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Sex differences in the relationship between body mass index in Chinese adolescents and future risk of hypertension: a decade-long cohort study

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Abstract

Objective This study aimed to investigate the relationship between body mass index (BMI) during adolescence and the future risk of developing hypertension, with a particular focus on potential sex differences.

Methods This study was a secondary analysis based on a cohort study involving 2,020 adolescents aged 10–15 years who underwent health check-ups at the MJ Health Screening Center between 1999 and 2008. Cox proportional hazards regression models were used to evaluate the association between BMI and hypertension risk, with stratification by gender. Cox proportional hazards regression with cubic spline functions was employed to explore potential nonlinear relationships, and sensitivity analyses were conducted to ensure robustness.

Results The multivariate Cox proportional hazards regression model showed a significant positive association between BMI and hypertension risk in the overall adolescent population and particularly in males, with hazard ratios (HRs) of 1.204 (95% CI: 1.038–1.396) and 1.181 (95% CI: 1.013–1.377), respectively. In females, a nonlinear relationship with a threshold effect was identified, with an inflection point at a BMI of 24.11 kg/m^2. Beyond this threshold, each 1 kg/m^2 increase in BMI was associated with a 3.491-fold higher risk of hypertension (HR = 4.491, 95% CI: 1.185–17.020).

Conclusion Among Chinese adolescent males, there was a positive dose-response relationship between BMI and future hypertension risk. In adolescent females, a specific nonlinear association with a threshold effect (inflection point: 24.11 kg/m^2) was observed. Maintaining a BMI below 24.11 kg/m^2 in adolescent females may reduce their future risk of developing hypertension.

Keywords Hypertension, Obesity, Nonlinear association, Smooth curve fitting, Metabolic syndrome

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Introduction

Hypertension is one of the important preventable risk factors for premature death and disability [1, 2]. It is estimated that about one-third of adults worldwide suffer from hypertension and about 10.4 million people die from its complications, placing an enormous economic and social burden on families and societies [3, 4]. According to epidemiologic studies, the incidence of hypertension has increased significantly among adolescents and children in recent years [5]. A recent study conducted in Poland showed that it was between 5.6% and 7.9% in adolescents aged 10 to 18 years [6]. A meta-analysis of 55 studies found that the prevalence of hypertension in adolescents was 11.2% [7]. Due to its high prevalence, hypertension is recognized as an emerging health problem among adolescents and has a tendency to become a global problem [8–10]. High values of hypertension in adolescence are associated with target organ damage, which may lead to premature onset of cardiovascular disease and death in adulthood, and often lead to hypertension in young adulthood [11]. The World Health Organization (WHO) categorizes adolescents as individuals aged between 10 and 19 years. This period represents a distinct phase of human development and serves as a crucial window for establishing the groundwork for lifelong health. Understanding the health status of adolescents and identifying and intervening early in the risk factors for hypertension is particularly important for preventing hypertension and its complications in adulthood and for developing relevant public health interventions. Previous studies have documented that gender, age, physical activity, smoking, family history, dietary habits, and body mass index are known major risk factors for hypertension [12–15].

Obesity is an established risk factor for hypertension, and prevention guidelines recommend weight reduction [16–18]. Most studies have confirmed a direct linear relationship between BMI and the risk of developing hypertension; this relationship appears to exist in adults and various ethnic populations, although the extent may vary [19–22]. In addition, some studies have focused on the relationship between obesity and hypertension during childhood and adolescence [23–26]. A cross-sectional study in Japan found that overweight and obese adolescents had a higher risk of hypertension compared to those with normal weight [23]. Similarly, a study involving 2,387 children aged 6–8 years in Mexico reported that overweight and obese children had significantly higher relative risks of hypertension [25]. Another cohort study conducted in Guangzhou, China, followed 7,203 children aged 6–8 years for four years and found that, compared to children with normal weight, the risk of hypertension increased by 31.3% in the overweight group and by 81.6% in the obese group [26].

It is worth noting that fewer studies have focused on adolescents without metabolic syndrome (MetS), and there is limited investigation into the potential nonlinear relationship between BMI and hypertension during childhood and adolescence. Furthermore, these studies differ in terms of ethnicity, study period, BMI range, sex proportions, and adjustment factors. Therefore, the specific relationship between BMI and hypertension risk among Chinese adolescents requires further exploration. Using data from a retrospective cohort study, we conducted a secondary analysis to explore both linear and nonlinear relationships between BMI during adolescence and the risk of future hypertension, as well as sex differences.

Methods

Study design

This study was a secondary analysis of the retrospective cohort established by Hai-Lun Sun et al. [27]. The target independent variable was BMI, and the outcome variable was a hypertension incident.

Data source and study population

The dataset discussed is available from the open-access journal PLOS ONE. It is derived from an article authored by Hai-Lun Sun et al., entitled "Uric Acid Levels Can Predict Metabolic Syndrome and Hypertension in Adolescents: A 10-Year Longitudinal Study," published in PLoS ONE, volume 10, issue 11, article e0143786, https://doi.org/10.1371/journal.pone.e0143786 [27]. This article is accessible under the Creative Commons Attribution-NonCommercial (CC BY-NC 4.0) license, allowing for sharing, reproduction, modification, and the creation of derivative works provided that proper credit is given to the author and source [27]. The original study was approved by the Institutional Review Boards of Cathay General Hospital (CTH IRB) and the Mei Jau Health Screening Center (MHSC IRB). Since all participants were under the age of 18, written informed consent was obtained from the children's close relatives, caregivers, or guardians for the study [27]. This study was a secondary analysis based on the original study; therefore, no additional ethical review was required. Furthermore, the conduct of the original study complied with the Declaration of Helsinki, and this secondary analysis adhered to the same standards [27].

The original researchers randomly selected 8,005 adolescents aged 10 to 15 years who underwent health examinations at the MJ Health Screening Center between 1999 and 2008. The exclusion criteria for the original study were as follows: (i) participants with only one follow-up visit; (ii) participants lacking complete data on MetS; and (iii) participants with a history of type 1 diabetes or those taking medications known to affect MetS components, such as triglycerides (TG ≥ 150 mg/dL) or high-density

lipoprotein cholesterol (HDL-c < 40 mg/dL) [27]. For our study, we further excluded participants with hypertension at baseline or those with unclear hypertension diagnoses during follow-up.

