

Original Article

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

Liping Lu MD, PhD^{1,2,3}, Cheng Chen PhD^{1,2}, Pengcheng Xun MD, PhD⁴, Jialu Wang MS³, Yanping Wan MD³, Ka He MD, ScD^{1,2}

¹Department of Obstetrics and Gynecology, Vagelos College of Physicians and Surgeons, Columbia University, New York, USA

²Department of Epidemiology, Mailman School of Public Health, Columbia University, New York, USA

³Department of Clinical Nutrition, Ren Ji Hospital, School of Medicine, Shanghai Jiao Tong University, Shanghai, China

⁴Department of Epidemiology and Biostatistics, School of Public Health-Bloomington, Indiana University, Indiana, USA

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 \geq 95th and \geq 90th age- and gender-specific percentile, respectively. Parental overweight was defined as BMI \geq 24.0 kg/m². **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 insuffi-

Corresponding Author: Dr. Liping Lu, Department of Obstetrics and Gynecology, Vagelos College of Physicians and Surgeons, Columbia University, 622 West 168th Street, New York, USA.

Tel: +1-212-305-6973

Email: ll3407@cumc.columbia.edu

Manuscript received 13 May 2019. Initial review completed 13 July 2019. Revision accepted 26 August 2019.

doi: 10.6133/apjcn.201912_28(4).0018

cient 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.

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 ≥ 95 th age- and gender-specific percentile; elevated BP was defined as SBP and/or DBP ≥ 90 th 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/m².²⁰

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 \pm 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 \pm 1.7 years, 17.4 \pm 3.3 kg/m², and 3.3 \pm 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

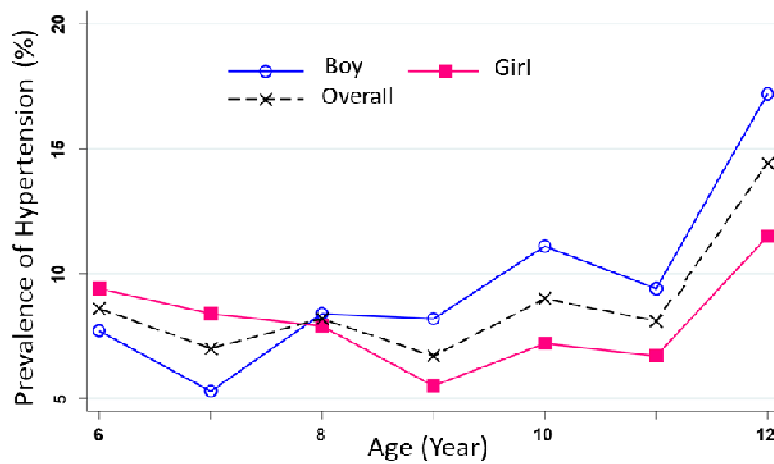


Figure 1. The prevalence of hypertension by gender in 1,949 children aged 6 to 12. The prevalence of hypertension was not statistically significantly different over time between boys and girls.

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, %				
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

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

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

Characteristics	NW parents (n=806)	OW father, NW mother (n=768)	NW father, OW mother (n=151)	OW parents (n=224)	<i>p</i> 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.

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 sodium²⁷ 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 activity³⁷ 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

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 \geq 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.

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 par-

ents may exert critical influence on the risk of childhood HTN.

ACKNOWLEDGEMENTS

We extremely grateful to all the participated children and their parents in this study.

AUTHOR DISCLOSURES

The authors have no conflict of interest to report. Liping Lu, Jialu Wang and Yanping Wan are partially supported by Health Science and Technology Joint Research Project of Pudong New Area Health and Family Planning Commission (Grant No. PW2016D-05).

