The association between alcohol consumption and blood lipids in Chinese children and adolescent: findings from the China Health and Nutrition Survey

Qingting Bu[1], Lingyan Fang[2], Bo Huang[3], Huijun Cai[3] and Zhenyu Pan[4,5]*

Abstract

Background Alcohol consumption by children and adolescents is receiving increasing attention. It may cause dyslipidemia, a risk factor for cardiovascular disease. However, the association between alcohol consumption and blood lipids in children and adolescents is unclear, and so we aimed to characterize this association.

Methods Data from the China Health and Nutrition Survey were extracted from children and adolescents aged 7–18 years for whom information was available on alcohol consumption. The population was divided into drinking and nondrinking groups. The χ2, Student's t, or Mann–Whitney U test was used to compare groups. Univariate and multivariate linear regression and propensity score matching (PSM) analysis were used to identify the association between alcohol consumption and blood lipids.

Results This study included 408 children and adolescents with 35 drinkers and 373 nondrinkers. The drinkers had significantly lower values of total cholesterol (TC) (3.8 mmol/L for nondrinkers versus 3.5 mmol/L for drinkers, $p = 0.002$) and high-density lipoprotein cholesterol (HDL-C) (1.3 mmol/L for nondrinkers versus 1.2 mmol/L for drinkers, $p = 0.007$), but not for low-density lipoprotein cholesterol (LDL-C) (2.1 mmol/L for nondrinkers versus 2.0 mmol/L for drinkers, $p = 0.092$) or triglyceride (TG) (0.9 mmol/L for nondrinkers versus 0.8 mmol/L for drinkers, $p = 0.21$). The univariate and multivariate analyses led to the same conclusions. After PSM there was still a significant negative association between alcohol consumption and TC or HDL-C.

Conclusion Alcohol consumption in children and adolescents exhibited significant negative associated with TC and HDL-C, but not with LDL-C or TG. These findings need to be confirmed in future prospective research, and the health effects of blood lipid changes caused by drinking in children and adolescents need to be clarified.

Keywords Alcohol consumption, Drink, Children, Adolescents, Blood lipid

*Correspondence:
Zhenyu Pan
panzhenyu1985@126.com
Full list of author information is available at the end of the article

Bu *et al. BMC Pediatrics* (2024) 24:320

Page 2 of 8

Background

Alcohol consumption is a global public health problem and a major risk factor for cardiovascular disease (CVD) [1, 2]. Alcohol was found to be responsible for an estimated net CVD burden of 593,000 deaths (3.3% of all CVD deaths) and 13 million CVD disability adjusted life years (DALYs) (3.2% of all CVD DALYs) globally [2]. CVD was responsible for 19.8% and 9.8% of all alcohol-attributable deaths and DALYs lost, respectively [2].

As a CVD risk factor, dyslipidemia may play a role in the relationship between alcohol and CVDs. The dyslipidemia characterized by the increase of low density lipoprotein cholesterol (LDL-C) or total cholesterol (TC) is an important risk factor for CVD [3]. Other types of dyslipidemia, such as increased TG or decreased HDL-C, are also associated with increased risk of CVD [4–7]. Some studies have investigated the association between alcohol consumption and blood lipids in adults, but the conclusions were inconsistent. Some studies found a significant relationship between alcohol consumption and high density lipoprotein cholesterol (HDL-C) levels [8–10]. The risk of low HDL-C in males and females decreases as alcohol consumption increases [8]. A J-shaped correlation was found between the alcohol consumption and the risk of high triglyceride (TG) levels in males, but there was no significant association between alcohol consumption and high TG levels in females [8]. Mansour et al. found that drinking was not significantly correlated with TG, TC, or HDL-C [11].

Alcohol consumption among children and adolescents has become an important public health issue. In 2018, a report released by the World Health Organization (WHO) showed that 25.0% of adolescents aged 12–15 drank alcohol, and drinking behavior is showing a trend towards younger age groups worldwide [12]. The study from China shows that the alcohol consumption rate of school-age children can reach 11.2%, and in some special groups, it can reach 22.1% [13]. This may foresee a high burden of CVD due to alcohol consumption in the future. Due to the difficulty in observing CVD in childhood and adolescence, elucidating the association between alcohol consumption and blood lipids in children and adolescents can help explain its relationship with CVD. This is of great significance for public health education. However, few studies have investigated the relationship between alcohol consumption and blood lipids in children and adolescents. Only two previous studies that we know of, published in 1979 and 1981 [14, 15], addressed the relationship between alcohol consumption and blood lipids in adolescents. However, the conclusions of those studies were inconsistent, with one [15] finding a significant positive association between alcohol consumption and HDL-C in adolescents, and the other [14] finding no significant association between adolescent drinking and blood lipids (TC, TG, HDL-C, or LDL-C).