Variables

Body mass index

BMI was treated as a continuous variable, calculated using the formula BMI = weight/height^2 (kg/m^2). It is worth noting that height and weight data were collected at baseline. According to the Chinese BMI classification standard "Screening for Overweight and Obesity in School-age Children and Adolescents" (WS/T 586–2018), issued by the National Health Commission of China in 2018, participants were categorized based on BMI and the corresponding percentiles for their age and sex into the following groups: underweight (BMI below the 5th percentile), normal weight (BMI between the 5th and 85th percentiles), overweight (BMI between the 85th and 95th percentiles), and obese (BMI at or above the 95th percentile) [28].

Hypertension and follow-up

The incidence of hypertension was recorded as a binary variable, categorized into hypertension and non-hypertension. Data on the incidence of hypertension was obtained through blood pressure measurements during follow-up. Participants with SBP ≥ 130 mmHg or DBP ≥ 85 mmHg during follow-up were classified as having newly developed hypertension [29]. The time interval between the diagnosis of hypertension and the baseline assessment was calculated to determine the time of hypertension onset. For participants not diagnosed with hypertension during follow-up, the follow-up time was determined based on the interval between the baseline assessment and the last survey date. It should be emphasized that many participants might not reach the 10-year follow-up endpoint, resulting in censored data. Since this is a multi-center cohort study involving community residents and spanning 10 years, participants may have been lost to follow-up due to relocation, unforeseen events, or other factors. Therefore, the primary reason for the censored data is likely loss to follow-up.

Covariates

The selection of covariates was informed by the original study and prior research [27]. The following variables were used as covariates: sex, age, HDL-c, SBP, TG, DBP, serum low-density lipoprotein cholesterol (LDL-c), Fasting Plasma Glucose (FPG), waist circumference (WC), and uric acid (UA).

Data collection and diagnosis of metabolic syndrome

The original study provided precise definitions for data collection and measurement techniques. WC was measured at the natural waist level, which was identified as the narrowest part of the torso when viewed from the side. The nursing staff employed a standard mercury sphygmomanometer to measure SBP and DBP in the right arm of seated subjects. After a 10-hour fasting period, blood samples were collected from the antecubital vein for biochemical analysis. Within 1 h of collection, plasma was separated from the blood and stored at -30 degrees Celsius until the FPG and lipid profile analysis. FPG was measured using the glucose oxidase method with a YSI 203 glucose analyzer (Yellow Springs Instrument Co., Science Department, Ohio, USA). TG was measured using the dry multilayer film method in a Fuji Dri-Chem 3000 analyzer (Fuji Photo Film Co., Minato-ku, Tokyo, Japan). HDL-c and LDL-c were determined using an enzymatic cholesterol assay following precipitation with sulfated polysaccharides. UA levels were measured using a Hitachi 7150 automatic analyzer (Hitachi, Tokyo, Japan) based on the uricase method. It is important to note that both the current study and the original study adopted the International Diabetes Federation's consensus definition of MetS for children and adolescents [29]. Subjects were diagnosed with MetS if they exhibited three or more of the following abnormalities: abdominal obesity (WC at or above the 90th percentile), hypertension (SBP > 130 and DBP > 85 mmHg), HDL-c < 40 mg/dL, and FPG concentration > 100 mg/dL [27, 29].

Statistical analysis

Continuous variables that follow a normal distribution are presented using the mean and standard deviation. Those with a skewed distribution are represented by the median. Categorical variables are expressed in terms of frequency and percentage. To test for differences between various BMI groups, we employ the Kruskal-Wallis H test for skewed distributions, one-way analysis of variance (ANOVA) for normal distributions, and the chi-square test for categorical variables.

Three different models were established using univariate and multivariate Cox proportional hazards regression to investigate the relationship between BMI and the risk of hypertension among all participants, as well as separately for male and female participants. Prior to the analysis, the proportional hazards (PH) assumption was tested using the Schoenfeld residuals correlation test (corr test), which produced a P-value of 0.1486. This result indicates that the data meets the assumptions required for the application of the Cox proportional hazards model. The specific models were as follows: (i) Model I, which does not adjust for any covariates; (ii) Model II, which is adjusted for age and sex; and (iii) Model III, which is

adjusted for sex, TG, age, LDL-c, HDL-c, FPG, WC, and UA. HR and their corresponding 95% Confidence Intervals (95% CIs) were calculated and reported for each model.

BMI was classified according to the Chinese BMI classification standard "Screening for Overweight and Obesity in School-age Children and Adolescents" (WS/T 586–2018), issued by the National Health Commission of China in 2018 [28]. The P-value for trend was estimated to validate the findings of BMI as a continuous variable and to investigate the potential for a nonlinear relationship between BMI and the risk of hypertension. Due to the close association between high LDL-c and high UA with the risk of hypertension, patients with LDL-c ≥ 130 mg/dl and UA ≥ 7 mg/dl were excluded in the sensitivity analysis to separately investigate the association between BMI and the incidence of hypertension among men, women, and the entire sample [30–33]. In addition, to examine whether the relationship between BMI and the risk of hypertension observed at different follow-up times remains stable, we further conducted a sensitivity analysis by limiting the follow-up time to a maximum of 5 years to analyze their relationship. The specific definitions of events and follow-up are as follows: for participants with a follow-up time exceeding 5 years, they were recorded as not having a hypertension event, and their follow-up time was set to 5 years. For participants with a follow-up time of less than 5 years, their events and follow-up time remained unchanged. Moreover, by calculating the E-value, the possibility of potential or unobserved confounding factors that could influence the relationship between BMI and the risk of hypertension was examined [34].

To further investigate the potential nonlinear relationship between BMI and hypertension risk, Cox proportional hazards models with cubic spline functions were employed separately for male, female, and all participants. When a nonlinear relationship was identified, a recursive algorithm was initially applied to determine the inflection point. Subsequently, a piecewise Cox proportional hazards model was constructed on both sides of the identified inflection point. The log-likelihood ratio test was then utilized to ascertain the most appropriate model for describing the association between BMI and the incidence of hypertension.

The reporting of this study was conducted in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines [35]. Empower Stats software (version 4.1, X&Y Solutions, Inc., Boston, MA) and R statistical software (version 3.5, http://www.R-project.org, The R Foundation) were employed to perform the statistical analyses. Statistical significance was defined as a two-sided P-value below 0.05.

Results

Characteristics and inclusion of study participants

A total of 8,005 adolescents aged 10 to 15 years who underwent health examinations at the MJ Health Screening Center between 1999 and 2008 were randomly selected by the original researchers. After applying the inclusion and exclusion criteria, 5,748 participants were included in the initial study, with a baseline hypertension prevalence of 0.33%. In the present study, 2,020 adolescents without hypertension at baseline were further screened and included among whom 1,128 (55.84%) were male. During a median follow-up period of 4.38 years, 60 participants (2.97%) were diagnosed with hypertension. The screening process is shown in Fig. 1.

