flasks after a 12-hour fast. The blood samples were transported in Styrofoam coolers containing reusable ice cubes. Analyses were performed no more than two hours after collection. Blood serum was separated from red blood cells by centrifugation and frozen for later analysis. The samples were processed and the serum analyzed by enzymatic method. LDL-c was estimated by the formula proposed by Friedewald: $LDL-c = TC - (HDL-c + TG) / 5$ for TG values below 400 mg/dL. Any change in the lipid fraction was defined as dyslipidemia. The following values provided by

the V Brazilian Guideline for Dyslipidemia and Prevention of Atherosclerosis⁸ were used: $TC \geq 170$ mg/dl; $LDL-c \geq 130$, $TG \geq 130$ mg/dl, and $HDL-c \leq 45.0$ mg/dl. The TG/HDL-c ratio was considered high when ≥ 2.50 . The non-HDL-c fraction was calculated by subtracting HDL-c from TC: $TC - HDL-c$, and considered high when > 149 mg/dL⁹.

Anthropometric assessment consisted of measuring weight and height twice and taking the average. Measurements with differences greater than 100g and 0.5 cm were discarded. The measurements were performed as originally recommended by Lohman et al. (1998). Weight was obtained from the OMRON (Full Body Sensor) Model HBF 510 W digital scale with capacity of 150 kg and accuracy of 100 g. Adolescents were measured barefoot and in light clothes and were asked to stand in the center of the scale platform with feet together and arms hanging loosely at the sides. Height was measured using the Altura Exata stadiometer with a length of 2.15 m and accuracy of 1 mm.

Nutritional status was classified according to BMIz, calculated by WHO-Anthro Plus 2007 as recommended by the WHO. No excess weight was defined as $BMIz < +1$ and excess weight, as $BMIz \geq +1$. BMI gain in z-scores was given by the difference between the BMIz at follow-up and baseline. Excessive weight gain was defined as weight gain $\geq$ the 75th percentile of the weight gain curve of the study population.

Physical activity was assessed by a questionnaire validated by Florindo et al.¹⁰ Individuals were considered active when they practiced physical activity for 300 or more minutes/week, and inactive when they practiced physical activity for less than 300 minutes/week, as recommended by Pate et al.¹¹. Participants were considered sedentary when the total time spent on sedentary activities (watching television, playing video games, and using a computer) was ≥ 4 hours and 30 minutes/day, as recommended by Suñé et al.¹².

Food habits were investigated using a semiquantitative food frequency questionnaire validated by Slater et al.¹³ and adapted for the present study. The foods were categorized into two groups according to the Brazilian Food Guide¹⁴: group I – fresh foods, which consisted of fruits and vegetables (FV), considered cardioprotective; and group II – ultra-processed foods, which consisted of soda, cookies, and packaged snacks, among others, considered risks for CVD.

Consumption of foods that protect against and promote cardiovascular risk was defined by the median number of servings consumed by the adolescents, since there is no recommended intake for ultra-processed foods, which should be avoided.

Alcohol intake and smoking status were considered positive when these items were consumed, regardless of amount and duration.

Socioeconomic level was determined by the “Critérios de Classificação Econômica do Brasil” (Brazilian Criteria for Socioeconomic Classification) developed by the Brazilian

Association of Anthropology and Brazilian Association of Survey Companies (ABEPE)¹⁵, which classify individuals in the following socioeconomic classes from highest to lowest purchasing power: A1, A2, B1, B2, C1, C2, D, and E. Socioeconomic class was then grouped as follows: high (categories A1, A2, and B1) and low (categories B2, C1, C2, D, and E).

Statistical analyses were performed using the software Statistical Package for the Social Sciences for Windows (SPSS Inc; Chicago, IL, USA) Student version 13.0. The distribution of the continuous variables was tested by the Kolmogorov-Smirnov test. Variables with normal distribution were expressed as means and SDs, and those without normal distribution were expressed as medians and interquartile ranges. In the description of proportions, binomial distribution was close to normal distribution using a confidence interval of 95%. The proportions of paired data were compared by the McNemar test. The means of paired variables were compared by the paired Student t-test or Wilcoxon test for the variables without normal distribution, and by the Student t-test or Mann-Whitney U test for unpaired variables. Pearson correlations and simple linear regression assessed the association between variation in BMI_z and lipid profile. Analysis of variance (ANOVA) and the Bonferroni post-hoc test compared the means of the tertiles of BMI_z variation, and the Kruskal-Wallis test compared variables without normal distribution. The null hypothesis was rejected at a 5.0% significance level.