This study therefore explored the effect of alcohol consumption on blood lipids in children and adolescents by analyzing the data from the China Health and Nutrition Survey (CHNS).

Methods

Study design and data sources

This was a cross-sectional study designed based on a subset of the CHNS, which is an ongoing national household-based study employing a multistage random cluster sampling method in nine provinces in China (Guangxi, Guizhou, Heilongjiang, Henan, Hubei, Hunan, Jiangsu, Liaoning, and Shandong) with different geographics, economic development levels, and health indicators [16, 17]. The CHNS was conducted successively in 1989, 1991, 1993, 1997, 2000, 2004, 2006, 2009, 2011, and 2015. The individual response rates were high for each survey wave from 1989 to 2006, with an average of 88% [17]. The obtained biomarker data needed to be analyzed at part of the present study. The CHNS 2009 data were adopted because this was the only time when fasting blood data were available [18]. We collected data on children and adolescents aged 7–18 years for whom information on alcohol consumption was available in the CHNS 2009 database, because such information for those younger than 7 years was not included in the database. Previous studies and the official CHNS website (https://www.cpc.unc.edu/projects/china) include detailed descriptions of the survey procedures and the anthropometric and clinical measurement methods [19–21]. In brief, demographic data were collected using a standardized questionnaire face-to-face; standardized questionnaires of the CHNS 2009 database can be obtained on this website, https://www.cpc.unc.edu/projects/china/data/questionnaires; standard procedures for anthropometric measurements were followed by well-trained examiners; 12 mL of fasting blood was collected via venipuncture, and then immediately centrifuged, and the serum was tested for related measurements. All children and their parents provided written informed consent.

Outcome variable

Serum TC was measured using the cholesterol oxidase–phenol and aminophenazone method; serum HDL-C and LDL-C were measured using the enzymatic method; serum TG was measured using the glycerol-3-phosphate oxidase–phenol and aminophenazone method; reagents were from Kyowa (Japan) and measure machine was Hitachi 7600 for blood lipids detection.

Bu *et al. BMC Pediatrics*   (2024) 24:320

Page 3 of 8

Covariates

The independent variable is drinking alcohol, and the covariates include age, sex, nationality, residence, per-capita household income, body mass index z score (BMIz), blood pressure, 3-day average dietary value. Drinking alcohol, age, sex, nationality, residence, per-capita household income and 3-day average dietary value were collected by face-to-face visit interviews and information questionnaires. For these variables surveyed through questionnaires, the accuracy of the 3-day average dietary value needs to be considered due to its difficulty in measuring compared to other variables. Although CHNS did not disclose the data for the questionnaire validity, it ensures the accuracy of the 3-day average dietary value and other variables through quality control measures. The survey questionnaire and survey manual used are uniformly printed. The interviewers have been graduates of post-secondary schools and uniformly trained. All interviewers have been trained nutritionists who are professionally engaged in nutrition work and have survey experience. The 3-day average dietary value from 24 h recall has been compared with each individual's average daily dietary intake calculated from the household survey. If significant differences are found, the household and the individual in question were revisited. Drinking alcohol is defined as drinking any alcoholic beverage within a year in CHNS. Height and weight were measured by using a standardized protocol from WHO [22]. Body mass index (BMI) is the square of height in meters divided by weight in kilograms. BMIz was calculated as (individual BMI value minus mean BMI value)/(standard deviation of BMI) based on the published data according to sex and age [23]. Blood pressure was measured with the subjects in a sitting position, using a appropriate size of cuff for the children's arm circumference and a sphygmomanometer with a precision of 5 mm Hg. For 3-day average dietary value, a 24 h recall method for three consecutive days for each participant was used to record the dietary. Parents were asked to recall the food consumption of children younger than 12 years. Average Energy, carbohydrate, fat and protein intake were calculated according to the China Food Composition by China CDC National Institute for Nutrition and Health. Children and adolescents with censored data on the analyzed variables were excluded. Cities, suburban areas, towns and county capital cities were classified as the urban area and villages as the rural area.

Statistical analysis

Categorical data were expressed as percentages and compared using χ2 tests. Shapiro–Wilk test was used to check normal distribution. Continuous data conforming to a normal distribution were expressed as mean ± standard-deviation values and compared using Student's t-test, and nonnormal continuous data were expressed as median (25th to 75th percentile) values and compared using Mann–Whitney U tests. Univariate and multivariate linear regression analyses were applied to the association between alcohol consumption and blood lipids. A propensity score matching (PSM) (1:4) analysis was used to minimize the bias between children and adolescents who do and do not drink alcohol. The matched data were used to further compare the blood lipid levels between the two groups. All analyses were conducted using R software (version 3.4.3), and a two-tailed probability values of $p < 0.05$ was considered indicative of statistical significance.