BMI followed a normal distribution, ranging from 11.62 to 37.65 kg/m², with a mean of 19.77 ± 3.81 kg/m² (Fig. 2). Participants were grouped according to the Chinese BMI classification standard for children and adolescents, Screening for Overweight and Obesity in School-age Children and Adolescents (WS/T 586–2018). The demographic and clinical characteristics of the participants are listed in Table 1. Compared with the underweight group, the obese group showed significantly higher measurements of WC, SBP, DBP, TG, LDL-c, UA, and age, while HDL-c was significantly lower. In addition, the obese group had a higher proportion of male participants.

Figure 2 illustrated the distribution of BMI among the study participants, which followed a normal curve. The BMI values ranged from 11.62 to 37.65, with a mean of 19.77 kg/m².

Incidence of hypertension in adolescents

During the median follow-up period of 4.38 years, hypertension was developed by 60 (2.97%) adolescents. The incidence of hypertension increased progressively across BMI categories, with rates of 1.16%, 2.08%, 7.19%, and 22.06% in the underweight, normal weight, overweight, and obese groups, respectively ($P < 0.001$). When expressed as incidence rates per 10,000 person-years, the values were 26.59, 44.63, 185.03, and 625.84 for the underweight, normal weight, overweight, and obese categories, respectively (Table 2; Fig. 3).

Figure 3. Participants with overweight and obesity have a higher risk of developing hypertension compared to those who are underweight and of normal weight. (P for trend < 0.001).

Factors affecting the risk of hypertension were analyzed using univariate Cox proportional hazards regression analysis

Univariate analysis indicated that the risk of hypertension in adolescents was not associated with baseline FPG and LDL-c ($P > 0.05$). However, baseline age, TG, UA,

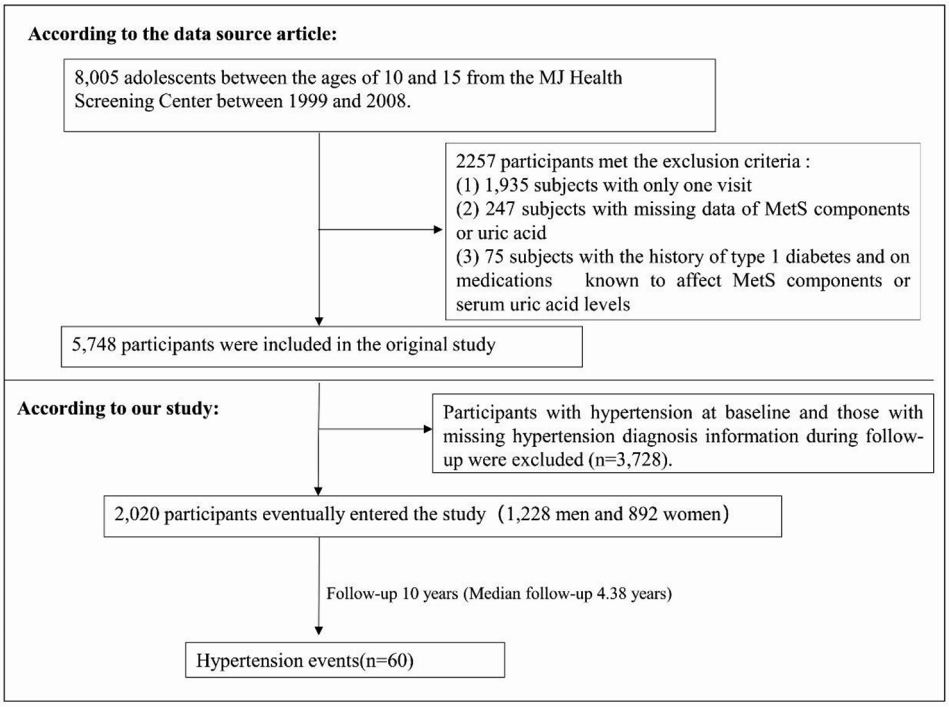

Fig. 1 Flowchart of study participants

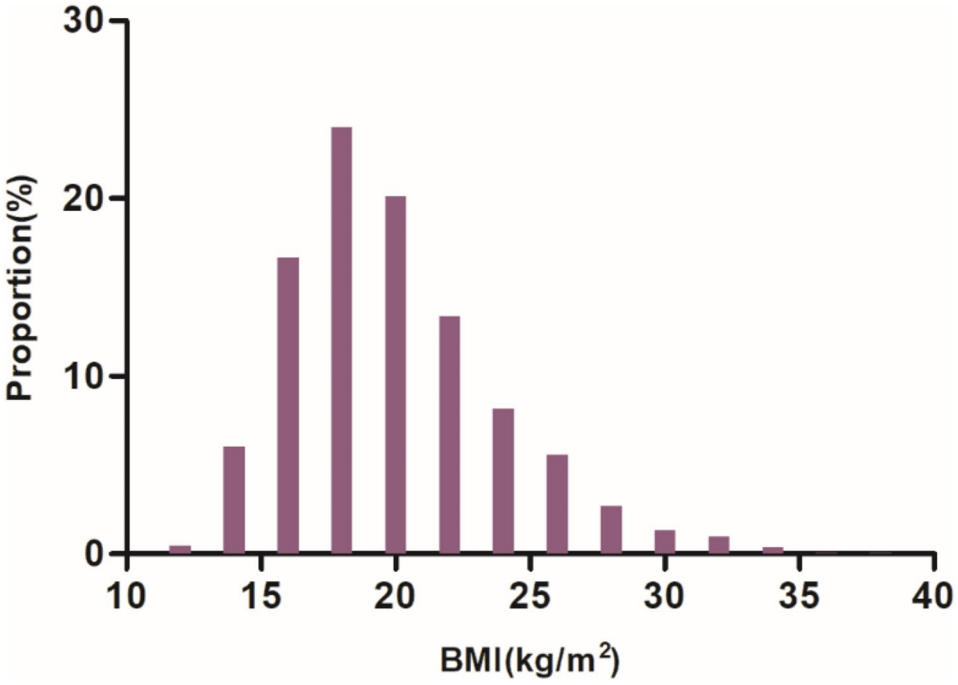

Fig. 2 Distribution of Body Mass Index

WC, SBP, and DBP were positively associated with the risk of hypertension, while HDL-c was negatively associated with the risk of hypertension (all $P < 0.05$). Additionally, females had a lower risk of hypertension compared to males (Table 3).