REFERENCES

- Ostchega Y, Carroll M, Prineas RJ, McDowell MA, Louis T, Tilert T. Trends of elevated blood pressure among children and adolescents: data from the National Health and Nutrition Examination Survey 1988-2006. *Am J Hypertens.* 2009;22:59-67. doi: 10.1038/ajh.2008.312.
- Din-Dzietham R, Liu Y, Bielo MV, Shamsa F. High blood pressure trends in children and adolescents in national surveys, 1963 to 2002. *Circulation.* 2007;116:1488-96. doi: 10.1161/CIRCULATIONAHA.106.683243.
- Xi B, Li H, Li S, Mi J. Recent prevalence of hypertension among Chinese children and adolescents based on 2010 China national blood pressure references. *Int J Cardiol.* 2014;174:870-1. doi: 10.1016/j.ijcard.2014.04.214.
- Daniels SR. Cardiovascular sequelae of childhood hypertension. *Am J Hypertens.* 2002;15:61S-3S. doi: 10.1016/s0895-7061(01)02305-6.
- Thompson M, Dana T, Bougatsos C, Blazina I, Norris SL. Screening for hypertension in children and adolescents to prevent cardiovascular disease. *Pediatrics.* 2013;131:490-525. doi: 10.1542/peds.2012-3523.
- Dyson PA, Anthony D, Fenton B, Matthews DR, Stevens DE. High rates of child hypertension associated with obesity: a community survey in China, India and Mexico. *Paediatr Int Child Health.* 2014;34:43-9. doi: 10.1179/2046905513Y.0000000079.
- Spagnolo A, Giussani M, Ambruzzi AM, Bianchetti M, Maringhini S, Matteucci MC et al. Focus on prevention, diagnosis and treatment of hypertension in children and adolescents. *Ital J Pediatr.* 2013;39:20. doi: 10.1186/1824-7288-39-20.
- Torrance B, McGuire KA, Lewanczuk R, McGavock J. Overweight, physical activity and high blood pressure in