Results

Baseline participant characteristics

This study included 408 children and adolescents aged 7–18 years. Table 1 lists their basic demographics, anthropometrics, and clinical characteristics according to their alcohol consumption status. In total, 35 children and adolescents drank alcohol and 373 did not. Children and adolescents who drank alcohol had significantly lower TC (3.8 mmol/L for nondrinkers versus 3.5 mmol/L for drinkers, $p = 0.002$) and HDL-C (1.3 mmol/L for nondrinkers versus 1.2 mmol/L for drinkers, $p = 0.007$) values, but not LDL-C (2.1 mmol/L for nondrinkers versus 2.0 mmol/L for drinkers, $p = 0.092$) or TG (0.9 mmol/L for nondrinkers versus 0.8 mmol/L for drinkers, $p = 0.21$) values (Table 1). Since the study was not a randomized controlled trial, there were inevitable significant differences in factors such as age, sex, blood pressure, and 3-day average dietary value between the two groups (nondrinkers versus drinkers) (Table 1).

Univariate and multivariate linear regression

Variables that were statistically significant in the univariate linear regression analysis were analyzed as covariates in the multivariate linear regression to adjust for the influence of confounding factors on the association between alcohol consumption and blood lipids. In univariate analysis, sex, nationality, per capita household income, BMIz and drinking were significantly associated with TC; per capita household income and drinking were significantly associated with HDL-C; sex, per capita household income and BMIz were significantly associated with LDL-C, nationality and BMIz were significantly associated with TG (Table 2). Regarding the association between alcohol consumption and blood lipids, as listed in Table 2, alcohol consumption had significant negative associations with TC (coefficient = − 0.36 and $p = 0.003$ for univariate regression, − 0.33 and $p = 0.006$ for

Bu *et al. BMC Pediatrics*   (2024) 24:320

Page 4 of 8

Table 1 Characteristics of the participants according to their alcohol consumption status

	Nondrinker	Drinker	*P* value
Participants, n	373	35	
Age (years), median (25th-75th percentile)	14.0 (13.0–16.0)	15.0 (13.0–17.0)	0.008
Sex, n (%)			0.008
Male	184 (49.3)	26 (74.3)	
Female	189 (50.7)	9 (25.7)	
Han Nationality, n (%)			0.351
Yes	313 (83.9)	32 (91.4)	
No	60 (16.1)	3 (8.6)	
Residence, n (%)			0.095
Urban	104 (29.9)	15 (42.9)	
Rural	269 (72.1)	20 (57.1)	
Per capita household income (RMB), median (25th-75th percentile)	5607.5 (3150.0–10500.0)	6650.0 (4252.5–11976.4)	0.194
BMIz, median (25th-75th percentile)	-0.3 (-0.7–0.5)	-0.2 (-0.8–0.4)	0.56
Blood pressure (mmHg), median (25th-75th percentile)			
Systolic blood pressure	103.3 (97.3–111.0)	109.3 (100.0–111.7)	0.038
Diastolic blood pressure	70.0 (61.0–76.0)	71.3 (67.3–78.3)	0.06
3-day average dietary value, median (25th-75th percentile)			
3-day average energy (kcal)	1786.2 (1472.6–2237.4)	2349.6 (1961.0–2529.2)	<0.001
3-day average carbohydrate (g)	256.2 (207.0–324.2)	320.7 (246.6–369.6)	0.006
3-day average fat (g)	57.0 (40.1–80.7)	79.1 (51.7–99.0)	0.003
3-day average protein (g)	56.6 (43.2–72.8)	69.5 (55.7–84.4)	0.006
Blood lipids (mmol/L), median (25th-75th percentile)			
Total cholesterol	3.8 (3.4–4.3)	3.5 (3.1–3.8)	0.002
High density lipoprotein cholesterol	1.3 (1.1–1.5)	1.2 (1.0–1.4)	0.007
Low density lipoprotein cholesterol	2.1 (1.7–2.6)	2.0 (1.8–2.2)	0.092
Triglyceride	0.9 (0.6–1.3)	0.8 (0.6–1.1)	0.21

multivariate regression) and HDL-C (coefficient = −0.15 and $p = 0.049$ for univariate regression, coefficient = −0.16 and $p = 0.04$ for multivariate regression) in both the univariate and multivariate regression analyses, but not with LDL-C (coefficient = −0.23 and $p = 0.058$ for univariate regression, −0.19 and $p = 0.098$ for multivariate regression) or TG (coefficient = −0.15 and $p = 0.173$ for univariate regression, −0.13 and $p = 0.232$ for multivariate regression).