Figure 4 showed the Kaplan-Meier curves for the probability of survival without hypertension. There are significant statistical differences in the probability of survival without hypertension among BMI categories (log-rank test, $P < 0.001$). Compared to individuals with underweight or normal weight status, those who are

Table 1 The baseline characteristics of participants

BMI group	Underweight	Normal weight	Over-weight	Obesity	*P*-value
Participants(number)	860	877	215	68	
Age(years)	12.35 ± 1.65	12.95 ± 1.63	12.93 ± 1.64	13.22 ± 1.45	< 0.001
WC (cm)	59.43 ± 4.91	68.09 ± 5.57	80.11 ± 6.01	89.76 ± 6.28	< 0.001
SBP (mmHg)	103.32 ± 11.30	108.57 ± 10.85	113.10 ± 10.65	118.56 ± 8.25	< 0.001
DBP (mmHg)	57.98 ± 7.63	59.42 ± 7.54	61.07 ± 8.28	64.35 ± 8.39	< 0.001
FPG (mg/dL)	93.35 ± 6.69	93.56 ± 6.56	94.22 ± 6.61	94.59 ± 8.12	0.204
TG (mg/dL)	66.50 (53.00-86.25)	75.00 (57.00-102.00)	83.00 (64.00-122.00)	104.50 (81.00-146.00)	< 0.001
HDL-c(mg/dL)	60.52 ± 13.10	53.91 ± 11.82	49.55 ± 10.75	47.47 ± 10.07	< 0.001
LDL-c(mg/dL)	91.79 ± 23.93	93.55 ± 25.14	98.72 ± 26.57	98.34 ± 25.40	0.001
UA (mg/dL)	5.69 ± 1.41	6.41 ± 1.56	7.22 ± 1.62	8.50 ± 2.01	< 0.001
Sex					< 0.001
Male	446 (51.86%)	471 (53.71%)	158 (73.49%)	53 (77.94%)	
Female	414 (48.14%)	406 (46.29%)	57 (26.51%)	15 (22.06%)	

SD, standard deviation; n, number (%)

BMI, body mass index; TG, triglyceride; LDL-c, low-density lipoproteins cholesterol; HDL-c, high-density lipoprotein cholesterol; FPG, Fasting plasma glucose; UA, uric acid; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; WC, waist circumference

Table 2 Incidence rate of hypertension (% or per 1000 person-year)

BMI groups	Participants(*n*)	Hypertension events(*n*)	Incidence rate (95% CI) (%)	Per 10,000 person-year
Total	2020	60	2.97(9.76–10.50)	77.97
Underweight	860	10	1.16(0.44–1.88)	26.59
Normal	877	18	2.08(1.11–2.99)	44.63
Overweight	215	17	7.19(4.27–11.54)	185.03
Obesity	68	15	22.06(11.95–32.17)	625.84
P for trend			< 0.001	

BMI, body mass index

overweight or obese demonstrate a significantly higher risk of developing hypertension.

Figure 4. The probability of hypertension-free survival differed significantly between the BMI categories (log-rank test, $p < 0.001$). Participants who are overweight and obese have a higher risk of developing hypertension compared to those who are underweight and of normal weight.

The relationship between BMI and future risk of hypertension among all participants, male, and female adolescents

To elucidate the relationship between BMI and future hypertension risk among adolescents, we developed three Cox proportional hazards regression models, stratifying the analysis by sex. In the overall cohort, a consistent, significant association was observed between increasing BMI and elevated hypertension risk. Each 1 kg/m^2 increment in BMI was associated with a 26.5% (HR = 1.265; 95% CI: 1.202–1.332; $P < 0.001$), 22.8% (HR = 1.228; 95% CI: 1.164–1.295), and 20.4% (HR = 1.204; 95% CI: 1.038–1.396) higher risk of hypertension in Model I, Model II, and Model III, respectively. Secondly, among male adolescents, the Cox proportional hazards regression analyses revealed consistent associations between BMI and hypertension risk across all three models. The HRs (95% CIs) for Models I, II, and III were 1.222 (1.158, 1.289), 1.217 (1.152, 1.286), and 1.181 (1.013, 1.377), respectively. In contrast, the multivariate Cox proportional hazards regression model (Model III) for the female adolescent subgroup showed a non-significant association between BMI and hypertension risk (HR = 1.476, 95% CI: 0.823–2.647).

Besides, BMI was converted from a continuous to a categorical variable, which was then reintegrated into the analysis. Based on the results of the multivariate-adjusted model for all participants, compared to participants with underweight, the HR (95% CI) for normal-weight participants was 1.238 (0.509, 3.013), for overweight participants, it was 4.291 (1.270, 14.496), and for obese participants, it was 10.841 (2.042, 57.571). Similar results were obtained in the male adolescent cohort, with HRs (95% CI) for normal weight, overweight, and obese adolescents being 1.295 (0.511, 3.281), 4.464 (1.246, 15.995), and 11.649 (2.044, 66.384), respectively, compared to those with underweight. In the female adolescent cohort, compared to participants with underweight, the future risk of hypertension for normal-weight and overweight female adolescents decreased by 68.3% (HR = 0.317, 95%CI: 0.009, 11.159) and 59.5% (HR = 0.405; 95%CI: 0.001, 68.437), respectively, while the risk for obese female adolescents increased by 4.587 times (HR = 5.587, 95%CI: (0.004, 75.260). However, none of these were statistically significant.

