The association between parental weight status and risk of hypertension in children aged 6 to 12 years

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Liping Lu MD, PhD[1,2,3], Cheng Chen PhD[1,2], Pengcheng Xun MD, PhD[4], Jialu Wang MS[3], Yanping Wan MD[3], Ka He MD, ScD[1,2]

[1]Department of Obstetrics and Gynecology, Columbia University Irving Medical Center, New York, USA
[2]Department of Epidemiology, Columbia University Mailman School of Public Health, New York, USA
[3]Department of Clinical Nutrition, Ren Ji Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai, China
[4]Department of Epidemiology and Biostatistics, School of Public Health-Bloomington, Indiana University, Indiana, USA

Authors' email addresses and contributions:
Liping Lu designed the research, performed anthropometric measurements, questionnaire survey and data collection, prepared the tables and figure, and carried out literature search and drafted the manuscript, and administered the overall scientific management. Email: ll3407@cumc.columbia.edu
Cheng Chen conducted data analysis, prepared the tables and figure, and contributed to the revision of the manuscript. Email: cc4568@cumc.columbia.edu
Pengcheng Xun conducted data analysis and prepared the tables and figure and contributed to the revision of the manuscript. Email: pxun@indiana.edu
Jialu Wang performed anthropometric measurements, questionnaire survey and data collection. Email: colliewang01@126.com
Yangping Wan designed the research, performed anthropometric measurements, questionnaire survey and data collection. Email: wanyp204@163.com
Ka He contributed to data interpretation and the revision of the manuscript. Email: kk3399@cumc.columbia.edu.

Corresponding Author: Dr Liping Lu, Department of Obstetrics and Gynecology, Columbia University Irving Medical Center, 622 West 168th Street, New York, USA. Tel: +1-212-305-6973. Email: ll3407@cumc.columbia.edu

ABSTRACT

Background and Objectives: Parents may play important roles in the regulation of children's weight status and consequently the development of childhood hypertension. Thus, this study aimed to examine parental weight status, as a marker of parents' diet and lifestyle, in relation to risk of hypertension in their children. **Methods and Study Design:** A total of 1,949 children aged 6 to 12 years (1,012 girls, 52%) and their parents were included. Information on demographics, anthropometrics, lifestyle, diet, and medical history were obtained from the participants and their parents through self-administered questionnaires. Childhood hypertension and elevated blood pressure were defined as SBP and/or DBP ≥95th and ≥90th age- and gender-specific percentile, respectively. Parental overweight was defined as BMI ≥24.0 kg/m^2. **Results:** The prevalence of childhood hypertension was 8.4%, with no significant gender difference (p=0.36). Parents' weight status, especially maternal, was associated with childhood hypertension. After adjustment for potential confounders, children with two parents being overweight were two times more likely to have hypertension as compared with children who had both parents being of normal weight [multivariable-adjusted odds ratio=2.09; 95% confidence interval: (1.26, 3.46)]. After further adjustment for children's body mass index, the observed association was substantially attenuated and became statistically non-significant. **Conclusions:** Findings from this study suggest that parental weight status is associated with the prevalence of hypertension in children presumably through influencing children's weight. Further studies are needed to establish causal inference. This study highlights the importance of parental lifestyle in children's health.

Key Words: hypertension, children, parents, weight status, overweight

INTRODUCTION

The prevalence of hypertension (HTN) in children and adolescents has increased substantially worldwide in the past 30 years and has become a major public health concern.[1] Similar to Western countries,[2] the prevalence of childhood HTN has risen in China, as high as 23% in certain provinces in recent years.[3] Mounting evidence suggests that elevated blood pressure (BP) during childhood is associated with higher risk of primary HTN later in adulthood,[4,5] which is a major risk factor for cardiovascular diseases.[6] Thus, identifying any risk factor for childhood HTN is of great public health importance.

