

BMJ Open Relationship between parental overweight and obesity and childhood metabolic syndrome in their offspring: result from a cross-sectional analysis of parent-offspring trios in China

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ABSTRACT

Objective Metabolic syndrome (MS) and its components are observed to emerge in childhood and may continue into adulthood. The study aimed to investigate the association between parental overweight and risk of childhood MS and its components in their offspring.

Methods Data were obtained from a cross-sectional survey conducted in Chinese children and adolescents; a total of 11 784 children aged 7–18 years were included in this study; child outcomes were obtained from objective measurements and parental data were obtained from questionnaires; MS was defined according to the modified criteria of Adult Treatment Panel III; correlation between parental overweight and offspring MS was assessed via multivariate logistic regression models adjusted for potential covariates.

Results 3476 (29.5%) children were found to have overweight fathers, 1041 (8.8%) had overweight mothers and 852 (7.2%) had both overweight parents. The prevalence of MS was 7.1% in total, 8.2% in boys and 5.9% in girls; children with overweight parents had a higher prevalence of MS and its components (except for elevated glucose) compared with children with normal-weight parents. Children with overweight fathers, mothers and both parents had 2.17 times (95% CI: 1.65–2.85), 2.89 times (95% CI: 2.03–4.11) and 2.81 times (95% CI: 1.91–4.15) higher risk of MS, respectively. Children with overweight mothers were likely to have a higher risk of MS compared with children with overweight fathers. Parental overweight was positively correlated with higher risk of MS, abdominal obesity and low HDL-C both in boys and girls.

Conclusion Parental overweight was strongly associated with increased risk of MS in their offspring, the risk was highest in children with both parents to be overweight. Maternal overweight seems to have a stronger correlation with offspring MS than paternal overweight. Parental overweight is one of the factors for identifying metabolic dysfunction risk in their offspring and other factors need to be considered as well.

INTRODUCTION

The metabolic syndrome (MS), known as a cluster of five cardiometabolic risk factors

Strengths and limitation of this study

- This study included a large number of parent-offspring trios and allows us to compare correlations between father-offspring and mother-offspring pairs.
- Data were from a cross-sectional survey, it limited us to make casual inferences.
- Information on the parental height and weight was recalled from the questionnaire, which was not as accurate as measured directly.
- We only collected information about parental current body mass index (BMI) information, but unable to obtain BMI status before and during pregnancy, which was regarded as an important factor affecting the health status of their offspring.

including dyslipidaemia, abnormal glucose and insulin regulation, abdominal obesity and hypertension, has attracted numerous public health concern worldwide.¹ According to the estimate, MS was observed in 20%–25% of adults globally.² In China, the prevalence in the adult population has rapidly increased and was estimated to be 15.5% in 2017²; it was predicted that China will be one of the highest risk areas in the world.

MS has been known as a risk factor for the development of cardiovascular disease,^{3 4} type 2 diabetes (T2D)⁵ and even all-cause mortality.⁶ Although most MS incidence occurred in the adult (especially the elder) population, it has been recognised that the causes or precursors are beginning to emerge in early life.^{7–10} Previous studies have demonstrated that children with metabolic disorders were associated with an increased risk for developing MS and T2D in later life,¹¹ and this risk can be reduced to normal by resolving MS before adulthood.¹² Thus, preventing and managing MS from childhood

is important for preventing the development of MS and cardiovascular disorders in later life. And MS in children and adolescents needs more public concern, even though the prevalence was relatively lower than adults.