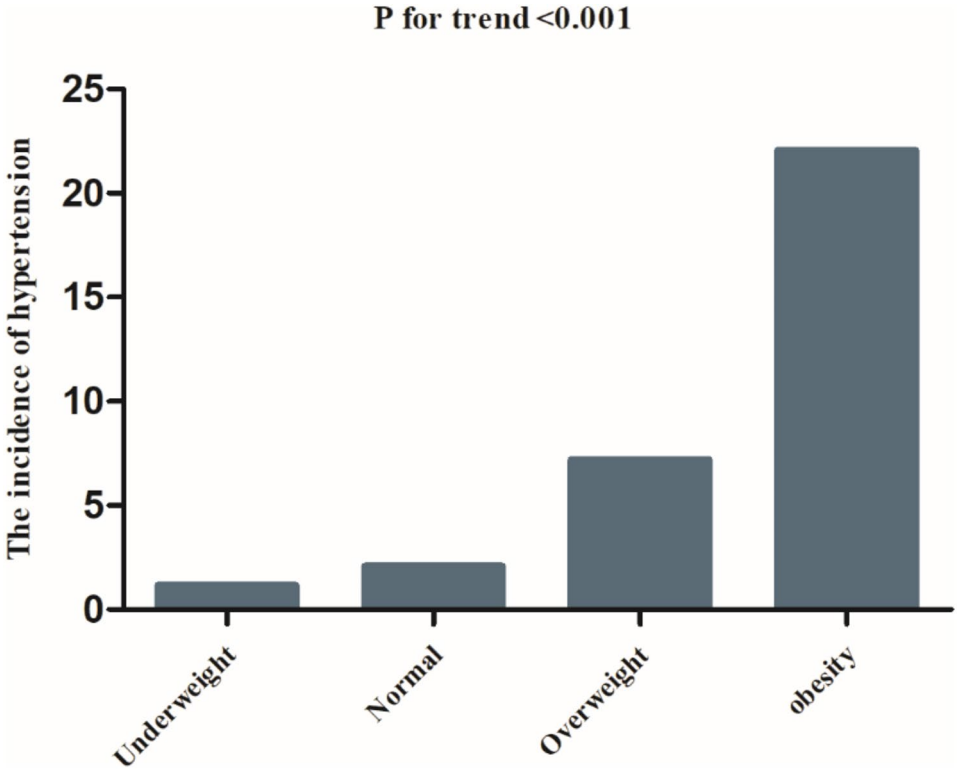

Fig. 3 The incidence rate for hypertension according to the different BMI categories

Table 3 Factors affecting the risk of hypertension were analyzed using univariate Cox proportional hazards regression analysis

	characteristics	HR (95%CI) *P*-value
Sex		
Male	1128 (55.842%)	Ref
Female	892 (44.158%)	0.090 (0.033, 0.249) < 0.001
BMI (kg/m^2)	19.766 ± 3.813	1.265 (1.202, 1.332) < 0.001
WC (cm)	66.411 ± 9.443	1.103 (1.080, 1.127) < 0.001
SBP (mmHg)	107.152 ± 11.616	1.087 (1.059, 1.115) < 0.001
DBP (mmHg)	59.150 ± 7.808	1.063 (1.031, 1.095) < 0.001
FPG (mg/dL)	93.574 ± 6.681	1.030 (0.992, 1.069) 0.120
TG (mg/dL)	83.141 ± 41.386	1.007 (1.003, 1.011) 0.001
HDL-c(mg/dL)	56.045 ± 12.915	0.978 (0.957, 1.000) 0.048
LDL-c(mg/dL)	93.508 ± 24.877	1.003 (0.993, 1.013) 0.586
UA (mg/dL)	6.263 ± 1.651	1.652 (1.471, 1.856) < 0.001
Age(years)	12.703 ± 1.662	1.200 (1.022, 1.408) 0.027

BMI, body mass index; TG, triglyceride; LDL-c, low-density lipoproteins cholesterol; HDL-c, high-density lipoprotein cholesterol; FPG, Fasting plasma glucose; UA, uric acid; SBP, Systolic blood pressure; DBP, Diastolic blood pressure; WC, waist circumference

HR, hazard ratio; Ref: reference; CI: confidence

Sensitivity analysis

To assess the robustness of the findings, a series of sensitivity analyses were performed. First, BMI was transformed from a continuous to a categorical variable, which was then reincorporated into the regression model. The results revealed that the effect sizes across categories were comparable, and the observed trend in effect sizes was congruent with the results obtained when BMI was analyzed as a continuous variable (Table 4, Model III).

Furthermore, sensitivity analyses were performed on participants with LDL-c < 130 mg/dL. After adjustment for confounding variables (including sex, age, LDL-c, TG, HDL-c, FPG, WC, and UA), it was shown that a positive association between BMI and the risk of hypertension was present among all participants (HR = 1.229, 95%CI: 1.045–1.445, $p < 0.001$). In the male cohort, a similar increase in the risk of hypertension by 21.1% for each 1 kg/m^2 increase in BMI was observed (HR = 1.211, 95%CI: 1.021–1.436, $p = 0.028$). However, in the female cohort, the association between BMI and the risk of hypertension was not found to be valid ($p = 0.183$) by multivariate proportional hazards regression analysis with an HR (95% CI) of 1.483 (0.830, 2.649). Additionally, participants with UA ≥ 7 mg/dL were excluded from the sensitivity analyses, and the results were similar, which yielded similar results. The HR (95% CI) for the association between BMI and risk of hypertension was 1.347 (1.206, 1.505) and 1.595 (1.178, 2.159) in all participants and in male participants with UA < 7 7 mg/dL, respectively. In female participants, the HR for the relationship between them was 1.333 (0.968, 1.835), which was not statistically different. Furthermore, Table 5 Model III is a sensitivity analysis with the follow-up time limited to a maximum of 5 years. The results showed that the relationship between BMI and the risk of hypertension,

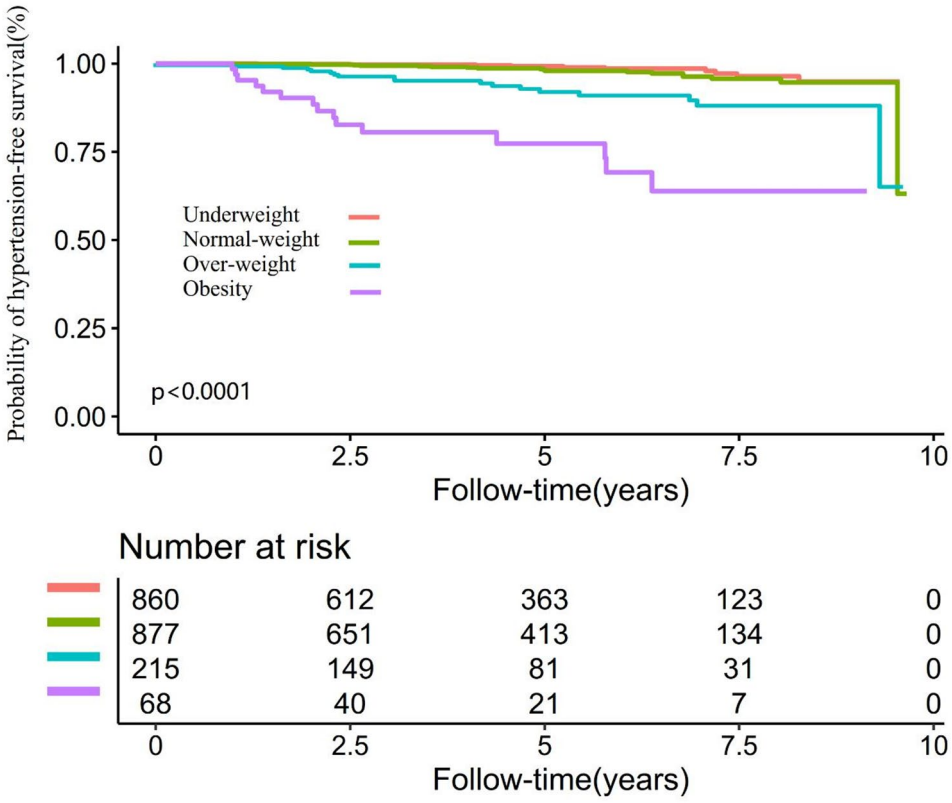

Fig. 4 Kaplan–Meier event-free survival curve

Table 4 The relationship between BMI and the risk of hypertension among all participants, male and female adolescents