The study was approved by the Research Ethics Committee of the Instituto Materno Infantil de Pernambuco (CEP/IMIP Registration number 1.024/07) and by the Research Ethics Committee of the University Hospital Lauro Wanderley of the Federal University of Paraíba, CAAE number 65270.000.126-10. Guardians signed an informed consent form before adolescents were enrolled in the study.

RESULTS

A total of 135 adolescents were studied, 62.2% [95%CI 53.4–70.4] female. They had a median age of 11 years [IQR: 10–12] at baseline. At follow-up, the median age was 15 years [IQR: 14–16], mean weight was 57.0 ± 13.2 kg, and BMI_z was 0.000 ± 1.30 , which did not differ by sex. Nearly 100% of the sample had low socioeconomic status.

The prevalence of dyslipidemia increased in males ($p < 0.001$), but did not change in females. Most adolescents were normal weight. In females, the prevalence of overweight decreased and normal weight increased ($p = 0.035$) (Table 1). A total of 23.0% [95%CI: 16.1–30.9] of adolescents experienced a change in their nutritional status.

The prevalence of active individuals increased in males and females ($p = 0.023$), and sedentary behavior did not change (Table 1). Approximately 90.4% of the adolescents did not consume alcohol and none smoked.

Males decreased their intake of protective foods ($p < 0.001$). In females, the percentile distribution of protective food intake in 2012 decreased significantly in comparison

with that of 2007 ($p = 0.003$) (Table 1).

In males, HDL-c decreased ($p = 0.008$). In females, TG ($p = 0.002$) and TG/HDL-c ratio ($p < 0.001$) decreased and TC ($p = 0.003$), LDL-c ($p = 0.01$), and HDL-c ($p < 0.001$) increased. Also in females, the non-HDL-c also tended to increase ($p = 0.07$) (Table 2). Variation in BMI_z was correlated with lipid profile variation in both sexes. In males, for each unit increase of BMI_z, TG increased by 14.7 mg/dL ($p = 0.006$) and TG/HDL-c ratio increased by 0.400 ($p = 0.003$). In females, TC increased by 9.40 mg/dL ($p = 0.024$), LDL-c increased by 11.6 mg/dL ($p = 0.003$), non-HDL-c increased by 11.8 mg/dL ($p = 0.002$), and HDL-c decreased by 2.30 mg/dL ($p = 0.037$) (Table 3).

When we compared lipid variation between the tertiles of BMI_z variation, we found that in males the TG and TG/HDL-c ratio of the third tertile (gain of +0.16) increased more than those of the first tertile. In females, TC, LDL-c, and non-HDL-c of the third tertile (gain of +0.14) increased more than those of the first tertile (Table 4).

When we compare the lipid profile variation of the groups with and without excess weight gain ≥ 75 th percentile (males +0.25 z and females +0.3 z) we found results similar to those previously observed for males. In women, the TG/HDL-c ratio ($p = 0.031$) and non-HDL-c ($p = 0.035$) increased more, and HDL-c increased less in the group with excess weight gain ($p = 0.006$) (Table 5).

DISCUSSION

According to the latest update of the Brazilian directive on dyslipidemia and prevention of atherosclerosis, the prevalence of dyslipidemia in Brazil ranges from 10.0 to 23.5%, according to region and criterion¹⁶. The prevalence of dyslipidemia in the study sample (70.4%) was higher than the prevalence found by França & Alves¹⁷ (29.7%) and Pereira et al.⁷ (63.8%) in Recife. This finding deserves attention because it indicates that an inappropriate lipid profile, which is one of the most important risk factors for CVD, is already present in adolescents¹⁶. CVD is one of the three main causes of morbidity and mortality in Brazil.

This prevalence increased significantly in males, with no changes in females, which may be related to inadequate diet, nutritional status and weight gain ratio. Most adolescents were normal weight, and females experienced an improvement in nutritional status, which may have been a protective factor. However, the prevalence of excess weight in males and females were 23.5% and 21.9%, respectively, confirming that adolescence is one of the stages of greatest risk for the development of obesity. Weight control at a population level is an important strategy in the control of dyslipidemia among adolescents. Ideally, intensive lifestyle modification should be instituted to achieve a BMI below the 85th percentile¹⁶. This datum is concerning because of the greater probability of this disorder persisting throughout adulthood, increasing the risk of obesity-related diseases.