Although childhood HTN is likely a multi-influenced phenotype,[7] being overweight/obese may be the most pronounced risk factor.[8,9] Children with obesity are more likely to have a

higher BP compared with their peers of normal weight.[10-12] In addition to genetics, children's weight status may be largely affected by their parents through diet and/or lifestyle. Studies have found that the risk of childhood obesity was significantly increased if one or both of a child's parents were overweight/obese.[13] It was also suggested that approximately 5% of childhood obesity cases were related to inherited impaired gene function, and the rest were associated with obesogenic behaviors, including unhealthy diet and sedentary lifestyle, which are most likely to be influenced by parents.[14,15] Therefore, presumably, parents may play important roles in the regulation of children's weight status and consequently risk of childhood HTN. Since obesity is directly caused by excess calorie intake and/or insufficient physical activity,[16,17] the weight status of an adult has been used as a marker of his/her diet and lifestyle.[15,18] However, data directly relating parents' dietary/lifestyle characteristics or weight status to risk of HTN in their children are lacking, especially in Asian populations.

Therefore, we examined the association between parental body mass index (BMI) and risk of HTN in children in China. We hypothesized that children with parents being of overweight/obesity were more likely to have HTN, and such an association was mediated through the child's weight.

MATERIALS AND METHODS

Study design and sampling

A total of 2,571 students were enrolled in June 2013 from four elementary schools, randomly selected in Pudong district, Shanghai, China. Of them, 622 were excluded in the analysis for one of the following reasons: missing data on pregnancy duration (n=166), birth weight (n=130), preterm birth (n=33), or abnormal birth weight (birth weight <2.5 or ≥4 kg, n=293). After these exclusions, 1,949 children (937 boys, 48%), 6–12 years old, full-term with normal birth weight, were included in the analyses. This study was approved by the Ethics Committee of Ren Ji Hospital, School of Medicine, Shanghai Jiao Tong University (AFINS-HOPE-2013-06). Written informed consents were obtained from all children and their parents.

Ascertainment of blood pressure and hypertension

Children were asked to rest for at least 10 minutes in a sitting position before the examination. BP was measured on the right arm at the heart level by clinicians using a mercury sphygmomanometer. Two measures were recorded with a 5-minute interval. The average of the two measures was used in the analyses. The cuff chosen for each child met the criteria that the bladder width is at least 40% of the mid-arm circumference and the bladder length covers

80–100% of the mid-arm circumference. The first Korotkoff sound (K1) and the fifth Korotkoff sound (K5) were defined as systolic blood pressure (SBP) and diastolic blood pressure (DBP), respectively. According to the 2010 Chinese BP reference standards for children and adolescents,[19] HTN was defined as SBP and/or DBP ≥95th age- and gender-specific percentile; elevated BP was defined as SBP and/or DBP ≥90th age- and gender-specific percentile (Supplemental table 1).

Assessment of height, weight, and body mass index

Standing height was measured without shoes to the nearest 0.1 cm by using a portable mounted stadiometer with a movable headpiece. Weight was measured in light clothing to the nearest 0.1 kg by using a body composition analyzer (TBF-410; TANITA Corporation, Tokyo, Japan). The weight of clothes was estimated and then deducted. BMI was calculated as body weight (in kilograms) divided by the square of height (in meters). The heights and weights of parents were self-reported and used to calculate parental BMI. The overweight status of parents was defined based on Chinese adult criteria as having BMI ≥24.0 kg/m.[2,20]

Measurements of covariates

Information on potential confounders was collected through a self-administered questionnaire to parents, including children's birth date, birth weight, feeding approach during infancy (breast milk only, bottle milk only, or mixed), parental education levels (high school and below, undergraduate, or graduate and above), and parental medical history of HTN and diabetes. Both feeding status and parents' education level are associated with parents' lifestyle, which have been shown to influence children's BP.[21-24] Thus, they were considered as potential confounders in the analysis. The questionnaire was designed, modified, and evaluated by a group of four experts in China.[25]

Statistical analysis

Characteristics of children were presented as means ± standard deviations for continuous variables and proportions for categorical variables. Any differences by HTN status or jointly classified parental weight status were determined by using a t-test, analysis of variance, a chi-squared test, a Wilcoxon rank sum test, or a Kruskal-Wallis equality-of-populations rank test, as appropriate. Multiple logistic regression was used to examine the association between paternal and/or maternal BMI levels and risk of childhood HTN. Model I was adjusted for age and gender. Model II was further adjusted for birth weight, feeding approach during infancy,

parents' education levels, and parental medical history of HTN and diabetes. Parents' BMI levels were considered both categorically (overweight versus normal weight) and continuously (in one unit increments). To assess whether the association was independent of children's weight status, the model was additionally adjusted for children's BMI. To explore the joint association of paternal and maternal BMI with risk of childhood HTN, we categorized the sample into four subgroups: children with both parents being normal weight (the reference), children with only the paternal parent overweight, children with only the maternal parent overweight, and children with both parents overweight. All analyses were performed by using STATA statistical software (Version 13.0; STATA Corporation LP, College Station, Texas, USA). A two-sided *p* value ≤0.05 was considered statistically significant.