	Exposure	Model I(HR,95%CI)	Model II (HR,95%CI)	Model III (HR,95%CI)
Total	BMI	1.265 (1.202, 1.332) < 0.001	1.228 (1.164, 1.295) < 0.001	1.204 (1.038, 1.396) 0.014
	BMI groups			
	Underweight	Ref	Ref	Ref
	Normal weight	1.673 (0.772, 3.627) 0.192	1.460 (0.668, 3.191) 0.342	1.238 (0.509, 3.013) 0.638
	Over-weight	6.818 (3.121, 14.893) < 0.001	5.185 (2.343, 11.473) < 0.001	4.291 (1.270, 14.496) 0.019
	Obesity	25.947 (11.600, 58.041) < 0.001	17.665 (7.723, 40.406) < 0.001	10.841 (2.042, 57.571) 0.005
	P for trend	< 0.001	< 0.001	< 0.001
Male	BMI	1.222 (1.158, 1.289) < 0.001	1.217 (1.152, 1.286) < 0.001	1.181 (1.013, 1.377) 0.034
	BMI group			
	Underweight	Ref	Ref	Ref
	Normal weigh	1.667 (0.743, 3.742) 0.215	1.489 (0.658, 3.371) 0.339	1.295 (0.511, 3.281) 0.585
	Over-weight	5.418 (2.384, 12.315) < 0.001	5.057 (2.207, 11.588) < 0.001	4.464 (1.246, 15.995) 0.022
	Obesity	18.091 (7.769, 42.127) < 0.001	16.585 (7.007, 39.253) < 0.001	11.649 (2.044, 66.384) 0.006
Female	BMI	1.399 (1.115, 1.756) 0.003	1.395 (1.076, 1.807) 0.012	1.476 (0.823, 2.647) 0.191
	BMI group			
	Underweight	Ref	Ref	Ref
	Normal weigh	0.946 (0.059, 15.124) 0.968	0.988 (0.059, 16.674) 0.993	0.317 (0.009, 11.159) 0.527
	Over-weight	6.456 (0.403, 103.510) 0.188	4.905 (0.301, 80.014) 0.264	0.405 (0.001, 68.437) 0.769
	Obesity	35.516 (2.198, 573.965) 0.012	34.647 (1.787, 671.681) 0.019	5.587 (0.004, 75.260) 0.640

Model I: we did not adjust other covariates

Model II: adjusted sex and age

Model III: adjusted sex, age, LDL-c, TG, HDL-c, FPG, WC, and UA

Note: In the sex subgroup, Models II and III were not adjusted for the variable sex

HR, hazard ratio; Ref: reference; CI: confidence

Table 5 The link between BMI and the risk of hypertension among all participants, male, and female adolescents in different sensitivity analyses

Model		Male (HR,95%CI) *P*	Female (HR,95%CI) *P*	Total (HR,95%CI) *P*
Model I	BMI (kg/m^2)	1.211 (1.021, 1.436) 0.028	1.483 (0.830, 2.649) 0.183	1.229 (1.045, 1.445) 0.013
Model II	BMI (kg/m^2)	1.595 (1.178, 2.159) 0.003	1.333 (0.968, 1.835) 0.179	1.347 (1.206, 1.505) < 0.001
Model III	BMI (kg/m2)	1.251 (1.174, 1.333) < 0.001	1.654 (0.822, 3.329) 0.159	1.219 (1.029, 1.444) 0.022

Model I was a sensitivity analysis conducted on participants with LDL-c < 130 mg/dL. Adjusted sex, age, LDL-c, TG, HDL-c, FPG, WC, and UA

Model II was a sensitivity analysis conducted on participants with.UA < 7.0 mg/dL. Adjusted sex, age, LDL-c, TG, HDL-c, FPG, WC, and UA

Model III was a sensitivity analysis with the follow-up time limited to a maximum of 5 years. Adjusted sex, age, LDL-c, TG, HDL-c, FPG, WC, and UA

Note: In the gender subgroup, Models I and II were not adjusted for the variable gender. HR, hazard ratio; CI: confidence

Table 6 Results of two-piecewise Cox hazard proportional regression models

Hypertension:	Male (HR,95%CI, *P*)	Female (HR,95%CI, *P*)	All participants (HR,95%CI, *P*)
Fitting model by two-piecewise Cox hazard proportional regression models			
Inflection points of BMI (kg/m^2)	28.196	24.113	27.257
≤ Inflection point	1.238 (1.043, 1.470) 0.015	0.714 (0.314, 1.621) 0.420	1.229 (1.042, 1.450) 0.014
> Inflection point	1.078 (0.849, 1.368) 0.536	4.491 (1.185, 17.020) 0.027	1.149 (0.944, 1.398) 0.166
P for log-likelihood ratio test	0.269	0.003	0.521

Adjusted sex, age, LDL-c, TG, HDL-c, FPG, WC, and UA. In the gender subgroup, Models1 and II were not adjusted for the variable gender

HR, hazard ratio; Ref: reference; CI: confidence

expressed as HR (95% CI), was 1.219 (1.029, 1.444) for all participants, 1.251 (1.174, 1.333) for male participants, and 1.420 (1.095, 1.843) for female participants, which is consistent with the results of Table 4 (Model III).

The E-value was also calculated to assess the sensitivity to unmeasured confounding factors. The potential impact of unknown or unmeasured variables on the relationship between BMI and the risk of hypertension was likely minimal, as the E-value (1.70) exceeded the relative risk of BMI and unmeasured confounding factors (1.44). Based on all sensitivity analyses, the findings of the study were evidently reliable (Table 6).

