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Sex difference in the relationship between childhood obesity and abnormal lipid profiles in young adults

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Abstract

Background Abnormal lipid profiles are a major risk factor for cardiovascular diseases, and childhood obesity has been linked to changes in lipid metabolism in adults. However, the relationship between childhood obesity and adult lipid profiles, as well as the potential sex differences, remain unclear. This study aimed to examine the association between childhood obesity and abnormal lipid metabolism in young adults, specifically focusing on sex differences.

Methods Data were obtained from the Beijing Blood Pressure Cohort Study, which included 1220 participants aged 28–45 years. Childhood obesity was defined based on body mass index (BMI) and subscapular skinfold thickness (SSFT) measurements. Adult lipid profiles, including triglycerides (TG), high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c), and total cholesterol (TC), were measured. Logistic regression models were used to assess the association between childhood obesity and adult lipid profiles, adjusting for potential confounders.

Results During the follow-up period, 18 (2.9%) of 617 male subjects with normal weight as children were obese as adults. Of 516 female subjects with normal weight as children, 9 (1.7%) were obese as adults. In males, childhood overweight/obesity was positively associated with high TG in adulthood (OR = 1.72, 95%CI 1.03–2.85). In females, childhood overweight/obesity was significantly associated with high TC (OR = 5.96, 95%CI 1.42-25.00) and high LDL-c (OR = 6.91, 95%CI 1.17–40.75) in adulthood. The analysis of change in adiposity status from childhood to adulthood revealed that males with normal childhood weight and adult obesity could have the highest risk of all lipid disorders. In females, those with childhood obesity and normal adult weight seemed to have the highest risk of hypercholesterolemia and high TC.

Conclusion This study demonstrates a sex difference in the relationship between childhood obesity and abnormal lipid profiles in young adults. Childhood overweight/obesity is associated with adverse lipid profiles in adulthood,

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with different patterns observed in males and females. These findings highlight the importance of early intervention and prevention strategies for childhood obesity to mitigate the risk of future cardiovascular diseases.

Keywords Childhood obesity, Abnormal lipid profiles, Sex differences

Background

Lipid abnormality is considered as one of the most important risk factors for cardiovascular diseases, which is a leading cause of death worldwide [1–4]. Obesity and its variability are risk factors of cardiovascular adverse outcomes [5, 6]. As the most common metabolic disorder in obesity, insulin resistance is the main driving force for the development of lipid abnormality [7]. In addition, obesity affects the function and structure of lipoproteins, which transport cholesterol and triglycerides in the blood [8]. Studies have shown that adult obesity and its cardiovascular consequences originated in childhood. However, only a few studies evaluated the relationship between childhood obesity and adult lipid profiles, and the results were inconsistent [9–12].

A previous study found a stronger association between BMI and body fat percentage in girls than in boys [13]. Furthermore, the association between sex hormones and obesity was different between men and women [14]. Therefore, it is hypothesized that there may be sex difference in the relationship between childhood overweight and lipid abnormalities in adult population. However, most previous studies did not examine the sex difference of the relationship. In addition, the relationship between childhood obesity and adult lipid profiles was not studied in the Chinese population. Therefore, this study seeks to examine the association between childhood obesity and abnormal metabolism of blood lipids in young adults in a Chinese population. We also aim to examine the sex difference in this relationship.

Methods

Study population

Data were from the Beijing Blood Pressure Cohort Study (BBS), and the rationale, protocol and main details of BBS have been published previously [15]. Briefly, BBS was a prospective cohort study conducted at six primary schools and six high schools in downtown of Beijing. This cohort was designed to examine the effect of childhood risk factors on adult cardiometabolic diseases. At baseline (from April 1987 to October 1988), demographic and physical data and left subscapular skinfold (LSSF) were obtained from 2242 students aged 6–19 years old. From March 2010 to June 2011, all participants were invited for a health examination, and 1,373 participants aged 28–45 years old underwent examinations including questionnaires, physical measurements, biochemical examination, and abdominal ultrasonography. After excluding 153 participants without information on BMI and LSSF in childhood and lipid profiles in adulthood, a total of 1220 subjects were included for final analyses. The flowchart was shown in Fig. 1.

Patient and public involvement

Patients or the public were not involved in the design, conduct, reporting, or dissemination plans of our research.