Propensity score matching

To strengthen the conclusions, we used PSM to further analyze the association between alcohol consumption and blood lipids in children and adolescents (35 drinkers and 140 matched nondrinkers). As shown in Fig. 1, the drinkers and nondrinkers groups had better comparability after PSM. Table 3 also indicates that there was no significant difference between the potential confounding factors of the two groups after PSM. PSM analysis indicated that the children and adolescents drinkers had significantly lower TC values (3.8 mmol/L for nondrinkers

versus 3.5 mmol/L for drinkers, $p = 0.002$) and HDL-C (1.3 mmol/L for nondrinkers versus 1.2 mmol/L for drinkers, $p = 0.012$); however, no significant association was observed between alcohol consumption and LDL-C (2.1 mmol/L for nondrinkers versus 2.0 mmol/L for drinkers, $p = 0.166$) or TG (0.9 mmol/L for nondrinkers versus 0.8 mmol/L for drinkers, $p = 0.194$) levels (Table 3).

Discussion

In this study, we used the CHNS database to investigate the association between alcohol consumption and blood lipids of children and adolescents. The present study was the first that we knew of to specifically investigate this association.

We observed that the children and adolescents who drank alcohol had significantly lower TC and HDL-C levels. However, no significant association was observed between alcohol consumption, and LDL-C and TG. After using multivariate regression analysis to adjust for the confounding factors that significantly affect blood

Bu *et al. BMC Pediatrics* (2024) 24:320

Page 5 of 8

Table 2 Univariate and multivariate linear regression analysis

	TC as dependent variable		HDL-C as dependent variable		LDL-C as dependent variable		TG as dependent variable	
	Univariate regression Coefficient (*P* value)	Multivariate regression Coefficient (*P* value)	Univariate regression Coefficient (*P* value)	Multivariate regression Coefficient (*P* value)	Univariate regression Coefficient (*P* value)	Multivariate regression Coefficient (*P* value)	Univariate regression Coefficient (*P* value)	Multivariate regression Coefficient (*P* value)
Age	0.02 (0.258)		0.01 (0.4)		0.02 (0.267)		0.003 (0.867)	
Sex								
Male	Reference	Reference	Reference		Reference	Reference	Reference	
Female	0.26 (< 0.001)	0.22 (< 0.001)	0.05 (0.262)		0.2 (0.003)	0.17 (0.011)	0.03 (0.605)	
Han Nationality								
Yes	Reference		Reference		Reference		Reference	Reference
No	-0.20 (0.038)	-0.18 (0.051)	-0.06 (0.293)		-0.14 (0.132)		0.18 (0.032)	0.19 (0.022)
Residence								
Urban	Reference		Reference		Reference		Reference	
Rural	-0.14 (0.069)		-0.08 (0.103)		-0.05 (0.533)		-0.01 (0.827)	
Per capita household income	0.00001 (< 0.001)	0.00001 (0.001)	0.000004 (0.0498)	0.000004 (0.04)	0.00001 (< 0.001)	0.00001 (0.002)	-0.000002 (0.573)	
BMIz	0.09 (0.005)	0.07 (0.036)	-0.04 (0.071)		0.09 (0.004)	0.07 (0.018)	0.06 (0.032)	0.07 (0.02)
Systolic blood pressure	0.004 (0.199)		0.0007 (0.722)		0.001 (0.626)		0.003 (0.261)	
Diastolic blood pressure	0.005 (0.234)		0.002 (0.49)		0.001 (0.708)		0.003 (0.406)	
3-day average energy	0.00001 (0.849)		-0.00002 (0.638)		0.00003 (0.637)		-0.00005 (0.324)	
3-day average carbohydrat	-0.0006 (0.126)		-0.0003 (0.172)		-0.0003 (0.463)		-0.0002 (0.495)	
3-day average fat	0.002 (0.1)		0.0002 (0.669)		0.001 (0.183)		-0.0009 (0.295)	
3-day average protein	0.002 (0.236)		0.0006 (0.537)		0.002 (0.273)		-0.00005 (0.973)	
Drinking								
No	Reference	Reference	Reference	Reference	Reference	Reference	Reference	Reference
Yes	-0.36 (0.003)	-0.33 (0.006)	-0.15 (0.049)	-0.16 (0.04)	-0.23 (0.058)	-0.19 (0.098)	-0.15 (0.173)	-0.13 (0.232)

TC Total cholesterol, *HDL-C* High density lipoprotein cholesterol, *LDL-C* Low density lipoprotein cholesterol, *TG* Triglyceride

lipids, the conclusion remained the same. We then used the PSM method to further characterize the association between alcohol consumption and blood lipids in order to increase the reliability of our conclusions. Applying PSM in this study reduced the number of samples in the nondrinking group from 373 to 140, and no significant difference in confounding factors was found between nondrinkers and drinkers. And after PSM there were no changes in our conclusions, which showed the credibility of our conclusions.