The high burden of MS heightens the needs for the investigation of its determinants. Previously studies have found that MS and its components tend to occur in familial aggregates; parents play an important role in offspring health conditions. A prospective study from the Netherlands reported that higher paternal and maternal pregnancy BMI were correlated with higher childhood BMI, systolic blood pressure (BP) and insulin levels and lower high-density lipoprotein cholesterol levels.¹³ Drake and Reynolds¹⁴ summarised results from both animal and human studies and concluded that maternal obesity during pregnancy has a profound impact on offspring cardiovascular and metabolic dysregulation, which may result in an 'intergenerational cycle'. However, most of those works of literature focused on parental weight status in pre-pregnancy or during gestational period¹³⁻¹⁹; few have focused on this relationship after the pregnancy period; whether the parental weight status several years after conception adds the risk of childhood MS in their offspring has not yet been well studied. Besides, it is also unclear whether paternal and maternal adiposities had similar effects on offspring, because more studies had published data on mother-offspring dyads, but few have compared the correlation in parent-offspring trios.¹⁷⁻²¹

The present study aims to: (1) explore associations between parental overweight and MS and its components in their offspring; (2) compare the effects of paternal and maternal overweight on MS and its components; (3) explore whether those associations differ between boys and girls.

METHODS

Study design and sample population

Data in this study come from the baseline cross-sectional survey of a national multicentre cluster-controlled trial addressing the intervention of obesity in children and adolescents from seven provinces or cities of China (Hunan, Ningxia, Tianjin, Chongqing, Liaoning, Shanghai and Guangzhou) (Registration number: NCT02343588). A more detailed description of the study design and conduct can be assessed elsewhere.²² Briefly, a multistage cluster random sampling method was used to determine participants. At first, several regions were randomly selected from each province/city, and 12 to 16 schools were randomly chosen from each region. In each school, two classes were randomly selected in each grade and the whole class students and their parents were invited to participate in this survey, then those who signed the informed consent were enrolled in this study for physical measurement, blood detection and questionnaire survey. All survey sites used the same protocol during the implementation process, and all processes of

randomisation were performed by a staff member who was not involved in the survey.

A number of 11 784 students aged 7–18 years were included in the analytical sample for the present study after excluding participants who did not collect the blood sample, or those who did not have valid data/information on child outcomes, parental weight, height, household income and history of a cardiovascular-related disease.

Data collection

All children enrolled in this study were asked to undergo anthropometric measurements and a venous blood sample collection procedure, which were conducted by trained investigators. Besides, all parents were required to complete a structured self-administered questionnaire at home.

Anthropometric measurements

Anthropometric measurements were conducted according to standard protocol and the measuring instruments were similar at all survey sites. Before measurement, children were required to take off their coat, shoes and wear only their underwear. Waist circumference (WC) was measured with an accuracy of 0.1 cm using a non-elastic tape at the end of a natural breath at the midpoint between the top of the iliac crest and the lower margin of the last palpable rib; every child was measured twice and then averaged as final WC value for data analysis. BP was measured through a mercury sphygmomanometer (model XJ11D, Shanghai Medical Instruments Co, Ltd, China) with the correct cuff size on the right arm of the participants in a relaxed, sitting position. Every participant was measured twice at 1 min intervals, and the readings of systolic BP (SBP) and diastolic BP (DBP) were recorded each time; we used the average of the two measurements on data analysis.

Blood sample collection and detection

The venous blood sample collection procedure was conducted in the morning after an overnight (at least 8 hours) fast: blood samples were collected from the antecubital vein and then transfused into vacuum tubes; blood specimens were transported in a chilled insulated container immediately after collected from venipuncture and then frozen at -80°C after centrifuged at $2000g$ for 10 min; plasma samples collected at each centre were shipped by air in dry ice to the laboratory in Beijing, where the samples were stored at -80°C until later laboratory detections were performed.

Fasting lipid profile (in enzymatic methods), including high-density lipoprotein cholesterol (HDL-C) and triglyceride (TG), and fasting glucose (glucose oxidase method) were measured by the automatic biochemistry analysis system (Cobas c 501, Roche Diagnostics, Risch-Rotkreuz, Switzerland).