Cox proportional hazards regression model with cubic spline functions to address nonlinearity

As depicted in Fig. 5, the application of the Cox proportional hazards regression model with cubic spline functions revealed a divergence in the relationship between BMI and hypertension risk among participants based on sex. While no nonlinear relationship was observed in young male participants, a nonlinear association between BMI and hypertension risk was identified in young female participants. Employing a recursive algorithm, an inflection point for BMI was determined at 24.11 kg/m^2. The two-piecewise Cox proportional hazards regression model showed that in adolescent females, the HR (95% CI) of the relationship between BMI and the risk of hypertension on the left side of the inflection point was 0.714 (0.314, 1.621) 0.420, which did not reach statistical significance. In contrast, the HR for the right side of the inflection point was 4.491 (1.185, 17.020).

Figure 5 explored the relationship between BMI and the risk of developing hypertension in male and female adolescents. The analysis indicated the absence of a nonlinear relationship between BMI and hypertension risk in male adolescents. In contrast, a distinct nonlinear relationship was observed in female adolescents.

Discussion

This study was designed to investigate the association between BMI and the risk of developing hypertension in adolescents. It was found that a dose-response relationship exists between BMI and hypertension risk in male adolescents. In female adolescents, the relationship between BMI and future hypertension risk is characterized by a threshold effect curve, with an inflection point identified at a BMI of 24.11 kg/m2. When the BMI exceeds 24.11 kg/m2, BMI is positively linked with the risk of hypertension in female adolescents.

Research conducted across different ethnic groups has extensively reported a positive association between BMI and hypertension incidence [21, 36–40]. A meta-analysis of 57 cohort studies examined the dose-response relationship between BMI and hypertension risk, revealing that for every 5-unit increase in BMI, the overall relative risk (RR) was 1.50 (95% CI: 1.40–1.59). Compared to individuals with a baseline BMI < 25 kg/m², those with a baseline BMI ≥ 25 kg/m² had a 25% higher risk of developing hypertension [20]. However, studies investigating the relationship between BMI and hypertension risk in children and adolescents remain relatively scarce. A cross-sectional study conducted in Japan found that compared to adolescents with normal weight, obese adolescents had the highest risk of hypertension, with an odds ratio (OR) of 13.54 (95% CI: 6.40-28.62), while overweight adolescents had an OR of 3.21 (95% CI: 1.72–5.98) [23]. Similarly, a cohort study from China demonstrated that after adjusting for potential confounders, each standard

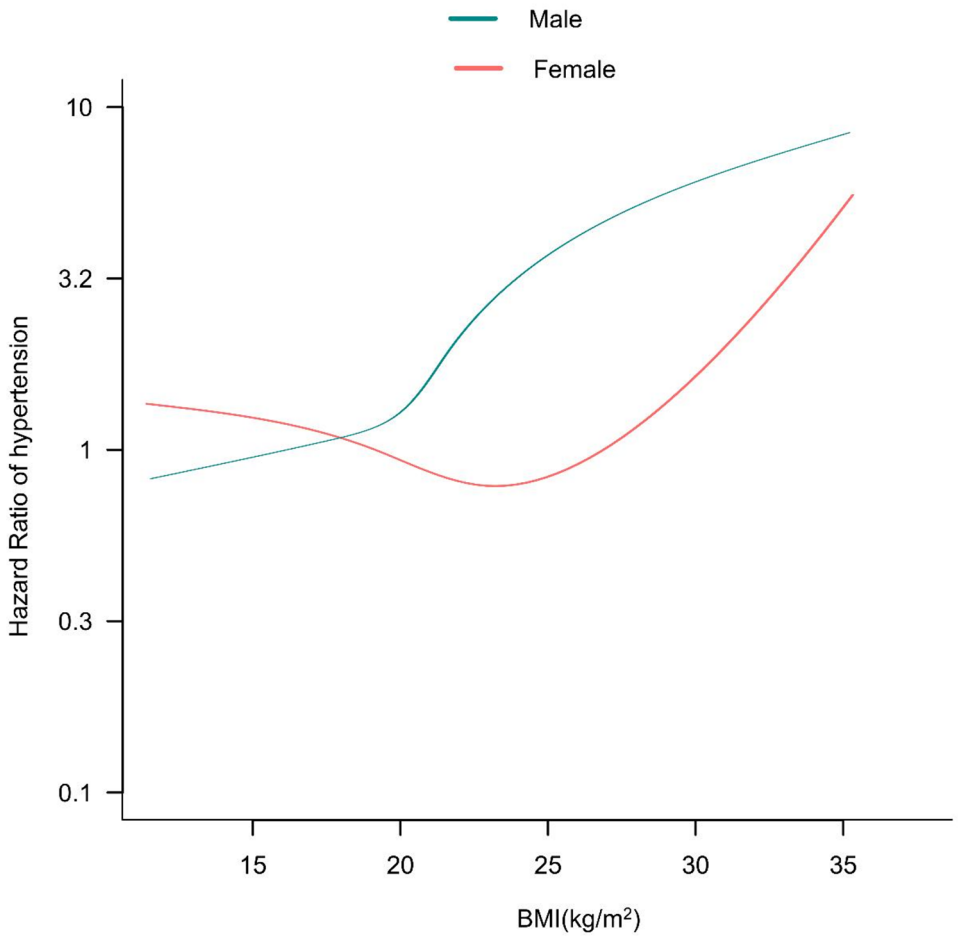

Fig. 5 Nonlinear associations between BMI and hypertension risk across sex

deviation increase in BMI (approximately 1.85 kg/m²) was associated with a 32% higher risk of hypertension (HR = 1.32; 95% CI: 1.003–1.73) [24]. Another cohort study conducted in Guangzhou, China, followed 7,203 children aged 6–8 years for four years and found that compared to children with normal weight, the adjusted HRs for hypertension were 1.313 (95% CI: 1.179–1.461) in the overweight group and 1.816 (95% CI: 1.634–2.081) in the obese group [26]. Additionally, a 14-month study in Mexico involving 2,387 children aged 6–8 years reported that compared to children maintaining a normal BMI, overweight children had a relative risk (RR) of 3.6 (95% CI: 1.5–8.5) for hypertension, while obese children had a significantly higher RR of 14.2 (95% CI: 7.2–27.7) [25]. Our findings align with these studies, demonstrating a positive association between BMI and the risk of future hypertension. These results confirm that overweight and obesity are significant risk factors for hypertension in children and adolescents, although the effect sizes vary considerably. The observed differences in effect sizes may be attributed to several factors. First, the sample sizes across studies vary widely. Second, these studies adjusted for different covariates. Third, differences in the ethnic populations studied and the BMI classification criteria used may also contribute to the observed variability. The relationship between BMI and hypertension risk has been shown to differ across ethnic groups due to variations in body composition, fat distribution, genetic predispositions, and environmental factors. For instance, studies have demonstrated that compared to Western populations, Asians tend to develop hypertension at lower BMI levels [41]. In addition, our investigation utilized BMI as both a categorical and a continuous variable to examine its relationship with hypertension risk, minimizing information loss and providing a quantitative analysis of the association. Furthermore, sensitivity analyses specifically focused on participants with LDL-c < 130 mg/dL and UA < 7 mmol/L, and the results further confirmed the robustness of the observed relationship in these subgroups. Identifying BMI as a risk factor for future hypertension in adolescents and elucidating the relationship between the two will be instrumental in preventing hypertension in this population.