Childhood measurements

Demographic data were obtained from questionnaires. Weight and height were measured twice with lightweight clothing and no shoes, and the mean values were used. BMI was calculated by dividing weight (kilogram) by height (meter) squared. Childhood obesity by BMI was defined according to the international sex- and age specific BMI cut points [16]. More detailed sex- and age specific BMI cut points are in Table S1 of the supplemental material.

According to the standard protocol [17], the LSSF was measured at 1 centimeter below the tip of left subscapular angle with an Eiken-type skinfold caliper (Shanghai Medical Instrument Development, Shanghai, China). The subscapular skinfold thickness was also measured twice to calculate the average values. Obesity assessed by LSSF was defined as LSSF above or equal to the 85th age - and sex-specific percentile [18]. Finally, childhood obesity was defined as abnormal BMI or LSSF.

Adulthood measurements

All subjects completed questionnaires, providing their demographic information, daily lifestyle, past medical history, and family history. Smoking status was defined as having smoked at least one cigarette a day in the past year. Alcohol drinking was defined as have regular drink in the past year. Weight and height were also measured twice to calculate the average values and BMI. Adulthood obesity was defined as $BMI \geq 28$ kg/m^2 [19]. After fasting for 12 h, venous blood was collected to measure the lipid profiles including fasting triglycerides (TG), high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c) and total cholesterol (TC). According to NCEP ATP III criteria [20], high TC was defined as $TC \geq 6.22$ mmol/L(240 mg/dl), high LDL-c was defined as LDL-c $\geq$ 4.14mmol/L(160 mg/dl), low HDL-c was defined as HDL-c $<$ 1.04(40 mg/dl) mmol/L, and high TG was defined as $TG \geq 2.26$ mmol/L(200 mg/dl). Hypercholesterolemia was defined as high TC and/or high LDL-c and/or low HDL-c. Dyslipidemia was defined as

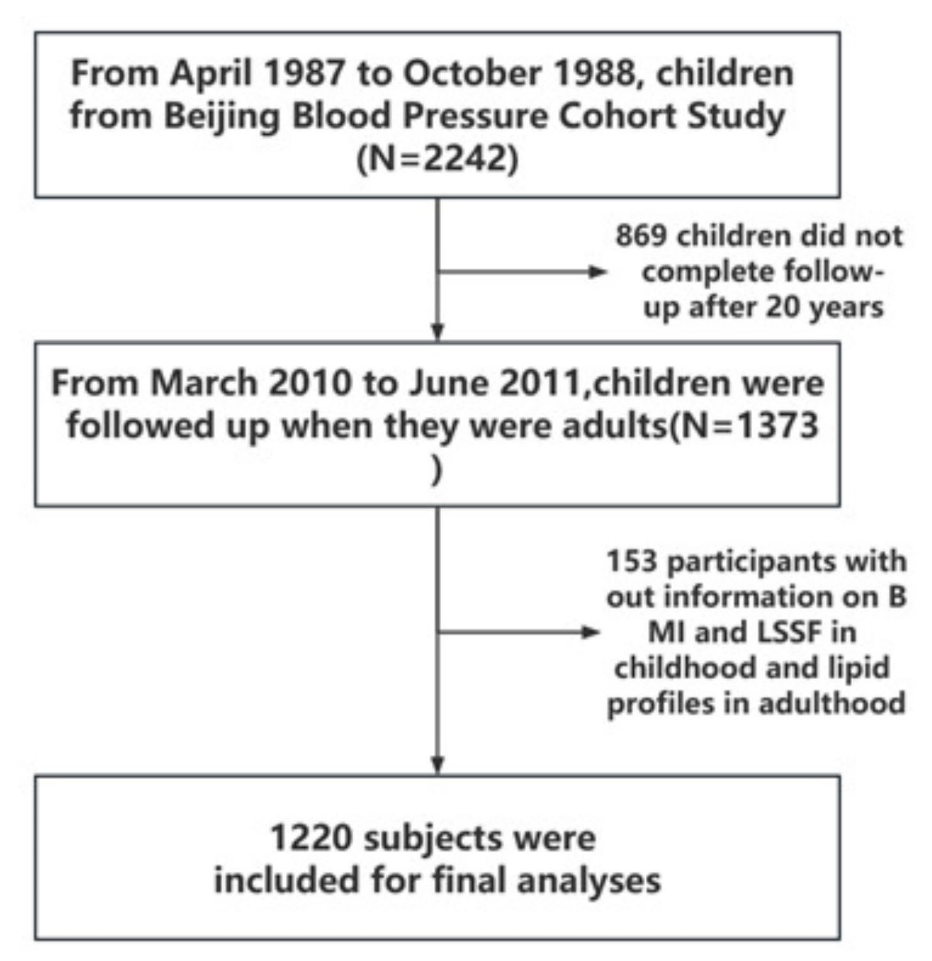

Fig. 1 Flowchart of the study

hypercholesterolemia and/or hypertriglyceridemia and/or being treated with lipid-lowering medications.