RESULTS

The prevalence of childhood HTN was 8.4% with no significant gender difference (boys vs. girls: 9.0% vs. 7.8%, p=0.36). The prevalence appeared rapidly increased after 11 years old (Figure 1). Table 1 presents the characteristics of 1,949 children stratified by hypertensive status (yes vs. no). The average age, BMI, and birth weight was 8.7±1.7 years, 17.4±3.3 kg/m^2, and 3.3±0.3 kg, respectively. Compared with non-hypertensive kids, hypertensive children had higher BMI levels and higher parental BMI levels. They were more likely to have a parental medical history of HTN.

Table 2 presents the characteristics of the study population based on parent weight status. Compared to children with two parents of normal weight, children with both parents being overweight had higher BMI levels. They were more likely to be boys, have parents with lower education levels, and have a medical history of HTN and diabetes.

Table 3 shows the association between parental BMI levels and risk of childhood HTN. The prevalence of childhood HTN was significantly higher for children with mothers being overweight as compared with those with mothers of normal weight (12.5% vs 7.4%, $p<0.01$). No significant difference was found in paternal weight status ($p=0.10$). After adjustment for potential confounders, maternal overweight status (yes vs no), but not paternal overweight status, was significantly associated with prevalence of childhood HTN (OR=1.78; 95% CI: 1.22, 2.59). A significant linear trend was found for maternal BMI (OR=1.08; 95% CI: 1.03, 1.14) and a marginally significant linear trend for paternal BMI (OR=1.05; 95% CI: 0.997, 1.11, $p=0.07$). The observed associations were attenuated and became statistically non-significant after further adjustment for children's BMI.

Table 4 shows the associations between jointly classified parental BMI levels with risk of childhood HTN. After adjustment for potential confounders, children with fathers being overweight (OR=1.95; 95% CI: 1.08, 3.51) and children with both parents of overweight (OR=2.09; 95% CI: 1.26, 3.46) had a significantly higher prevalence of HTN as compared to those with two parents of normal weight. Similarly, the observed associations were attenuated and became non-significant after further adjustment for children's BMI.

DISCUSSION

Findings from this cross-sectional study suggest that parental weight status, especially maternal weight status, was associated with a higher prevalence of childhood HTN, presumably through influencing children's weight.

Data directly relating parents' weight status to risk of childhood HTN are lacking. Previous studies mainly focused on the association between maternal BMI during pregnancy or pre-pregnancy with offspring's BP,[26,27] which reported that pregnant women's BMI levels were positively related to childhood SBP at age 5. The findings from animal studies also suggested that perinatal exposure to the metabolic milieu of maternal obesity (e.g., the concentration of leptin) might change the central regulatory pathways involved in BP regulation in offspring,[28] which support a role of maternal BMI in offspring's BP. In addition, studies indicated that most cases of childhood HTN might be attributable to children being overweight/obese.[29,30] For example, in a large-scale cross-sectional study conducted in Brazil, children with overweight and obesity status had a 3.6 times higher risk of elevated SBP and a 2.7 times higher risk of elevated DBP as compared with peers of normal-weight.[31] Evidence from clinical trials also indicated that the intervention against childhood obesity had a favorable effect on BP reduction in children.[32,33] In the present study, we considered parental weight status as a surrogate of parents' diet and lifestyle characteristics.[34] Since children's diets are largely dependent on parents' choices, those who live in a family with an unhealthy diet, such as one with high sodium[27] and low fruit and vegetable intake,[35,36] may have a higher risk of HTN. Moreover, parents are usually role models for their children. If parents have unhealthy behavior, e.g., low physical activity[37] and/or prolonged screen time,[38] their children are likely to adopt the same lifestyle, which may increase their risk of obesity and consequently HTN. The fact that the association was substantially attenuated and became statistically non-significant after adjustment for children's BMI suggests that the possible influence of parents on children's BP or childhood HTN may be mediated through children's weight. Indeed, our mediation analysis further support this hypothesis (data not shown). Of note, our findings