Questionnaire survey

The parental questionnaire was performed to collect information about household income, parental height

and weight, family history of hypertension and diabetes. Household income was calculated as a summarise of monthly income (in CNY) of all household members and classified as ≤ 5000 , $5000\text{--}12000$ or ≥ 12000 CNY. Parents were asked to report their height (in centimetre) and weight (in kilogram), and BMI was calculated as the weight (kg) divided by the square of the height (m^2), parental overweight was defined as $\text{BMI} \geq 24 \text{ kg/m}^2$ according to the criteria established by the Working Group on Obesity in China.²³ To obtain a family history of hypertension and diabetes, we asked questions of ‘whether you have been diagnosed with hypertension by a doctor in medical institutions or taken drugs to control blood pressure?’ and ‘whether you have been diagnosed with diabetes mellitus by a doctor in medical institutions or taken drugs to control blood glucose?’ When both parents answered ‘Yes’ or ‘No’, respectively, and either responded ‘Yes’, we defined the child has a family history.

MS definition

No universal diagnostic criteria exist for paediatric MS. In this study, MS was defined according to the modified criteria of Adult Treatment Panel III (ATP III) defined by the National Cholesterol Education Program,²⁴ that is, the presence of three or more of the following five components: (1) central obesity, WC >90 th percentile for sex and age; (2) elevated BP, SBP or DBP >90 th percentile for sex, age and height; (3) elevated TG, $\text{TG} \geq 1.24 \text{ mmol/L}$; (4) low HDL-C, $\text{HDL-C} \leq 1.03 \text{ mmol/L}$; and (5) elevated glucose, fasting glucose $\geq 5.6 \text{ mmol/L}$.

Patient and public involvement

Students and their parents were not involved in study design or outcome measurements, nor were they involved in the recruitment and conduct of the study. School doctors and class teachers helped us to organise and maintain the order of the physical examination by class held in the school.

Statistical analysis

Kolmogorov-Smirnov tests were used to assess the normality of the measured data. Continuous variables were characterised as mean \pm SD, and categorical variables were characterised by frequencies and percentages. Participant children were grouped into four categories according to their parental overweight status (none, father, mother and both parents). A comparison of covariates between boys and girls was performed using the Student t-test for continuous variables and Pearson’s χ^2 test for categorical variables. Differences in the prevalence of MS and its components among parental overweight groups were tested using Pearson’s χ^2 test with Bonferroni correction. Bivariate and multivariate logistic regression models were used to estimate ORs and 95% CI for association among parental weight groups and offspring MS and its components risk. The crude model was a bivariate regression analysis, and the adjusted model was adjusted for sex, age, residence, household income,

family diabetes history and family hypertension history. Furthermore, in order to understand whether the effects of parental overweight status differ between boys and girls, the regression models were processed stratified by sex and adjusted for residence, sex, age, family household income, family diabetes history and family hypertension history. All statistical analyses were conducted using SPSS V.20.0 (IBM) and R software V.3.3.2 for Windows, and a two-sided $p < 0.05$ was considered statistically significant in bivariate tests and a two-sided $p < 0.05$ was considered statistically significant in multiple tests.

RESULTS

Baseline characteristics of the study population

Descriptive characteristics of the entire participants are presented in table 1, stratified by sex. A total of 11 784 children (49.9% boys and 56.1% live in urban areas) were included in this study. Of them, 3476 (29.5%) children had overweight fathers, 1041 (8.8%) children had overweight mothers and 852 (7.2%) children had both overweight parents. About 2.4% of parents had diabetes history and 7.4% had hypertension history. In offspring, the mean age was 11.3 (SD: 3.1) years and we observed significant differences in WC, systolic BP, diastolic BP, TG, HDL-C and fasting glucose levels among four groups.

Prevalence of MS and its components

Figure 1 and online supplemental table S1 summarise the prevalence of MS and its components in offspring stratified by parental weight groups. The prevalence of MS was 7.1% in total, boys (8.2%) have a higher prevalence than girls (5.9%). For MS components, the prevalence of abdominal obesity, elevated BP, elevated TG, low HDL-C and elevated glucose in total participants were 23.1%, 21.9%, 16.9%, 13.8% and 3.4%, respectively. We found that children with overweight parents had a higher prevalence of MS and abdominal obesity, elevated BP, elevated TG and low HDL-C when compared with children whose both parents were not overweight ($P < 0.05$), and those with overweight parents had the highest prevalence of MS and its components (except for elevated glucose). Similar results were observed in boys and girls; in girls, participants with overweight mothers seem to have a relatively higher prevalence of MS, abdominal obesity, elevated BP, elevated TG and low HDL-C compared with those with overweight fathers. In contrast, we only observed a higher prevalence of elevated BP and elevated glucose in boys with overweight mothers compared with overweight fathers.