Two lipid indices were also analyzed in this study. TG/HDL-C ratio, a cardiovascular risk marker in the general population [21] and in the Chinese population [22], as well as the TC/HDL ratio, which was found to be a strong predictor of ischemic heart disease mortality [23].

Statistical analysis

Data with normal distribution were expressed as mean ± standard deviation, and the differences between groups were tested by t-test. Qualitative variables were expressed as frequencies (percentages), and the Chi-Square test or Fisher's exact probability test was used for comparison between groups.

We used two different multivariate logistic regression models to evaluate the association of childhood adiposity (measured by BMI or SSFT) and lipid profile in adulthood. Given the small sample sizes, we examine BMI or SSFT as continuous variables to build logistic regression models. In model 1, we did not adjust for any variables. In model 2, we adjusted for adult age, smoking status, and alcohol status. Interaction analysis by sex were also performed.

In order to further examine the influence of change in obesity status on adult lipid profiles, we divided participants into four groups according to obesity status in childhood and adulthood: (1) Normal childhood weight and adult non-obese group(N = 1106 boys: 599; girls: 507), (2) Childhood overweight/obese and adult non-obese group(N = 41, boys: 26; girls: 15), (3) Normal childhood weight and adult obese group(N = 27, boys: 18; girls: 9), (4) Childhood overweight/obese and adult obese group(N = 46, boys: 27; girls: 15). This study compared whether there were significant differences in adult lipid profiles among the four groups using Chi-Square test. This study also used two different multivariate logistic regression models to evaluate the influence of change in adiposity status from childhood on adult lipid profile. Similarly, we examine the outcome markers as continuous variables. Model 1 was adjusted for none, Model 2 was adjusted adult age, smoke status and alcohol status.

Table 1 Characteristics of the study populations at baseline and follow-up according to weight status in childhood

Variables	Male			Female		
	Childhood Normal Weight	Childhood Overweight/ Obesity	*P*	Childhood Normal Weight	Childhood Overweight/ Obesity	*P*
N	599	75	--	507	39	--
Childhood						
Age, y	11.97 ± 3.79	10.85 ± 3.01	0.015	11.10 ± 3.54	12.80 ± 3.61	0.004
BMI, kg/m^2	16.78 ± 2.42	22.08 ± 3.36	< 0.001	16.14 ± 2.49	23.40 ± 3.24	< 0.001
SSFT, mm	7.61 ± 2.66	15.98 ± 5.95	< 0.001	8.99 ± 4.22	22.09 ± 7.19	< 0.001
Adulthood						
Age, y	34.86 ± 3.80	33.83 ± 2.95	0.024	34.05 ± 3.52	35.65 ± 3.87	0.007
BMI, kg/m^2	25.54 ± 3.51	30.74 ± 4.39	< 0.001	22.53 ± 3.53	27.79 ± 4.22	< 0.001
WC, cm	89.73 ± 9.39	101.59 ± 10.99	< 0.001	76.89 ± 8.40	85.43 ± 9.35	< 0.001
TC, mmol/L	4.92 ± 0.96	4.99 ± 1.17	0.539	4.49 ± 0.75	4.70 ± 0.87	0.094
HDL-c, mmol/L	1.18 ± 0.28	1.15 ± 0.24	0.324	1.45 ± 0.32	1.35 ± 0.22	0.056
LDL-c, mmol/L	3.01 ± 0.77	2.97 ± 0.73	0.669	2.60 ± 0.64	2.89 ± 0.72	0.006
TG, mmol/L	2.11 ± 2.76	2.34 ± 2.22	0.475	0.99 ± 0.66	1.25 ± 0.73	0.018
Dyslipidemia	273 (45.58%)	40 (53.33%)	0.204	51 (10.06%)	8 (20.51%)	0.043
Hypercholesterolemia	210 (35.06%)	29 (38.67%)	0.538	35 (6.90%)	6 (15.38%)	0.053
Drinking	415 (69.28%)	46 (61.33%)	0.163	254 (50.10%)	17 (43.59%)	0.433
Smoking status			1.000			0.331
Smoker	374 (62.4%)	47 (62.7%)		148 (29.2%)	8 (20.5%)	
Non smoker	225(37.6%)	28(37.3%)		359(70.8%)	31(79.5%)	