The prevalence of physically active individuals increased

Table 1. Sociodemographic, dyslipidemia, nutritional status, and lifestyle variables at baseline and follow-up of a cohort of adolescents from Recife, Brazil 2007-2012.

Variables	Males						p-valor*
	n	2007 %	CI _{95%}	n	2012 %	CI _{95%}	
Dyslipidemia							
Yes	32	62.7	[48.0—75.8]	45	88.2	[76.1—95.5]	<0.001*
No	19	37.3	[24.1—51.9]	06	11.8	[4.40—23.8]	
Nutritional status							
With excess weight	14	27.5	[15.8—41.7]	12	23.5	[12.7—37.4]	0.727
Without excess weight	37	72.5	[58.2—84.1]	39	76.5	[62.5—87.2]	
Level of activity							
Little active/inactive	38	74.5	[60.3—85.6]	26	51.0	[36.5—65.2]	0.023*
Active	13	25.5	[14.3—39.6]	25	49.0	[34.7—63.4]	
Sedentary behavior							
≥4,5h	30	60.0	[45.1—73.5]	26	51.0	[36.5—65.2]	0.442
<4,5h	20	40.0	[26.4—54.8]	25	49.0	[34.7—63.4]	
Food Intake							
	n	M	IQR	n	M	IQR	
Protective foods	50	05	03—05	50	04	02—04	<0.001†
Ultra-processed foods	50	04	03—05	50	05	03—05	<0.001†
Females							
Dyslipidemia							
Yes	57	67.9	[58.3—79.2]	50	59.5	[49.5—71.5]	0.311
No	27	32.1	[22.3—43.2]	34	40.5	[29.8—51.7]	
Nutritional Status							
With excess weight	27	32.1	[22.3—43.2]	18	2.40	[13.2—31.7]	0.035*
Without excess weight	57	67.9	[56.7—77.6]	66	78.6	[68.2—86.7]	
Level of activity							
Little active/inactive	76	90.5	[84.7—97.2]	64	76.2	[67.5—86.4]	0.023*
Active	08	9.50	[4.3—18.3]	20	23,8	[15.5—35.1]	
Sedentary behavior							
≥4,5h	44	52.4	[42.2—64.7]	40	47.6	[37.5—60.0]	0.644
<4,5h	40	47.6	[37.5—60.0]	44	52.4	[42.2—64.7]	
Food Intake							
	n	M	IQ	n	M	IQ	
Protective foods	82	04	02—05	82	04	02—04	0.003†
Ultra-processed foods	82	03	03—05	82	05	05—06	<0.001†

Dyslipidemia: change in at least one of the lipid fractions. Little active/inactive: <300 minutes/week of physical activity. *CI95% - 95.0% confidence interval; * McNemar test; † Wilcoxon test; N= 50 for the variable sedentary behavior in males in 2007; n= 50 for the variable food intake in males; n= 82 for the variable food intake in females.

Table 2. Means of the biochemical variables at baseline and follow-up of a cohort of adolescents from Recife/PE, 2007-2012.

Males							
Variables	n	2007 $\bar{X}$	SD	n	2012 $\bar{X}$	SD	p-value
TC	51	147	±33.4	51	143	±34.3	0.296
LDL-c	51	84.6	±30.4	51	81.1	±31.5	0.244
TG	51	91.0 [†]	73.0-108 [§]	51	79.0 [†]	73.0-122 [§]	0.286 [†]
HDL-c	51	45.2	±9.60	51	42.7	±6.90	0.008*
TG/HDL	51	2.10	±0.800	51	2.40	±1.00	0.084
Non-HDL chol	51	102	±32.7	51	101	±33.2	0.756
Female							
TC	82	145	±27.4	82	153	±27.4	0.003*
LDL-c	84	81.8	±23.7	84	88.5	±25.1	0.010*
TG	84	93.0 [†]	73.2-127 [§]	84	82.5 [†]	75.0-102 [§]	0.002*
HDL-c	84	42.8	±8.30	84	46.5	±8.00	<0.001*
TG/HDL	84	2.10 [†]	1.60-3.00 [§]	84	1.80 [†]	1.50-2.18 [§]	<0.001*
Non-HDL chol	82	102	±26.4	82	107	±27.1	0.072