The results of research on the association between adult drinking and blood lipids remain somewhat controversial, in that some studies [8–10, 24–28] have suggested that drinking increases blood lipids and another [11] suggested that drinking does not affect blood lipids; however, relatively few studies have suggested that drinking decreases blood lipids in adults. A meta-analysis of 25 experimental studies found that an increase in alcohol intake of 30 g/day in adults resulted in a mean HDL-C increase of 8.3% [24]. Some previous studies involving adults also found that alcohol consumption was associated with a higher HDL-C, in a dose–response manner, and a higher TC concentration [25–28]. The mechanism of HDL-C elevation caused by drinking in adults may be related to ethanol promoting HDL-C synthesis in the liver and reducing lipoprotein lipase activity [12]. The conclusion on the association between alcohol consumption and blood lipid in children and adolescents is different from that in adults, which might be attributable to the following factors: First, children and adolescents
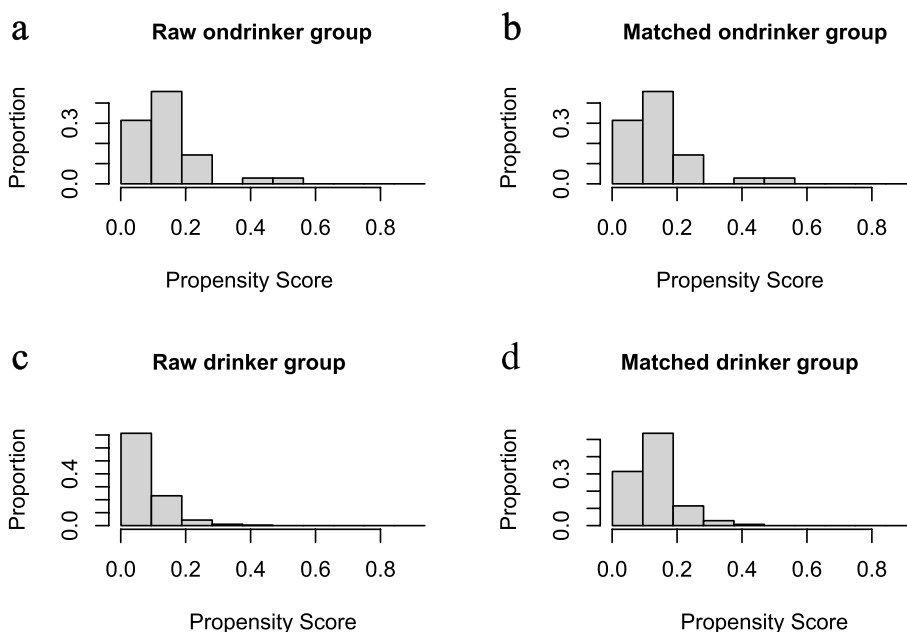
Bu *et al. BMC Pediatrics*   (2024) 24:320

Page 6 of 8

Fig. 1 The distribution of propensity scores before and after matching. **a** Nondrinker group before matching, **b** Nondrinker group after matching, **c** Drinker group before matching, **d** Drinker group after matching

Table 3 Characteristics of the participants according to their alcohol consumption status after propensity score matching

	Nondrinker	Drinker	*P* value
Participants, n	140	35	
Age (years), median (25th-75th percentile)	15.0 (14.0–16.0)	15.0 (13.0–17.0)	0.753
Sex, n (%)			0.900
Male	100 (71.4)	26 (74.3)	
Female	40 (28.6)	9 (25.7)	
Han Nationality, n (%)			0.475
Yes	119 (85.0)	32 (91.4)	
No	21 (15.0)	3 (8.6)	
Residence, n (%)			0.181
Urban	41 (29.3)	15 (42.9)	
Rural	99 (70.7)	20 (57.1)	
Per capita household income (RMB), median (25th-75th percentile)	6102.1 (3139.6–10,848.1)	6650.0 (4252.5–11,976.4)	0.324
BMIz, median (25th-75th percentile)	-0.3 (-0.7–0.4)	-0.2 (-0.8–0.4)	0.748
Blood pressure (mmHg)			
Systolic blood pressure**,** median (25th-75th percentile)	107.7 (100.0–116.0)	109.3 (100.0–111.7)	0.571
Diastolic blood pressure**,** median (25th-75th percentile)	70.0 (63.8–78.0)	71.3 (67.3–78.3)	0.493
3-day average dietary value			
3-day average energy (kcal), median (25th-75th percentile)	2106.6 (1648.0–2530.7)	2349.6 (1961.0–2529.2)	0.146
3-day average carbohydrate (g), mean (SD)	301.7 (95.6)	310.9 (93.9)	0.612
3-day average fat (g), median (25th-75th percentile)	67.5 (46.2–90.8)	79.1 (51.7–99.0)	0.228
3-day average protein (g), median (25th-75th percentile)	63.7 (47.1–81.9)	69.5 (55.7–84.4)	0.305
Blood lipids (mmol/L), median (25th-75th percentile)			
Total cholesterol	3.8 (3.4–4.3)	3.5 (3.1–3.8)	0.002
High density lipoprotein cholesterol	1.3 (1.1–1.6)	1.2 (1.0–1.4)	0.012
Low density lipoprotein cholesterol	2.1 (1.7–2.6)	2.0 (1.8–2.2)	0.166
Triglyceride	0.9 (0.6–1.5)	0.8 (0.6–1.1)	0.194