Abbreviation: BMI: body mass index; SSFT: subscapular skinfold thickness; WC: waist circumference

Table 2 Association between Childhood Obesity/ overweight and adult lipid profiles according to sex

	OR(95%CI) for Male		OR(95%CI) for Female		*P* for interaction	
	Obesity/ Overweight vs. Normal Weight		Obesity/ Overweight vs. Normal Weight			
	Model 1	Model 2	Model 1	Model 2	Model 1	Model 2
Dyslipidemia	1.36 (0.84–2.22)	1.47 (0.91–2.41)	2.31 (1.01, 5.29)	2.22 (0.95, 5.14)	0.2945	0.4618
Hypercholesterolemia	1.17 (0.71–1.90)	1.21 (0.73–1.98)	2.45 (0.96, 6.25)	2.44 (0.94, 6.33)	0.1870	0.2582
High TC	1.16 (0.46–2.50)	1.21 (0.48–2.65)	5.95 (1.48, 24.00)	5.96 (1.42, 25.00)	0.0633	0.0725
Low HDL	1.28 (0.75–2.13)	1.29 (0.75–2.15)	0.89 (0.20, 3.88)	0.87 (0.20, 3.86)	0.6366	0.5716
High LDL	0.73 (0.21–1.87)	0.77 (0.23-2.00)	6.80 (1.21, 38.34)	6.91 (1.17, 40.75)	0.0474	0.0546
High TG	1.59 (0.96–2.62)	1.72 (1.03–2.85)	1.93 (0.55, 6.77)	1.66 (0.46, 5.93)	0.7854	0.9843

Model 1: adjusted for none. Model 2: adjusted for adult age, smoke status and alcohol status

All analyses were performed using the statistical software packages R (The R Foundation; http://www.R-project.org). Statistical significance was set at $P<0.05$.

Results

Characteristics of study participants

A total 1220 participants (674 males and 546 females) were included for analysis. The baseline and follow-up characteristics of participants by childhood obesity status and sex were shown in Table 1. During the follow-up period, 129 (21.54%) of 599 male subjects with normal weight as children were obese as adults. Of 507 female subjects with normal weight as children, 41 (8.09%) were obese as adults.

Among males, there were no significant differences in triglycerides, total cholesterol, LDL-c, and HDL-c between childhood normal-weight group and childhood overweight/obesity group. Among females, the childhood overweight/obesity group had significantly higher LDL-c (2.60 ± 0.64 mmol/L [100.43 ± 24.77 mg/dL] vs. 2.89 ± 0.72 mmol/L [111.73 ± 27.84 mg/dL]; $P=0.006$) and triglycerides (0.99 ± 0.66 mmol/L [87.56 ± 58.49 mg/dL] vs. 1.25 ± 0.73 mmol/L [110.74 ± 64.64 mg/dL]; $P=0.018$) than the childhood normal weight group, while there were no significant differences in total cholesterol and high-density lipoprotein levels between the two groups. There was no significant difference in the prevalence of hypercholesterolemia between childhood normal-weight group and childhood overweight/obesity group in both male and female groups.

The association between childhood overweight/obesity and adult lipid profiles

The association between childhood overweight/obesity (measured by BMI or SSFT) and adult lipid profile was shown in Table 2.

In males, childhood overweight/obesity was positively associated with High TG in the adulthood (OR = 1.72[1.03, 2.85], Model 2), and had no significant association with dyslipidemia, hypercholesterolemia, high TC, low HDL-c and high LDL-c in the adulthood.

In females, childhood overweight/obesity was positively associated with adult high TC (OR = 5.96[1.42, 25.00], Model 2) and high LDL-c (OR = 6.91[1.17, 40.75], Model 2) in the adulthood and had no significant association with and dyslipidemia, hypercholesterolemia, low HDL-c and high TG in the adulthood.

We also put the results of BMI and SSFT separately in the diagnosis of childhood obesity in Table S2-3 of supplementary materials, and the consistency of the results is strong. In addition to the above trend, when examining BMI and SSFT as continuous variables in Table S4, we found childhood BMI and SSFT were also significantly positively correlated with adulthood dyslipidemia, hypercholesterolemia in both sexes.