remained after adjustment for parental medical history of HTN, a marker of inherited genetic impairment on BP regulation. Furthermore, we found that the prevalence of HTN increased rapidly at the age of 11 to 12 years. BP usually increases with age and height. Changes in BP may accelerate during puberty caused by complicated physical and physiological development, e.g., the elevation of gonadal hormones and growth hormone may accelerate BP increase.[39-42] It was reported that the rise of BP during adolescence (>11 years) was steeper than that in the age group of 6-10 years.[41] The prevalence of pre-HTN was also higher in puberty than that in pre-pubertal cohort.[40]

Our study has some strengths, including a large sample size compared to previous studies. Also, BPs were measured by clinicians rather than being from self-reported estimates. Some limitations need to be acknowledged. First, the cross-sectional study design does not allow the assessment of a temporal relationship between parental weight status and childhood HTN. A reverse causation is possible but unlikely based on evidence in previous studies and the biological mechanism. Because of the limited data in the literature regarding the influence of parents' lifestyles on their offspring's BP, this study generates useful data for future prospective or intervention studies. Second, similar to other observational studies, residual confounding from genetic, dietary, lifestyle, or environmental factors could not be completely ruled out. However, the fact that the significant association remained after adjustment for many other important confounders suggested by the literature indicates that our findings should not be substantially biased. Third, parental BMI was calculated from self-reported height and weight. But self-reported BMI is highly correlated with the value measured by medical professionals in adults (weighted kappa=0.73),[43] which ensures the quality of the data. Fourth, the analysis was not stratified by gender because of insufficient sample size in each age group, though no significant difference in HTN prevalence was observed in this cohort.

In conclusion, findings from this cross-sectional study support the hypothesis that parental weight status is associated with the prevalence of childhood HTN, presumably by affecting children's weight. Because parental weight status is considered as a marker of the diet and lifestyle choices in a family, actions focusing on improving health awareness and changing unhealthy behaviors among parents may exert critical influence on the risk of childhood HTN.

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CONFLICT OF INTEREST AND FUNDING DISCLOSURE

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Table 1. Characteristics of 1,949 school children aged 6 to 12 stratified by their hypertensive status[†]

Characteristics	Total (*n*=1,949)	Hypertension[‡]		*p* value[§]
		No (*n*=1,786)	Yes (*n*=163)	
Age, year	8.7±1.7	8.7±1.7	9.0±1.9	0.03
BMI, kg/m^2	17.4±3.3	17.2±3.1	19.8±4.6	<0.01
Birth weight, kg	3.3±0.3	3.3±0.3	3.3±0.3	0.90
Girl, %	51.9	52.2	48.5	0.36
Information on nutrition in infants, %				
Breast milk only	50.4	50.6	49.1	0.59
Bottle milk only	17.5	17.7	15.5	
Mixed	32.1	31.8	35.4	
Paternal/maternal education, %[d]				
High school and below	50.3	50.3	51.0	0.80
Undergraduate	44.5	44.5	44.6	
Graduate and above	5.2	5.3	4.5	
Father's BMI, kg/m^2	24.2±3.1	24.2±3.0	24.8±3.1	0.01
Mother's BMI, kg/m^2	21.8±2.8	21.7±2.8	22.4±2.7	<0.01
Parental medical history of hypertension, %	8.1	7.3	16.0	<0.01
Parental medical history of diabetes, %	1.4	1.5	1.2	0.81

BMI: body mass index; DBP: diastolic blood pressure; SBP: systolic blood pressure.
[†]Data were means±standard deviations or percentages (%).
[‡]Children's HTN was defined as SBP and/or DBP≥age and gender specific 95th percentile.
[§]*p* values were obtained by using the t-test, chi-squared test, or Wilcoxon rank sum test, as appropriate

Table 2. Characteristics of 1,949 school children aged 6 to 12 stratified by their hypertensive status[†]