TC – total cholesterol; HDL-c – high-density lipoprotein cholesterol; LDL-c – low-density lipoprotein cholesterol; TG- triglycerides; TG/HDL-c – triglycerides-to-high-density lipoprotein cholesterol ratio; non-HDL cholesterol – difference between TC and HDL-c. [†]median, [§]interquartile range; *Paired Student's t-test; [†]Wilcoxon test. N= 82 for the variables TC and non-HDL cholesterol in females.

Table 3. Correlation and simple linear regressions between lipid profile variation and BMIz in a cohort of adolescents from Recife, Brazil 2007-2012.

Variation in BMIz												
Variables	N	r²	B	Males			N	r²	Females			
				SD	B	p-value*			B	SD	β	p-value*
ΔTC	51	0.000	-1.40	3.30	-0.000	0.677	82	0.060	9.40	4.10	0.200	0.024*
ΔLDL-c	51	0.000	-1.80	2.90	-0.000	0.534	84	0.100	11.6	3.70	0.300	0.003*
ΔTG	51	0.140	14.7	5.10	0.300	0.006*	84	0.000	1.90	6.30	0.000	0.765
ΔHDL-c	51	0.020	-1.00	0.800	-0.100	0.230	84	0.050	-2.30	1.10	-0.200	0.037*
ΔTG/HDL-c	51	0.160	0.40	0.100	0.400	0.003*	84	0.000	0.100	0.200	0.000	0.491
ΔCholesterol non-HDL	51	0.000	-0.30	3.30	-0.000	0.924	82	0.110	11.8	3.70	0.300	0.002*

BMIz variation – difference between the BMI z-score in 2012 and that of 2007; ΔTC – total cholesterol variation; ΔLDL-c – low-density lipoprotein cholesterol variation; ΔTG – triglyceride variation; ΔHDL-c –high-density lipoprotein cholesterol variation; ΔTG/HDL-c – variation of the triglyceride-to-high-density lipoprotein cholesterol ratio; Δ non-HDL cholesterol – variation of the difference between TC and HDL-c. *Pearson correlation and simple linear regression. N= 82 for the variables TC and non-HDL cholesterol in females.

Table 4. Association between lipid profile variation and BM Iz variation in tertiles in a cohort of adolescents from Recife, Brazil 2007-2012.

Males										
Variables	N	BM Iz variation in tertiles								
		Tertile 1		Tertile 2		Tertile 3				P-value*
		$\bar{X}$	SD	N	$\bar{X}$	SD	N	$\bar{X}$	SD	
ΔTC	17	-1.30	±24.9	17	-8.00	±25.4	17	-1.20	±22.8	0.652
ΔLDL-c	17	-1.60	±20.2	17	-5.30	±22.0	17	-3.40	±22.0	0.886
ΔTG	17	-14.9 ^a	±33.2	17	3.10	±42.8	17	29.5 ^b	±32.3	0.003*
ΔHDL-c	17	-0.500	±6.10	17	-3.20	±6.70	17	-3.70	±6.40	0.302
ΔTG/HDL-c	17	-0.200 ^a	±0.800	17	0.100	±1.00	17	0.800 ^b	±0.700	0.002*
Δ non-HDL	17	-0.800	±24.2	17	-0.700	±24.9	17	2.42	±24.6	0.696
Females										
ΔTC	28	0.000 ^a	±21.4	26	8.20	±25.9	28	16.7 ^b	±25.1	0.041*
ΔLDL-c	28	-3.20 ^a	±19.4	28	6.80	±22.4	28	16.6 ^b	±24.5	0.005*
ΔTG	28	-15.6	±34.6	28	-13.9	±25.7	28	-10.0	±49.6	0.855
ΔHDL-c	28	5.60	±6.00	28	3.50	±6.60	28	1.90	±7.40	0.137
ΔTG/HDL-c	28	-0.400 [†]	-0.900- -0.000 [§]	28	-0.300 [†]	-1.3-0.000 [§]	28	-0.200 [†]	-0.700-0.200 [§]	0.224 [†]
Δ non-HDL cholesterol	28	-5.60 ^a	±21.5	26	5.10	±24.1	28	14.7 ^b	±21.1	0.004*