Comparison of the risk of MS and its components

The associations between parental weight status and offspring MS and its components are shown in figure 2 and online supplemental table S2. In general, parental overweight was observed to be positively associated with a higher risk of paediatric MS both in the crude model and adjusted model, with 1.82 times (95% CI: 1.47–2.26)

Table 1 Descriptive characteristics of the study population stratified by parental OW status

Variables	Total (n=11784)	Parental OW status			P value	
		None (n=6415)	Father (n=3476)	Mother (n=1041)		Both parents (n=852)
Residence area, %						
Urban	6612 (56.1)	3553 (55.4)	2087 (60.0)	497 (47.7)	475 (55.8)	< 0.001
Rural	5172 (43.9)	2862 (44.6)	1389 (40.0)	544 (52.3)	377 (44.2)	
Parental factors						
Paternal BMI (kg/m ²)	24.2±3.2	22.3±1.9	27.5±2.2	22.5±1.8	27.9±2.6	< 0.001
Maternal BMI (kg/m ²)	22.3±3.0	21.1±2.0	21.5±1.9	27.2±2.3	27.4±2.7	< 0.001
Household income (%)						
≤5000	3287 (32.4)	1650 (42.4)	945 (43.0)	388 (57.0)	304 (54.9)	< 0.001
5000–12 000	3024 (29.8)	1648 (42.3)	930 (42.3)	239 (35.1)	207 (37.4)	
≥12 000	1015 (10.0)	597 (15.3)	321 (14.6)	54 (7.9)	43 (7.8)	
Diabetes history						
Yes	259 (2.4)	108 (1.8)	102 (3.2)	26 (2.7)	23 (3.0)	< 0.001
No	10639 (97.6)	5855 (98.2)	3116 (96.8)	921 (97.3)	747 (97.0)	
Hypertension history						
Yes	840 (7.4)	331 (5.4)	307 (9.1)	103 (10.3)	99 (12.1)	< 0.001
No	10510 (92.6)	5840 (94.6)	3060 (90.9)	894 (89.7)	716 (87.9)	
Offspring factors						
Sex						
Boys	5888 (49.9)	3196 (49.8)	1766 (50.8)	508 (48.8)	410 (48.1)	0.434
Girls	5904 (50.1)	3219 (50.2)	1710 (49.2)	533 (51.2)	442 (51.9)	
Age (years)						
7	1477 (12.5)	836 (13.0)	445 (12.8)	105 (10.1)	91 (10.7)	
8	1436 (12.2)	748 (11.7)	473 (13.6)	128 (12.3)	87 (10.2)	
9	1364 (11.6)	720 (11.2)	440 (12.7)	111 (10.7)	93 (10.9)	
10	1321 (11.2)	726 (11.3)	377 (10.8)	95 (9.1)	123 (14.4)	
11	428 (3.6)	217 (3.4)	135 (3.9)	42 (4.0)	34 (4.0)	
12	1366 (11.6)	741 (11.6)	408 (11.7)	118 (11.3)	99 (11.6)	
13	1319 (11.2)	696 (10.8)	359 (10.3)	149 (14.3)	115 (13.5)	
14	418 (3.5)	236 (3.7)	111 (3.2)	44 (4.2)	27 (3.2)	
15	1167 (9.9)	654 (10.2)	335 (9.6)	104 (10.0)	74 (8.7)	
16	1081 (9.2)	608 (9.5)	284 (8.2)	108 (10.4)	81 (9.5)	
17	371 (3.1)	212 (3.3)	101 (2.9)	34 (3.3)	24 (2.8)	
18	36 (0.3)	21 (0.3)	8 (0.2)	3 (0.3)	4 (0.5)	< 0.001
WC	66.0±10.9	64.5±10.1	67.1±11.1	68.0±11.2	70.8±12.5	< 0.001
Systolic BP	104.7±12.0	103.7±11.9	104.9±12.0	107.1±11.8	107.9±12.1	< 0.001
Diastolic BP	66.2±8.9	65.6±8.8	66.4±8.8	67.5±8.8	68.4±9.1	< 0.001
Triglycerides	0.9±0.5	0.9±0.4	0.9±0.5	1.0±0.4	1.0±0.5	< 0.001
HDL-C	1.4±0.3	1.4±0.3	1.4±0.3	1.3±0.3	1.3±0.3	< 0.001
Fasting glucose	4.7±0.6	4.7±0.6	4.7±0.7	4.8±0.6	4.8±0.5	< 0.001