Moreover, it is worth noting that the relationship between BMI and hypertension risk may also differ by sex, given the differences in body fat percentage and distribution patterns between males and females. A cross-sectional study from Israel stratified participants by BMI deciles, multivariable logistic regression analysis revealed that in males, the relationship between BMI and hypertension was significant from the third decile (OR = 1.67, 1.06–2.65) to the tenth decile (OR = 30.17, 20.83–43.69), using the first quartile as a reference. In females, a significant increase in the risk of hypertension was observed in the ninth decile (OR = 3.82, 1.42–10.22) and the tenth decile (OR = 18.92, 7.7-46.51), with no significant trend observed in the lower deciles [40]. This also suggests that the relationship between BMI and the risk of hypertension in adolescents differs between males and females. Therefore, we further conducted stratified analysis and found that in males, BMI and the incidence of hypertension showed a positive dose-response relationship, while in females, no association was found between them based on a linear regression equation. At the same time, sensitivity analysis found that in populations with LDL-c < 130 mg/dl and UA < 7 mg/dl, the relationship between BMI and the incidence of hypertension remained positively associated in males, while the linear association between them was not valid in females.

Notably, after stratifying by BMI, the results of the multivariable-adjusted model showed that among all participants and among males, HRs increased progressively from normal weight to obesity when using the underweight group as the reference. In contrast, Cox proportional hazards regression analysis in adolescent females revealed no significant association between BMI—whether treated as a continuous variable or categorized into quartiles—and hypertension risk. These findings suggest that the relationship between BMI and hypertension risk differs by sex and may be non-linear. In this study, we employed Cox proportional hazards regression with cubic spline functions to test our hypothesis. Ultimately, we observed a linear dose-response relationship between BMI and hypertension risk in adolescent males, whereas in adolescent females, a nonlinear relationship was identified, with a turning point for BMI at 24.11 kg/m². The identification of a curvilinear relationship between BMI and the incidence of hypertension in adolescent females holds significant clinical implications. It contributes to providing clinical counseling and optimizing hypertension prevention strategies for adolescent females. Specifically, for adolescent females, reducing BMI to 24.11 kg/m² through exercise and lifestyle interventions could significantly minimize their future risk of developing hypertension.

Several strengths can be identified in our study. First, both categorical and continuous measures of BMI were utilized to evaluate its relationship with hypertension risk, thereby minimizing information loss and allowing the association between the two to be quantified. Second, significant improvements in handling nonlinear relationships were made compared to previous studies. Additionally, a series of sensitivity analyses were conducted to ensure the robustness of the findings. After participants with LDL-c ≥ 130 mg/dL and UA ≥ 7 mg/dL were excluded, the relationship between BMI and hypertension risk was reanalyzed in adolescent males, females, and the entire cohort, and E-values were calculated to assess the potential impact of unmeasured confounding factors.

This study has several limitations that should be considered. First, the population consisted exclusively of Chinese adolescents, requiring further research to examine the association between BMI and hypertension risk in populations with different genetic backgrounds. Future collaborations with researchers outside China are planned to validate these findings in diverse groups. Second, BMI and other parameters were only measured at baseline, without accounting for changes over time and their relationship to hypertension risk. Future studies should address this by collecting longitudinal data through new research or collaborations. Third, as with all observational studies, unmeasured or uncontrolled confounding factors may exist despite adjustments for known confounders. Additionally, this study relied on secondary data analysis, limiting adjustments for variables not included in the dataset, such as physical activity, diet, and genetic factors. However, E-values suggest that unmeasured confounders are unlikely to explain the findings. Finally, this observational study demonstrates an independent association between adolescent BMI and future hypertension risk but cannot establish causality.

Conclusion

This study reveals distinct associations between BMI and the risk of future hypertension in Chinese adolescents: a linear dose-response relationship in males and a nonlinear relationship in females, with a BMI turning point of 24.11 kg/m². When female BMI exceeds 24.11 kg/m², the risk of hypertension increases significantly. Clinically, these findings can guide personalized interventions: females should maintain a BMI below 24.11 kg/m² through lifestyle and exercise modifications to reduce future hypertension risk, while males require continuous monitoring and management of BMI at all levels to lower their risk. From a public health perspective, this study highlights the importance of early BMI screening and targeted hypertension prevention strategies during adolescence, with a focus on gender-specific BMI management approaches.

Abbreviations

BUN	Blood urea nitrogen
SBP	Systolic blood pressure
BMI	Body mass index
HDL-c	High-density lipoprotein cholesterol
DBP	Diastolic blood pressure
LDL-c	Low-density lipoprotein cholesterol
FPG	Fasting plasma glucose
TG	Triglyceride
HR	Hazard ratio
Ref	Reference
CI	Confidence interval

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Author contributions
Rongtian Liu, Haofei Hu, and Changchun Cao were instrumental in conceptualizing the study, performing the statistical analysis, and drafting the initial manuscript. Yong Han, Yuxin Bai, and Wei Feng contributed significantly by revising the manuscript critically and refining the study design. All authors have meticulously reviewed and endorsed the final version of the manuscript for publication.

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Data availability
Data utilized in this study are available for download from the "PLOS ONE" database, which can be accessed at the following URL: https://journals.plos.org/plosone/.

Declarations

Ethical approval
The original study was approved by the Institutional Review Boards of Cathay General Hospital (CTH IRB) and the Mei Jau Health Screening Center (MHSC IRB).

Informed consent
Since all participants were under the age of 18, written informed consent was obtained from the children's close relatives, caregivers, or guardians for the study.

Consent for publication
Not applicable.

Competing interests
The authors declare no competing interests.

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