Characteristics	NW parents (*n*=806)	OW father, NW mother (*n*=768)	NW father, OW mother (*n*=151)	OW parents (*n*=224)	*p* value[‡]
Age, year	8.7±1.7	8.7±1.7	8.9±1.8	9.0±1.8	0.10
BMI, kg/m^2	16.4±2.7	17.7±3.2	18.1±3.5	19.3±4.4	<0.01
Birth weight, kg	3.3±0.3	3.3±0.3	3.3±0.3	3.4±0.3	0.06
Girl, %	55.3	48.1	53.6	51.8	0.04
Information on nutrition in infants					
Breast milk only	51.2	49.7	50.3	50.2	0.38
Bottle milk only	17.4	17.0	23.5	15.2	
Mixed	31.4	33.3	26.2	34.6	
Parents' education, %					
High school and below	45.6	49.7	66.0	59.2	<0.01
Undergraduate	49.6	43.7	31.3	37.2	
Graduate and above	4.8	6.6	2.7	3.7	
Parental medical history of hypertension, %	3.5	10.7	6.0	17.0	<0.01
Parental medical history of diabetes, %	0.3	2.3	0.7	3.1	<0.01

NW: normal weight; OW: overweight.
[†]Data were means±standard deviations or percentages (%).
[‡]*p* values were obtained by using analysis of variance, a chi-squared test, or a Kruskal-Wallis equality-of-populations rank test, as appropriate

Table 3. The association between parental BMI levels and prevalence of hypertension[†]

	Paternal BMI level			Maternal BMI level		
	NW	OW	↑ 1 kg/m^2	NW	OW	↑ 1 kg/m^2
No. of events / participants	70/957	93/992	--	116/1,574	47/375	--
Prevalence, %	7.3	9.4	--	7.4	12.5	--
Model 1[‡]	1.00 (Ref.)	1.31 (0.95, 1.81)	1.07 (1.01, 1.12)	1.00 (Ref.)	1.80 (1.26, 2.58)	1.09 (1.03, 1.14)
Model 2[§]	1.00 (Ref.)	1.31 (0.94, 1.81)	1.06 (1.01, 1.12)	1.00 (Ref.)	1.77 (1.23, 2.53)	1.08 (1.03, 1.14)
Model 3[¶]	1.00 (Ref.)	1.27 (0.90, 1.79)	1.05 (0.997, 1.11)	1.00 (Ref.)	1.78 (1.22, 2.59)	1.08 (1.03, 1.14)
Model 4[††]	1.00 (Ref.)	0.96 (0.67, 1.37)	0.999 (0.94, 1.06)	1.00 (Ref.)	1.27 (0.85, 1.90)	1.02 (0.96, 1.08)

BMI: body mass index; CI: confidence interval; NW: normal weight; OR: odds ratio; OW: overweight.
[†]OR (95% CI) was estimated by using a logistic regression model. Hypertension was defined as SBP and/or DBP ≥age- and gender-specific 95th percentile.
[‡]Model 1 was unadjusted model.
[§]Model 2 was adjusted for children's age and gender.
[¶]Model 3 (final model) was additionally adjusted for birth weight, feeding status in infants (breast milk only, bottle milk only, or mixed), parents' education (high school and below, undergraduate, graduate and above), parental medical history of hypertension (yes vs. no), and diabetes (yes vs. no).
[††]Model 4 (sensitivity analysis) was additionally adjusted for children's BMI.

Table 4. The association between joint classification of parental BMI levels and the prevalence of hypertension[†]

	Parental BMI levels [(paternal level, maternal level)]			
	(NW, NW)	(NW, OW)	(OW, NW)	(OW, OW)
No. of events / participants	53/806	63/768	17/151	30/224
Prevalence, %	6.6	8.2	11.3	13.4
Model 1[‡]	1.00 (Ref.)	1.27 (0.87, 1.86)	1.77 (0.99, 3.15)	2.15 (1.33, 3.45)
Model 2[§]	1.00 (Ref.)	1.27 (0.87, 1.85)	1.77 (0.99, 3.15)	2.15 (1.33, 3.45)
Model 3[¶]	1.00 (Ref.)	1.28 (0.86, 1.90)	1.95 (1.08, 3.51)	2.09 (1.26, 3.46)
Model 4[††]	1.00 (Ref.)	1.00 (0.67, 1.50)	1.42 (0.77, 2.62)	1.18 (0.68, 2.05)

BMI: body mass index; CI: confidence interval; NW: normal weight; OR: odds ratio; OW: overweight.
[†]OR (95% CI) was estimated by using a logistic regression model. Elevated blood pressure and hypertension were defined as SBP and/or DBP ≥age and gender specific 90th percentile and 95th percentile, respectively.
[‡]Model 1 was unadjusted model.
[§]Model 2 was adjusted for children's age and gender.
[¶]Model 3 (final model) was additionally adjusted for birth weight, feeding status in infants (breast milk only, bottle milk only, or mixed), parents' education (high school and below, undergraduate, graduate and above), parental medical history of hypertension (yes vs. no), and diabetes (yes vs. no).
[††]Model 4 (sensitivity analysis) was additionally adjusted for children's BMI.