Bu *et al. BMC Pediatrics*   (2024) 24:320

Page 7 of 8

are in a period of rapid growth and development, and their nutritional intake is more important than that in adults. Ethanol can reduce lipid absorption in the digestive tract [29–32]. This may be more sensitive in children and adolescents, reducing their blood lipids. We found that alcohol consumption in children and adolescents was negatively associated with blood lipids, which suggests that blood lipid reductions were somewhat related to lipid absorption. Second, adults tend to have a longer drinking history and higher total alcohol consumption than children and adolescents. Drinking might affect blood lipids differently during different age stages. Third, drinking alcohol is associated with high levels of steroid hormones, which may increase the effect of steroid hormones on lowering total cholesterol [33]. In the two above-mentioned studies [14, 15] on the association between alcohol consumption and blood lipids, the conclusions were also inconsistent, with one suggesting that there is a significant positive association between alcohol consumption and blood lipids [15], and the other that there was no such association [14]. The difference between these results and ours might be attributable to the study populations, since those two studies included non Chinese adolescents (mainly white, 12–19 years old) while we included Chinese children and adolescents (mainly Han, 7–18 years old).

Considering the research design and sample size in this study, we used multiple methods to correct for the effects of confounding factors. In addition to univariate and multivariate linear regression analyses, we also applied PSM. PSM can be used to reduce confounding biases in retrospective analyses of clinical trial data sets, registries, observational studies, and electronic medical records to improve comparability among groups, and it has been widely used in clinical research [34–36]. As an alternative to multivariate regression analysis, PSM attempts to reduce confounder effects by matching already-treated subjects with control subjects who exhibit a similar propensity for treatment based on preexisting covariates that influence treatment selection. PSM therefore establishes a new control group by excluding control subjects with outlying data. This new control group is less influenced by covariates, allowing for more accurate measurements of the variables [37]. PSM is uniquely valuable in its utility and simplicity, but has the disadvantge of requiring data removal.

Several limitations of the present study should be considered. First, there was no amount and the length of alcohol consumption and standard for mild and heavy drinking in children and adolescents. Therefore, the dose effect association between alcohol consumption and blood lipids cannot be studied, which makes the demonstration intensity of the association between alcohol consumption and blood lipids not strong. More accurate grouping may produce more-reliable information. Second, blood lipids were analyzed as continuous variables in our study because there was no testing standard for the blood lipids of children. If the adult blood lipid test standards were applied, there would be almost no children and adolescents with high blood lipids or dyslipidemia in the population we collected, so blood lipids cannot be analyzed as classified variables in the present study. Third, this is a cross-sectional study which is severely limited in causal inference. A larger sample or prospective study is still needed to further determine the relationship between alcohol consumption and blood lipids in children and adolescents. Fourth, due to the data sourced from CHNS, there is a lack of research factors that may affect blood lipids, such as fish oil intake and familial hypercholesterolemia.

Conclusions

This study suggests that alcohol consumption has significant negative association with TC and HDL-C in children and adolescents, but not with LDL-C or TG. Future prospective studies are needed to confirm these findings and to determine the health implications, such as for cardiovascular or other diseases.

Authors' contributions
Study design and Data extraction: QB, BH and ZP. Statistical analysis: QB, HC and ZP. Manuscript draft: QB, LF, BH, HC and ZP. All authors approved the final version.

Availability of data and materials
Datasets used are available from the website, https://www.cpc.unc.edu/projects/china/data/datasets/data-downloads-registration. Enter this website and fill in several required information (such as email and country), then the data can be freely downloaded.