- children: a review of the literature. *Vasc Health Risk Manag.* 2007;3:139-49.
9. Moyer VA. Screening for primary hypertension in children and adolescents: U.S. Preventive Services Task Force recommendation statement. *Pediatrics.* 2013;132:907-14. doi: 10.1542/peds.2013-2864.
 10. Betz HH, Eisenmann JC, Laurson KR, DuBose KD, Reeves MJ, Carlson JJ, Pfeiffer KA. Physical activity, BMI, and blood pressure in US youth: NHANES 2003-2006. *Pediatr Exerc Sci.* 2018;30:418-25. doi: 10.1123/pes.2017-0127.
 11. Noubiap JJ, Essouma M, Bigna JJ, Jingi AM, Aminde LN, Nansseu JR. Prevalence of elevated blood pressure in children and adolescents in Africa: a systematic review and meta-analysis. *Lancet Public Health.* 2017;2:e375-86. doi: 10.1016/S2468-2667(17)30123-8.
 12. Parker ED, Sinaiko AR, Kharbanda EO, Margolis KL, Daley MF, Trower NK et al. Change in weight status and development of hypertension. *Pediatrics.* 2016;137:e20151662. doi: 10.1542/peds.2015-1662.
 13. Dubois L, Girard M. Early determinants of overweight at 4.5 years in a population-based longitudinal study. *Int J Obes (Lond).* 2006;30:610-7. doi: 10.1038/sj.ijo.0803141.
 14. Bouchard C. Childhood obesity: are genetic differences involved? *Am J Clin Nutr.* 2009;89:1494S-501S. doi: 10.3945/ajcn.2009.27113C.
 15. Perez-Rodrigo C, Gianzo-Citores M, Gil A, Gonzalez-Gross M, Ortega RM, Serra-Majem L, Varela-Moreiras G, Aranceta-Bartrina J. Lifestyle patterns and weight status in Spanish adults: The ANIBES Study. *Nutrients.* 2017;9:606. doi: 10.3390/nu9060606.
 16. Chau JY, Grunseit AC, Chey T, Stamatakis E, Brown WJ, Matthews CE, Bauman AE, van der Ploeg HP. Daily sitting time and all-cause mortality: a meta-analysis. *PLoS One.* 2013;8:e80000. doi: 10.1371/journal.pone.0080000.
 17. Teixeira PJ, Silva MN, Mata J, Palmeira AL, Markland D. Motivation, self-determination, and long-term weight control. *Int J Behav Nutr Phys Act.* 2012;9:22. doi: 10.1186/1479-5868-9-22.
 18. Arabshahi S, Ibiebele TI, Hughes MCB, Lahmann PH, Williams GM, van der Pols JC. Dietary patterns and weight change: 15-year longitudinal study in Australian adults. *Eur J Nutr.* 2017;56:1455-65. doi: 10.1007/s00394-016-1191-3.
 19. Mi J, Wang T, Meng L, Zhu G, Han S, Zhong Y et al. Development of blood pressure reference standards for Chinese children and adolescents. *Chin J Evid Based Pediatr.* 2010;5:4-14. (In Chinese)
 20. Zhou BF. Predictive values of body mass index and waist circumference for risk factors of certain related diseases in Chinese adults--study on optimal cut-off points of body mass index and waist circumference in Chinese adults. *Biomed Environ Sci.* 2002;15:83-96.
 21. Martin RM, Ness AR, Gunnell D, Emmett P, Davey Smith G. Does breast-feeding in infancy lower blood pressure in childhood? The Avon Longitudinal Study of Parents and Children (ALSPAC). *Circulation.* 2004;109:1259-66. doi: 10.1161/01.CIR.0000118468.76447.CE.
 22. Amorim Rde J, Coelho AF, de Lira PI, Lima Mde C. Is breastfeeding protective for blood pressure in schoolchildren? A cohort study in northeast Brazil. *Breastfeed Med.* 2014; 9:149-56. doi: 10.1089/bfm.2013.0118.
 23. Bouthoorn SH, Van Lenthe FJ, De Jonge LL, Hofman A, Van Osch-Gevers L, Jaddoe VW, Raat H. Maternal educational level and blood pressure, aortic stiffness, cardiovascular structure and functioning in childhood: the generation R study. *Am J Hypertens.* 2014;27:89-98. doi: 10.1093/ajh/hpt180.
 24. Kwok MK, Schooling CM, Subramanian SV, Leung GM, Kawachi I. Pathways from parental educational attainment to adolescent blood pressure. *J Hypertens.* 2016;34:1787-95. doi: 10.1097/HJH.0000000000001003.
 25. Wan Y, Xu R, Feng H, Zhou Y, Zhang X, Lu L, Tan T, Jiang Y, Chen Z, Wu Y. Is parental body weight related with their children's overweight and obesity in Gao Hang Town, Shanghai? *Asia Pac J Clin Nutr.* 2015;24:509-14. doi: 10.6133/apjcn.2015.24.3.09.
 26. Lawlor DA, Najman JM, Sterne J, Williams GM, Ebrahim S, Davey Smith G. Associations of parental, birth, and early life characteristics with systolic blood pressure at 5 years of age: findings from the Mater-University study of pregnancy and its outcomes. *Circulation.* 2004;110:2417-23. doi: 10.1161/01.CIR.0000145165.80130.B5.
 27. Oostvogels AJ, Stronks K, Roseboom TJ, van der Post JA, van Eijsden M, Vrijkotte TG. Maternal prepregnancy BMI, offspring's early postnatal growth, and metabolic profile at age 5-6 years: the ABCD Study. *J Clin Endocrinol Metab.* 2014;99:3845-54. doi: 10.1210/jc.2014-1561.
 28. Taylor PD, Samuelsson AM, Poston L. Maternal obesity and the developmental programming of hypertension: a role for leptin. *Acta Physiol (Oxf).* 2014;210:508-23. doi: 10.1111/apha.12223.
 29. Fowokan AO, Sakakibara BM, Onsel N, Punthakee Z, Waddell C, Rosin M, Lear SA. Correlates of elevated blood pressure in healthy children: a systematic review. *Clin Obes.* 2018;8:366-81. doi: 10.1111/cob.12271.
 30. Jackson SL, Zhang Z, Wiltz JL, Loustalot F, Ritchey MD, Goodman AB, Yang Q. Hypertension among youths - United States, 2001-2016. *MMWR Morb Mortal Wkly Rep.* 2018;67:758-62. doi: 10.15585/mmwr.mm6727a2.
 31. Ribeiro RQ, Lotufo PA, Lamounier JA, Oliveira RG, Soares JF, Botter DA. Additional cardiovascular risk factors associated with excess weight in children and adolescents: the Belo Horizonte heart study. *Arq Bras Cardiol.* 2006;86: 408-18. doi: 10.1590/S0066-782X2006000600002. (In Portuguese)
 32. Cai L, Wu Y, Wilson RF, Segal JB, Kim MT, Wang Y. Effect of childhood obesity prevention programs on blood pressure: a systematic review and meta-analysis. *Circulation.* 2014;129:1832-9. doi: 10.1161/CIRCULATIONAHA.113.005666.
 33. Hvidt KN, Olsen MH, Ibsen H, Holm JC. Effect of changes in BMI and waist circumference on ambulatory blood pressure in obese children and adolescents. *J Hypertens.* 2014;32:1470-7. doi: 10.1097/HJH.000000000000188.
 34. Avis JL, Jackman A, Jetha MM, Ambler KA, Krug C, Sivakumar M, Ball GD. Lifestyle behaviors of parents of children in pediatric weight management: Are they meeting recommendations? *Clin Pediatr (Phila).* 2015;54:1068-75. doi: 10.1177/0009922814566930.
 35. Shi L, Krupp D, Remer T. Salt, fruit and vegetable consumption and blood pressure development: a longitudinal investigation in healthy children. *Br J Nutr.* 2014;111:662-671. doi: 10.1017/S0007114513002961.
 36. Novotny R, Nigg CR, Li F, Wilkens LR. Pacific kids DASH for health (PacDASH) randomized, controlled trial with DASH eating plan plus physical activity improves fruit and vegetable intake and diastolic blood pressure in children. *Child Obes.* 2015;11:177-86. doi: 10.1089/chi.2014.0141.
 37. Gopinath B, Hardy LL, Teber E, Mitchell P. Association between physical activity and blood pressure in prepubertal children. *Hypertens Res.* 2011;34:851-5. doi: 10.1038/hr.2011.46.
 38. Gopinath B, Baur LA, Hardy LL, Kifley A, Rose KA, Wong TY, Mitchell P. Relationship between a range of sedentary