The association between childhood BMI and SSFT (continuous variables) and adult TC/HDL-c and TG/HDL-c was shown in Table 3.

In the males, childhood BMI and SSFT are significantly positively correlated with adulthood TG/HDL-C(BMI:β = 0.12, 95%CI (0.00, 0.25), Model 2; SSFT:β = 0.09, 95%CI (0.01, 0.17), Model 2), but not with TC/HDL-C. In the female population, childhood BMI and SSFT were significantly positively correlated with both adulthood TG/HDL-C(BMI:β = 0.06, 95%CI (0.01, 0.11), Model 2; SSFT:β = 0.04, 95%CI (0.01, 0.07), Model 2) and TC/HDL-C(BMI:β = 0.03, 95%CI (0.00, 0.05), Model 2; SSFT:β = 0.02, 95%CI (0.00, 0.03), Model 2).

Comparisons of adult lipid profiles according to change in adiposity status from childhood

We divided the population into four groups as described in the method: Group 1 (Normal childhood weight and adult non-obese), Group 2 (Childhood overweight/obese and adult non-obese), Group 3 (Normal childhood weight and adult obese), Group 4 (Childhood overweight/obese and adult obese).

As shown in Fig. 2, there were significant differences in adult lipid profiles among the four groups. Group 1 is the lowest in every category, while Group 3 is the highest in every category. (all p value < 0.05)

The association between change in adiposity status from childhood and adult lipid profiles

The association between change in adiposity status from childhood and adult lipid profiles was explored according to different sex and presented in Table 4.

The study found that among males, Groups 3 and 4 were more likely to develop dyslipidemia than Group 1. (Group 3: OR = 3.57[2.36–5.49]; Group 4: OR = 2.52 [1.42–4.57]). In the female population, Groups 2 and 3 were more likely to have dyslipidemia than Group 1. (Group 2: OR = 3.90[1.33–10.15]; Group 3: OR = 5.33[2.48–11.06]). Interestingly, compared with the OR values of the Group 3, the OR values of Group 4 was smaller in both sexes, which was also consistent with the results in Table 4.

In the male population, the risk of hypercholesterolemia was significantly higher in Group 3 compared to Group 1. (OR = 2.20[1.47–3.28]) In the female population, similar to dyslipidemia, the risks of hypercholesterolemia in the Groups 2 and 3 were significantly higher than that in Group 1. (Group 2: OR = 3.54[1.10-11.34]; Group 3: OR = 3.30[1.24–7.82]). However, for hypercholesterolemia, there was no statistically significant difference between group 4 and group 1 in both sexes.

In terms of high TC, the risk of Group 3 in males (OR = 2.59 [1.38–4.75]) and Group 2 in females (OR = 12.58[2.36–57.52]) was significantly higher than that in Group 1. In terms of low HDL-c, the risk of Group 3 was significantly higher in both sex than in Group 1. (Group 3 males: OR = 1.63 [1.07–2.48]; Group 3 females: OR = 4.30 [1.60-10.48]). As for the risk of High LDL-c, there was no significant difference between the other groups and the Group 1 in both sexes.

In the male population, the risks of high TG were significantly higher in Group 3 and 4 compared to Group 1 (Group 3: OR = 4.02 [2.65–6.13]; Group 4: OR = 3.16 [1.73–5.72]). In the female population, the risk of high

Table 3 Association between Childhood BMI and SSFT and TC/HDL-c and TG/ HDL-c according to sex

	β (95%CI) for Male		β (95%CI) for Female	
	Obesity/ Overweight vs. Normal Weight		Obesity/ Overweight vs. Normal Weight	
	Model 1	Model 2	Model 1	Model 2
TC/HDL-c				
BMI	0.03 (-0.08, 0.14)	0.02 (-0.11, 0.15)	0.04 (-0.00, 0.08)	0.06 (0.01, 0.11)
SSFT	0.04 (-0.04, 0.12)	0.03 (-0.06, 0.12)	0.02 (-0.00, 0.05)	0.04 (0.01, 0.07)
TG/ HDL-c				
BMI	0.14 (0.04, 0.25)	0.12 (0.00, 0.25)	0.03 (0.01, 0.05)	0.03 (0.00, 0.05)
SSFT	0.11 (0.03, 0.18)	0.09 (0.01, 0.17)	0.02 (0.01, 0.03)	0.02 (0.00, 0.03)

Model 1: adjusted for none. Model 2: adjusted for adult age, smoke status and alcohol status