Declarations

Ethics approval and consent to participate
The studies were reviewed and approved by the Institutional Review Boards of the University of North Carolina at Chapel Hill and the Chinese Center for Disease Prevention and Control. Written informed consent to participate in this study was provided by the participants' legal guardian/next of kin. All authors state that all methods were carried out in accordance with the Declaration of Helsinki, relevant guidelines and regulations.

Consent to publication
Not applicable.

Bu *et al. BMC Pediatrics*　　(2024) 24:320

Page 8 of 8

Author details
[1]Department of Genetics, Northwest Women's and Children's Hospital, Xi'an, Shaanxi, China. [2]Department of Medical Quality Control, Yantaishi Penglai Second People's Hospital, Yantai, Shandong, China. [3]Department of Clinical Laboratory, Xi'an Children's Hospital, Xi'an, Shaanxi, China. [4]Department of Pharmacy, Xi'an Children's Hospital, Xi'an, Shaanxi, China. [5]School of Public Health, Xi'an Jiaotong University Health Science Center, Xi'an, Shaanxi, China.

References
1. Lim SS, Vos T, Flaxman AD, Danaei G, Shibuya K, Adair-Rohani H, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990–2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet. 2012;380(9859):2224–60.
2. WHO. Global status report on alcohol and health 2018. 2018. Available from: https://www.who.int/publications/i/item/9789241565639.
3. Baigent C, Keech A, Kearney PM, Blackwell L, Buck G, Pollicino C, et al. Efficacy and safety of cholesterol-lowering treatment: prospective meta-analysis of data from 90,056 participants in 14 randomised trials of statins. Lancet. 2005;366(9493):1267–78.
4. Ren J, Grundy SM, Liu J, Wang W, Wang M, Sun J, et al. Long-term coronary heart disease risk associated with very-low-density lipoprotein cholesterol in Chinese: the results of a 15-Year Chinese Multi-Provincial Cohort Study (CMCS). Atherosclerosis. 2010;211(1):327–32.
5. Wang M, Zhao D, Wang W, Liu J, Liu J, Liu S, et al. Serum triglyceride is an independent risk factor for acute coronary heart disease events in 35–64 years old Chinese-Chinese Multi-provincial Cohort Study. Zhonghua Xin Xue Guan Bing Za Zhi. 2008;36(10):940–3.
6. Satoh H, Tomita K, Fujii S, Kishi R, Tsutsui H. Lower high-density lipoprotein cholesterol is a significant and independent risk for coronary artery disease in Japanese men. J Atheroscler Thromb. 2009;16(6):792–8.
7. Boudi FB, Kalayeh N, Movahed MR. High-Density Lipoprotein Cholesterol (HDL-C) levels independently correlates with cardiac arrhythmias and atrial fibrillation. J Intensive Care Med. 2020;35(5):438–44.
8. Wakabayashi I. Cross-sectional relationship between alcohol consumption and prevalence of metabolic syndrome in Japanese men and women. J Atheroscler Thromb. 2010;17(7):695–704.
9. Park H, Kim K. Relationship between alcohol consumption and serum lipid levels in elderly Korean men. Arch Gerontol Geriatr. 2012;55(2):226–30.
10. Wannamethee SG, Shaper AG, Perry IJ, Alberti KG. Alcohol consumption and the incidence of type II diabetes. J Epidemiol Community Health. 2002;56(7):542–8.
11. Mansour M, Tamim H, Nasreddine L, El Khoury C, Hwalla N, Chaaya M, et al. Prevalence and associations of behavioural risk factors with blood lipids profile in Lebanese adults: findings from WHO STEPwise NCD cross-sectional survey. BMJ Open. 2019;9(8):e026148.
12. WHO. Global status report on alcohol and health 2018: executive summary. 2018. Available from: https://www.who.int/publications/i/item/a-summary-of-the-global-status-report-on-alcohol.
13. Ma H, Yu Y, Guo D, Zhang J, Su Y, Yu X, et al. Analysis of alcohol drinking among school-aged children in Beijing in 2015. Chin J Sch Health. 2020;41(1):66–9.
14. Morrison JA, Kelly K, Mellies M, deGroot I, Khoury P, Gartside PS, et al. Cigarette smoking, alcohol intake, and oral contraceptives: relationships to lipids and lipoproteins in adolescent school-children. Metabolism. 1979;28(11):1166–70.
15. Glueck CJ, Heiss G, Morrison JA, Khoury P, Moore M. Alcohol intake, cigarette smoking and plasma lipids and lipoproteins in 12–19-year-old children. The Collaborative Lipid Research Clinics Prevalence Study. Circulation. 1981;64(3 Pt 2):Iii 48-56.