- behaviours and blood pressure during early adolescence. *J Hum Hypertens.* 2012;26:350-6. doi: 10.1038/jhh.2011.40.
39. Ataei N, Hosseini M, Fayaz M, Navidi I, Taghiloo A, Kalantari K, Ataei F. Blood pressure percentiles by age and height for children and adolescents in Tehran, Iran. *J Hum Hypertens.* 2016;30:268-77. doi: 10.1038/jhh.2015.52.
40. Liang Y, Mi J. Pubertal hypertension is a strong predictor for the risk of adult hypertension. *Biomed Environ Sci.* 2011;24:459-66. doi: 10.3967/0895-3988.2011.05.002.
41. Sayeemuddin M, Sharma D, Pandita A, Sultana T, Shastri S. Blood pressure profile in school children (6-16 years) of southern India: a prospective observational study. *Front Pediatr.* 2015;3:24. doi: 10.3389/fped.2015.00024.
42. Shankar RR, Eckert GJ, Saha C, Tu W, Pratt JH. The change in blood pressure during pubertal growth. *J Clin Endocrinol Metab.* 2005;90:163-7. doi: 10.1210/jc.2004-0926.
43. Skeie G, Mode N, Henningsen M, Borch KB. Validity of self-reported body mass index among middle-aged participants in the Norwegian Women and Cancer study. *Clin Epidemiol.* 2015;7:313-23. doi: 10.2147/CLEP.S83839.

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.¹⁹