BMI, body mass index; BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; OW, overweight; WC, waist circumference.

risk of MS in children with overweight fathers, 2.36 times (95% CI: 1.77–3.15) higher risk in children with overweight mothers and 2.49 times (95% CI: 1.82–3.41)

higher risk in children with overweight parents, when compared with those whose both parents were not overweight. For individual MS components, the results showed

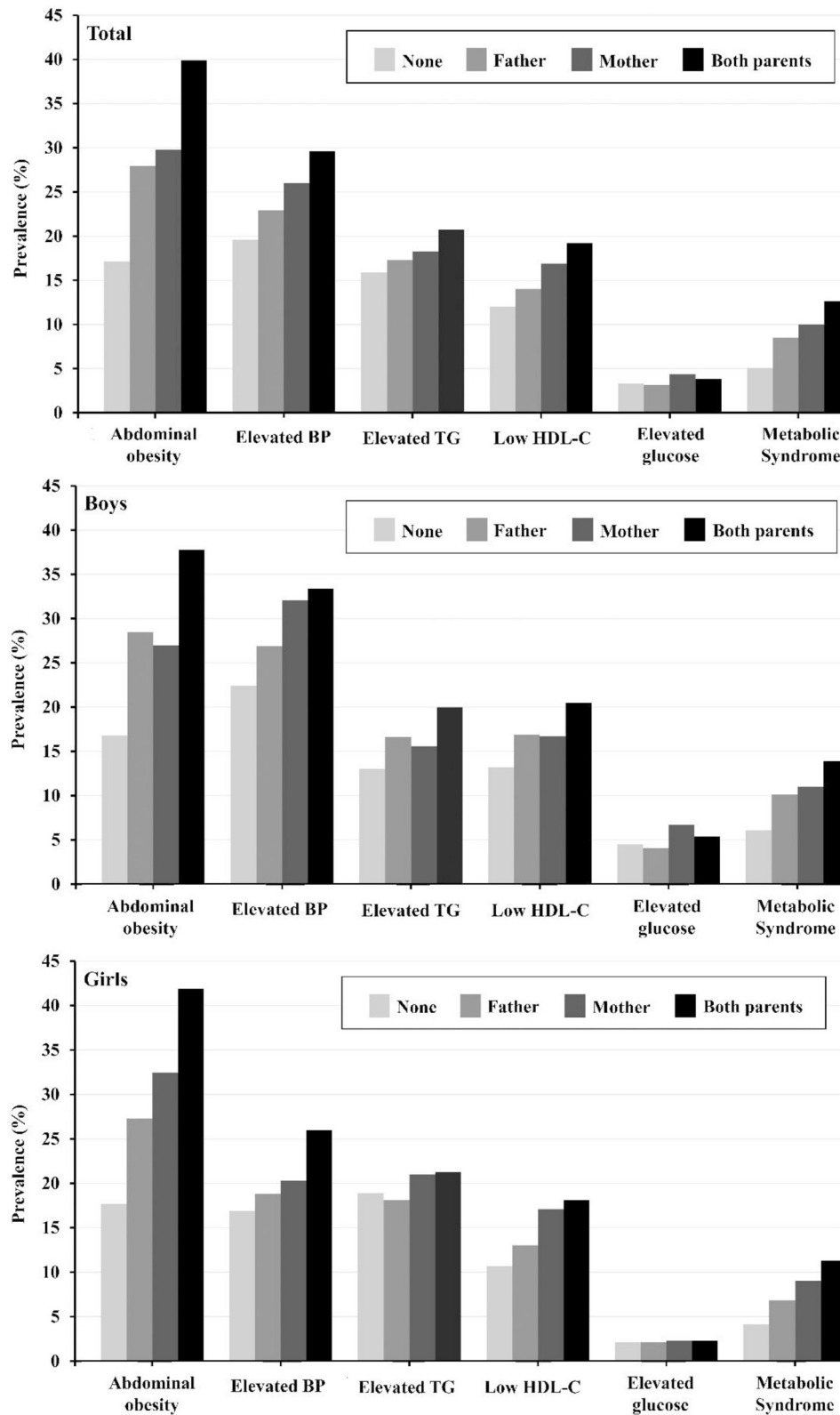


Figure 1 Prevalence of metabolic syndrome and its components in offspring between different parental overweight groups stratified by sex. BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; TG, triglyceride.