BM Iz variation in tertiles – difference between the BMI z-score of 2012 and that of 2007 in tertiles; ΔTC – total cholesterol variation; ΔLDL-c – low-density lipoprotein cholesterol variation; ΔTG – triglyceride variation; ΔHDL-c – high-density lipoprotein cholesterol variation; ΔTG/HDL-c – variation of triglyceride-to-high-density lipoprotein cholesterol ratio; Δ non-HDL cholesterol – variation of the difference between TC and HDL-c. *One-way analysis of variance. Letters a,b represent a statistically significant difference between the means at a significance level of 5.0%. (Bonferroni post hoc test); [†] median, [§] Interquartile range.

Table 5. Association between variation in lipid profile and excess weight gain in a cohort of adolescents from Recife, Brazil 2007-2012.

Excessive weight gain							
Males							
Variables	BM Iz variation < 75th percentile			BM Iz variation ≥ 75th percentile			p-value*
	N	$\bar{X}$	SD	n	$\bar{X}$	SD	
ΔTC	39	-5.40	±24.3	12	2.50	±23.3	0.324
ΔLDL-c	39	-4.40	±20.6	12	-0.400	±23.1	0.571
ΔTG	39	-3.20	±37.4	12	35.8	±35.0	0.003*
ΔHDL-c	39	-1.90	±6.30	12	-4.10	±6.90	0.307
ΔTG/HDL-c	39	-0.000	±0.900	12	1.00	±0.800	0.001*
Δ non-HDL cholesterol	39	-3.40	±23.9	12	6.60	24.8	0.229
Females							
Variables							
ΔTC	61	6.60	±25.7	21	13.2	±22.1	0.298
ΔLDL-c	63	4.50	±24.9	21	13.4	±17.1	0.131
ΔTG	63	-16.7	±37.6	21	-2.40	±35.9	0.128
ΔHDL-c	63	4.80	±6.30	21	0.100	±7.00	0.006*
ΔTG/HDL-c	63	-0.400 [†]	-1.00- -0.000 [§]	21	0.000 [†]	-0.600-0.300 [§]	0.031 [†]
Δ non-HDL cholesterol	61	1.80	24.4	21	13.0	18.7	0.035*

Excessive weight gain – difference between BMI z of 2012 and that of 2007 ≥ 75th percentile; ΔTC – total cholesterol variation; ΔLDL-c – low-density lipoprotein cholesterol variation; ΔTG – triglyceride variation; ΔHDL-c – high-density lipoprotein cholesterol variation; ΔTG/HDL-c – variation of triglyceride-to-high-density lipoprotein cholesterol ratio; Δ non-HDL-c – variation of the difference between TC and HDL-c. [†]median, [§] Interquartile range. *Student's t-test; [¶] Mann-Whitney U test. N= 82 for the variables TC and non-HDL cholesterol.

in the study sample. According to Gitau et al. (2014)¹⁸, as adolescents age, they become more concerned with body appearance and shape, which encourages males to practice more physical activity to gain muscle mass and females, to lose weight, indicating greater pressure to reach the ideal weight established by society, that is, slimness.

The instrument used for assessing physical activity is subject to memory bias, which could lead to overestimation by more active adolescents. This trend is common in males as they tend to overvalue this practice, regardless of weight status¹⁹.

Although the frequency of active individuals increased, on average 55.5% of the adolescents presented sedentary behavior for more than 4hs and 30min/day, similar to the 58.2% found by Hallal et al.²⁰, who used the same classification method. The high prevalence of sedentary behavior is a current trend as television watching is inexpensive and safe, unlike many outdoor areas in large cities.

Physical activity should be stimulated day by day. For the treatment of dyslipidemia, 60 minutes of vigorous activity and less than 2 hours of sedentary screen activities per day are recommended. The more varied this activity in adolescence the greater the chance of maintaining it in adulthood¹⁶.

The median FV intake was four servings/day for both sexes, which is lower than the intake recommended by the Brazilian Food Guide²¹, which recommends three servings of fruit and three servings of vegetables per day. The updated Dyslipidemia and Atherosclerosis Prevention Guideline recommends at least 5 servings per day of FV¹⁶. This means that this population needs to increase the intake frequency of these foods. The sample presented a higher intake of unhealthy foods, consisting mainly of saturated and trans fats, and simple carbohydrates. Worrisomely, the adoption of such food habits may increase the risk of excess weight²².