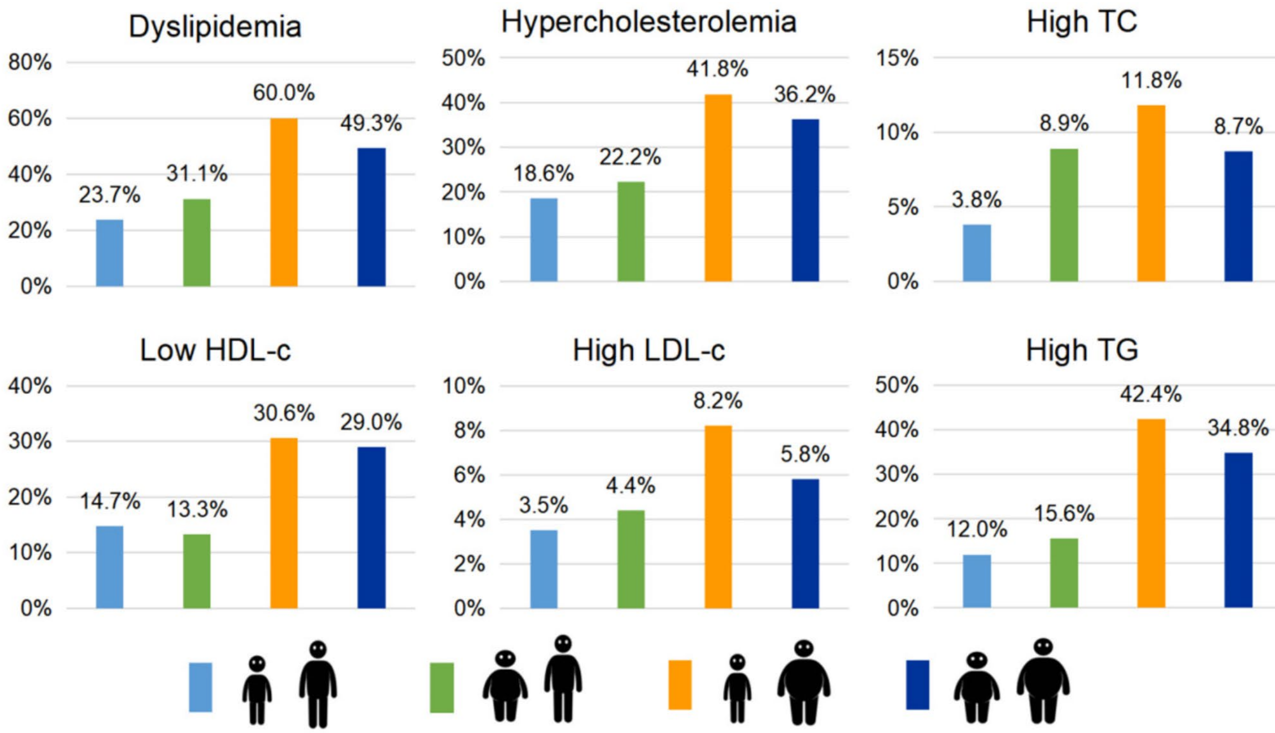

Fig. 2 Adult lipid profiles among the four groups. Group **1** (Normal childhood weight and adult non-obese) was cyan; Group **2** (Childhood overweight/obese and adult non-obese) was green; Group **3** (Normal childhood weight and adult obese) was orange; Group **4** (Childhood overweight/obese and adult obese) was blue

TG in the Groups 3 was significantly higher than that in Group 1. (OR = 6.83[2.43–17.86]).

Further, we examine the outcome markers as continuous variables in Table S5. Among males, there was no significant difference between the other groups and the Group 1 in TC, HDL-c, LDL-c and TG. But in the female population, Group 4 was more likely to develop higher TC($\beta = 0.60$, 95%CI(0.20, 0.99), Model 2) and LDL-c($\beta = 0.64$, 95%CI(0.31, 0.97), Model 2) than Group 1. In terms of higher TG, the risk of Group 3 was significantly higher than that in Group 1 ($\beta = 0.63$, 95%CI(0.19,1.07), Model 2) for females.