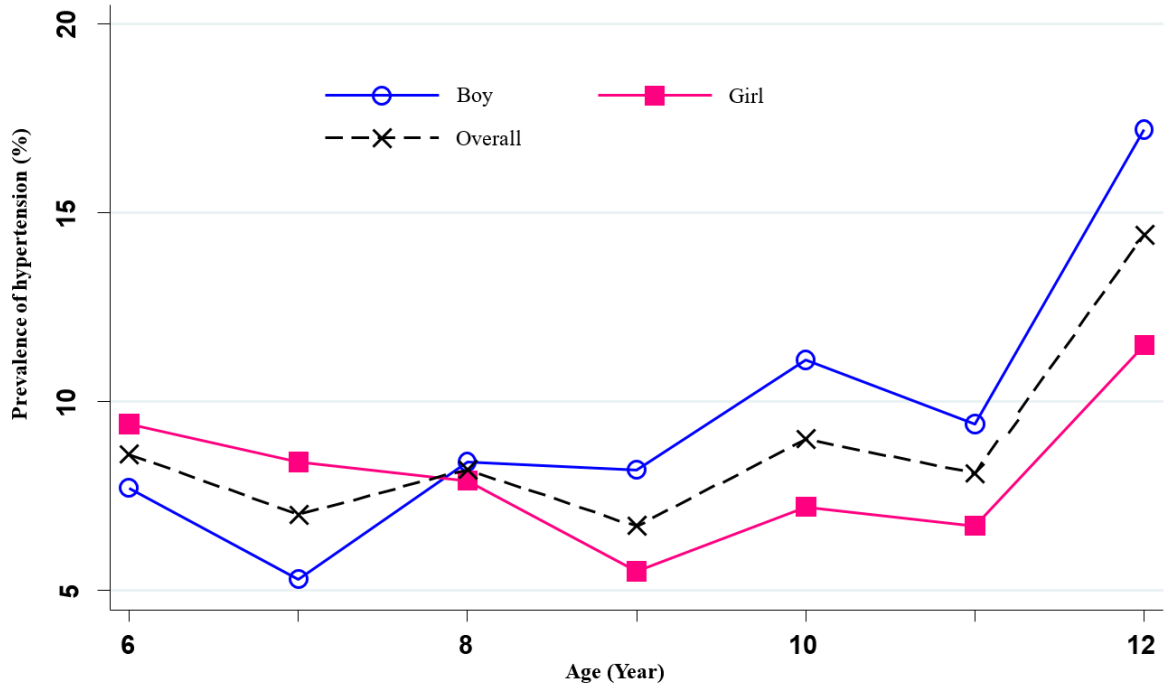

Figure 1. The prevalence of hypertension by gender in 1,949 children aged 6 to 12. The prevalence of hypertension was not statistically significantly different over time between boys and girls.

Supplementary table 1. Recommended blood pressure reference cut-offs for Chinese boys and girls (mmHg)[†]

Age/years	Boys				Girls			
	SBP		DBP-K5		SBP		DBP-K5	
	P_{90}	P_{95}	P_{90}	P_{95}	P_{90}	P_{95}	P_{90}	P_{95}
6	108	112	69	73	106	110	69	72
7	111	115	71	74	108	112	70	73
8	113	117	72	76	111	115	71	74
9	114	119	74	77	112	117	72	76
10	115	120	74	78	114	118	73	77
11	117	122	75	78	116	121	74	77
12	119	124	75	78	117	122	75	78

SBP: systolic blood pressure; DBP: diastolic blood pressure.
[†]The recommended blood pressure reference cut-offs were based on the 2010 Chinese BP reference standards for children and adolescents.[23]