16. Zhang B, Zhai FY, Du SF, Popkin BM. The China Health and Nutrition Survey, 1989–2011. Obes Rev. 2014;15 Suppl 1(1):2–7.
17. Popkin BM, Du S, Zhai F, Zhang B. Cohort profile: The China Health and Nutrition Survey–monitoring and understanding socio-economic and health change in China, 1989–2011. Int J Epidemiol. 2010;39(6):1435–40.
18. Xi B, He D, Hu Y, Zhou D. Prevalence of metabolic syndrome and its influencing factors among the Chinese adults: the China Health and Nutrition Survey in 2009. Prev Med. 2013;57(6):867–71.
19. Yan S, Li J, Li S, Zhang B, Du S, Gordon-Larsen P, et al. The expanding burden of cardiometabolic risk in China: the China Health and Nutrition Survey. Obes Rev. 2012;13(9):810–21.
20. Xi B, Zhang M, Zhang T, Liang Y, Li S, Steffen LM. Hypertension screening using blood pressure to height ratio. Pediatrics. 2014;134(1):e106–11.
21. Yan W, Liu F, Li X, Wu L, Zhang Y, Cheng Y, et al. Blood pressure percentiles by age and height for non-overweight Chinese children and adolescents: analysis of the China Health and Nutrition Surveys 1991–2009. BMC Pediatr. 2013;13:195.
22. Eveleth PB. Physical status: the use and interpretation of anthropometry. Report of a WHO Expert Committee. World Health Organ Tech Rep Ser. 1995;854:1–452.
23. Song P, Li X, Gasevic D, Flores AB, Yu ZBMI. BMI, waist circumference reference values for chinese school-aged children and adolescents. Int J Environ Res Public Health. 2016;13(6):589.
24. Rimm EB, Klatsky A, Grobbee D, Stampfer MJ. Review of moderate alcohol consumption and reduced risk of coronary heart disease: is the effect due to beer, wine, or spirits. BMJ. 1996;312(7033):731–6.
25. Brinton EA. Effects of ethanol intake on lipoproteins. Curr Atheroscler Rep. 2012;14(2):108–14.
26. Gaziano JM, Hennekens CH, Godfried SL, Sesso HD, Glynn RJ, Breslow JL, et al. Type of alcoholic beverage and risk of myocardial infarction. Am J Cardiol. 1999;83(1):52–7.
27. Hansel B, Thomas F, Pannier B, Bean K, Kontush A, Chapman MJ, et al. Relationship between alcohol intake, health and social status and cardiovascular risk factors in the Urban Paris-Ile-de-France Cohort: is the cardioprotective action of alcohol a myth? Eur J Clin Nutr. 2010;64(6):561–8.
28. Fan G, Wang Z, Zhang L, Chen Z, Wang X, Guo M, et al. Relationship between alcohol consumption and dyslipidemia among rural residents in Jilin Province. J Med Res. 2015;44(1):28–32.
29. Huang CC, Chen JR, Liu CC, Chen KT, Shieh MJ, Yang SC. Effects of long-term ethanol consumption on jejunal lipase and disaccharidase activities in male and female rats. World J Gastroenterol. 2005;11(17):2603–8.
30. Barros H, Chen Q, Florén CH, Nilsson A. Arachidonic acid absorption in human jejunum in organ culture: effects of ethanol. Eur J Clin Invest. 1990;20(5):506–10.
31. Thomson AB, Keelan M, Clandinin MT. Feeding rats a diet enriched with saturated fatty acids prevents the inhibitory effects of acute and chronic ethanol exposure on the in vitro uptake of hexoses and lipids. Biochem Biophys Acta. 1991;1084(2):122–8.
32. Pownall HJ. Dietary ethanol is associated with reduced lipolysis of intestinally derived lipoproteins. J Lipid Res. 1994;35(12):2105–13.
33. Takala H, Yang Q, El Razek AMA, Ali M, Al-Hendy A. Alcohol consumption and risk of uterine fibroids. Curr Mol Med. 2020;20(4):247–58.
34. Baek S, Park SH, Won E, Park YR, Kim HJ. Propensity score matching: a conceptual review for radiology researchers. Korean J Radiol. 2015;16(2):286–96.
35. Benedetto U, Head SJ, Angelini GD, Blackstone EH. Statistical primer: propensity score matching and its alternatives. Eur J Cardiothorac Surg. 2018;53(6):1112–7.
36. Reiffel JA. Propensity score matching: the "devil is in the details" where more may be hidden than you know. Am J Med. 2020;133(2):178–81.
37. Kane LT, Fang T, Galetta MS, Goyal DKC, Nicholson KJ, Kepler CK, et al. Propensity score matching: a statistical method. Clin Spine Surg. 2020;33(3):120–2.

Publisher's Note
Springer Nature remains neutral with regard to jurisdictional claims in published maps and institutional affiliations.