Discussion

This study showed that childhood BMI was related to the blood lipid profile in adulthood, and there were sex differences. In male population, childhood overweight/obesity was significantly associated with high TG in the adulthood. In female population, childhood overweight/obesity was significantly associated with high TC and high LDL-c in the adulthood. In addition, we found there could be sex difference in the association between change in adiposity status from childhood and adult lipid profiles. In male population, people with normal childhood weight and adult obese could have highest risk of lipid disorders. However, in female population, people with normal childhood weight and adult obese seem to have highest risk of dyslipidemia, Low HDL-c, and High TG, while people with childhood overweight/obese and adult normal weight seem to show highest risk of hypercholesterolemia and high TC. Even if female participants were not obese in adulthood, overweight/obesity in childhood was possibly correlated with adult dyslipidemia.

There were previous studies investigating the association between childhood BMI and adult blood lipid profile. The Minneapolis Children's Blood Pressure Study on a cohort of students found that triglyceride and HDL-cholesterol levels in adulthood were significantly positively related to childhood BMI [9]. The total cholesterol was also found to be positively related to childhood BMI in males in this study, while the relationships were not significant in females. The Beaver County Lipid Study followed a group of individuals for 9 years also found positive and significant relationships between childhood BMI and triglyceride and HDL-cholesterol levels [10]. A longitudinal study reported a significant positive association between childhood BMI and adult total cholesterol level, LDL-c, and triglyceride [12]. Consistent with previous studies, we found a positive relationship between childhood BMI and the risk of dyslipidemia in adulthood among the total population. Childhood overweight/obesity was positively associated with high TG in the adulthood in overall population. In addition, our study also found a sex difference of the relationship between

Table 4 Cross-grouping of childhood and obesity/ overweight status and lipid profiles according to sex

	OR (95%CI) for Male		OR (95%CI) for Female	
	Model 1	**Model 2**	**Model 1**	**Model 2**
Dyslipidemia				
Group 1	reference	reference	reference	reference
Group 2	0.96 (0.37–2.31)	1.00 (0.39–2.45)	3.75 (1.30–9.56)	3.90 (1.33–10.15)
Group 3	3.46 (2.30–5.29)	3.57 (2.36–5.49)	5.23 (2.44–10.78)	5.33 (2.48–11.06)
Group 4	2.26 (1.28–4.06)	2.52 (1.42–4.57)	1.73 (0.26–6.58)	1.46 (0.22–5.68)
Hypercholesterolemia				
Group 1	reference	reference	reference	reference
Group 2	0.89 (0.31–2.23)	0.91 (0.31–2.32)	3.13 (0.87–8.97)	3.54 (0.96–10.47)
Group 3	2.19 (1.47–3.25)	2.20 (1.47–3.28)	3.22 (1.22–7.57)	3.30 (1.24–7.82)
Group 4	1.65 (0.92–2.91)	1.71 (0.95–3.05)	2.41 (0.37–9.29)	1.99 (0.30–7.94)
High TC				
Group 1	reference	reference	reference	reference
Group 2	0.73 (0.04–3.71)	0.74 (0.04–3.78)	10.95 (2.20-44.67)	12.58 (2.36–57.52)
Group 3	2.53 (1.36–4.64)	2.59 (1.38–4.75)	1.92 (0.10–11.60)	1.93 (0.10–11.81)
Group 4	1.83 (0.66–4.35)	1.98 (0.71–4.75)	NA	NA
Low HDL-c				
Group 1	reference	reference	reference	reference
Group 2	0.95 (0.31–2.49)	0.98 (0.31–2.57)	0.88 (0.05–4.48)	0.97 (0.05–5.08)
Group 3	1.63 (1.07–2.48)	1.63 (1.07–2.48)	4.16 (1.55–10.02)	4.30 (1.60-10.48)
Group 4	1.66 (0.90–2.98)	1.44 (0.89–3.01)	1.44 (0.08–7.69)	1.18 (0.06–6.56)
High LDL-c				
Group 1	reference	reference	reference	reference
Group 2	0.76 (0.04–3.86)	0.77 (0.04–3.94)	5.02 (0.25–35.67)	5.19 (0.25–39.64)
Group 3	1.85 (0.92–3.56)	1.89 (0.94–3.64)	NA	NA
Group 4	0.89 (0.21–2.63)	0.97 (0.23–2.91)	8.25 (0.41-60-47)	8.14 (0.38–65.83)
High TG				
Group 1	reference	reference	reference	reference
Group 2	1.19 (0.38–3.11)	1.25 (0.40–3.33)	2.94 (0.44–11.40)	2.41 (0.36–9.60)
Group 3	3.86 (2.56–5.83)	4.02 (2.65–6.13)	6.65 (2.38–17.14)	6.83 (2.43–17.86)
Group 4	2.82 (1.56–5.03)	3.16 (1.73–5.72)	2.31 (0.12–12.79)	2.09 (0.11–12.12)

Model 1: adjusted for none. Model 2: adjusted for adult age, smoke status and alcohol status. Group 1: childhood no-obesity + adult no-obesity; 2: childhood obesity + adult no-obesity; 3: childhood no-obesity + adult obesity; 4: childhood obesity + adult obesity. Group 1 was used as the control group

childhood overweight/obesity blood lipid profile in adulthood, which is consistent with the results from The Minneapolis Children's Blood Pressure Study.