The lipid profiles of males and females varied differently. Ribas & Silva²³ also found sex differences in the behavior of lipoproteins and lipids, regardless of age. A possible explanation is the variation in prepubertal growth, which is inherently different between sexes and may be associated with lipid profile²⁴.

Another possible explanation could be sexual maturation, which may promote variations in the levels of lipids and lipoproteins, progressively increasing TC, LDL-c, and HDL-c in females and the opposite in males due to Tanner pubertal stages²⁵. However, prepubertal anthropometric or physiological data of this cohort was not available, and self-assessed sexual maturation stage is often inconsistent, since males tend to overestimate the size of their genitalia and females tend to underestimate the size of their breasts. These data could shed some light on the etiology of these associations.

BMIz variation was associated with lipid profile variation. For each unit increase in BMIz, the lipid fractions in both sexes increased. BMIz variation explained 14% and 16% of the increase in TG and TG/HDL-c ratio, respectively, in

males; and 6.0% of the increase in TC, 10% of the increase in LDL-C, 11% of the increase in non-HDL-c, and 5% of the decrease in HDL-c among females. When the lipid profile variation was compared between the tertiles of BMIz variation, lipid fractions increased more in the third tertile compared to the first.

The literature contains few comparative studies of the lipid profile stratified by BMI variation during adolescence, and most compare only one assessment during adolescence with the outcome in adulthood. Imai et al.²⁶, assessed the association between high BMIz in childhood and adolescence and cardiovascular risk factors, and found that TG increased more in the third tertile than in the first tertile of BMIz, although TC did not differ in either sex. On the other hand, the lipid profile was only investigated in adults, which may justify the differences.

Carvalho et al.²⁷ assessed the association between BMI categorized into tertiles and lipid profile, and found that TC and LDL-c in the third tertile were higher than those of the first. Contrary to the present study, their results did not differ by sex, which may be explained by methodological differences, given that in a cross-sectional study, BMI tertiles do not represent an increase over time but a one-time measurement.

When the lipid profiles of the groups with and without excess weight gain were compared, the group with excess weight gain presented higher variations in lipid levels, regardless of sex. It is possible that the proportion of fat gain increases with BMI, which can increase susceptibility to adverse metabolic effects²⁸ and potentially affect lipoprotein metabolism by increasing peripheral insulin resistance or by directly affecting hepatic lipoprotein metabolism²⁹. The mechanism that may explain this association is the activation of the AMP-dependent kinase pathway, which is induced by high insulin and leptin activation, and low adiponectin activation, which increases fatty acid oxidation. Adiponectin is positively associated with insulin sensitivity and HDL-c, and negatively associated with TG. Moreover, the association between body mass and dyslipidemia can also be explained by higher levels of cholesterylester transfer proteins secreted by adipocytes²³.

The Bogalusa Heart Study found a strong correlation between BMI and fat mass and a weak correlation between BMI and lean mass in individuals with BMI above the median.

Although some authors found that most of the BMI gained during adolescence may be explained by the disproportional increase in lean mass, other authors, such as Victora et al.¹ and Biro et al.³⁰ found that higher BMI increases at the end of childhood and adolescence were associated with an increase in body fat. Wills et al.³¹ found that a higher weight gain during late childhood and adolescence may be harmful, resulting in higher fat mass-to-lean mass ratio and android/gynoid fat distribution.

Given the growing evidence of association between excess body fat and the development of chronic diseases, more studies are needed to elucidate the effect of weight

gain during adolescence on the lipid profile. In conclusion, BMI variation can be a good predictor of changes in the lipid profile of adolescents, including those with normal BMI, and BMI should be monitored during growth.

Since this was a cohort study, some participants were lost to follow-up because they changed schools, resulting in a reduction in sample size. Another limitation was the absence of body composition and fat distribution variables, which could increase the explanatory capacity of the influence of fat mass on lipid profile. Additionally, it was not possible to confirm whether the variation of the lipid profile observed in this investigation was exclusively attributed to an increase in BMI, given that food intake and sedentary behavior may have affected the outcome.

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