This was of particular interest with regard to the finding of the association between change in adiposity status from childhood and adult hypercholesterolemia and dyslipidemia. It is empirically believed that the effect of adult obesity on dyslipidemia might be determined in childhood since evidence has proven that obesity tracks from childhood to adulthood [24, 25]. Although being proven to be significantly correlated with BMI in adulthood, childhood BMI was not found to be associated with percentage body fat age [11], suggesting that the association between childhood BMI and adult BMI might not reflect real adiposity. Factors such as the size of the bone frame and muscle mass also contributed to BMI. In childhood, lean body mass contributed a higher proportion of BMI than fat mass [26]. This may explain why childhood BMI was not significantly associated with Low HDL and High LDL in the adulthood in our study.

Our study also found sex differences in the relationship between childhood overweight/obesity and blood lipid profile in adulthood. First of all, the association between childhood overweight/obesity and adult lipid disorder was significant in females, while this association was not significant in males. In addition, in the four groups with different changes in weight status from childhood to adulthood, there were sex difference in its relationship with adult lipid profiles. One reason for the sex difference might be the association between BMI and percent body fat is stronger in girls than boys [13]. Besides, the sexual dimorphism of the hormones might be a possible explanation. Uroguanylin (UGN) has been recently proposed as a key component of the gut-brain axis involved in the regulation of energy and glucose metabolism and pro-UGN was the precursor of UGN [27]. However, recent studies showed that the pro-UGN levels were similar between lean and with obesity girls, while the pro-UGN levels were higher in boys with obesity compared to lean boys [28]. Moreover, the association of sex hormones with adiposity was different between males and females [14]. Further investigations are needed elucidate the intricate mechanisms in greater depth.

There were some limitations to our study. First, the follow-up interval of the study was long, and the results might be biased. Second, the study was a single-center study, so the conclusion might not be applicable to the whole population. Third, the sample size for girls with overweight/obesity was small, and this sample size was further reduced when stratifying by whether these girls were normal weight or obese in adulthood. Consequently, the analysis in Table 3, which examines a 3-way interaction, may be underpowered. Despite the limitations mentioned above, our study took into account the influence of adult BMI on the results, which had a significant impact on the outcome. Future studies should consider this factor when evaluating the relationship between childhood obesity and adult lipid profiles, and the mechanisms behind sex differences remain to be further explored.

Conclusion

In conclusion, we found that childhood obesity/overweight was associated with adult lipid profile after adjusted for adult BMI, and there were sex differences. Childhood obesity/overweight in males was positively associated with high TG in adults. Childhood obesity/overweight in female was positively correlated with high TC and high LDL-C in adulthood, and even with normal weight in adulthood, obesity/overweight in childhood could be positively correlated with dyslipidemia, hypercholesterolemia and high TC in adulthood.

Abbreviations

BMI	Body mass index
SSFT	Subscapular skinfold thickness
WC	Waist circumference
TG	Triglycerides HDL-c: high-density lipoprotein cholesterol
LDL-c	Low-density lipoprotein cholesterol
TC	Total cholesterol
BBS	Beijing blood pressure cohort study
LSSF	Left subscapular skinfold

Supplementary Information

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Supplementary Material 1

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Not applicable.

Author contributions

KZ, YY and JM completed the writing of the paper. LM, YW, ZZ and DH collected and processed the data. RL, TZ and JL made statistical analysis. WC, HC and XZ were responsible for the revision of the paper. All authors confirmed the final version of the paper.

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Data availability

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

Declarations

Ethics approval and consent to participate

This study was established according to the ethical guidelines of the Helsinki Declaration and was approved by the Institutional Review Committee and Ethics Committee of the Capital Institute of Pediatrics, Beijing, China. All subjects and their guardians signed informed consent at baseline and follow-up visit.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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