that children with overweight parents had a higher risk of abdominal obesity, elevated BP, elevated TG and low HDL-C, and children with both parents classified as overweight had the highest risk of abdominal obesity, elevated BP, elevated TG and low HDL-C compared with those

with one or less overweight parent, even though the CIs overlapped for some of the components. Furthermore, we also found a relatively higher risk of MS, elevated TG and elevated glucose in children with overweight mothers than overweight fathers.

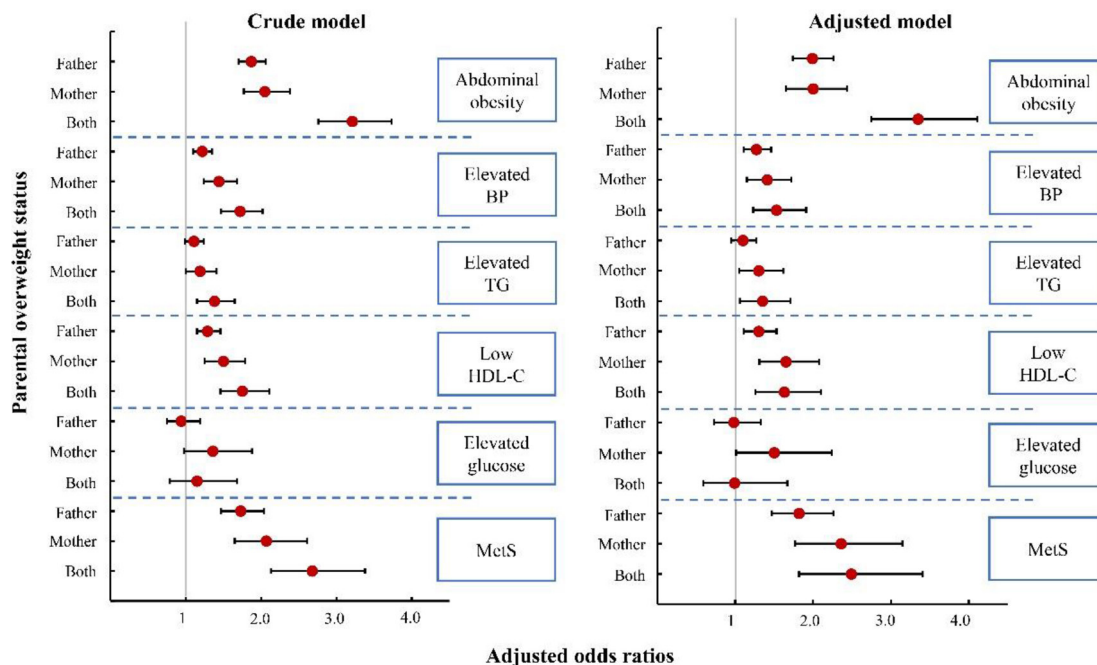


Figure 2 ORs for offspring MetS and its components with parental overweight status. Error bars represent 95% CIs. Crude model was a univariate model, and adjusted model was adjusted for sex, age, residence, household income, family diabetes history and family hypertension history. BP, blood pressure, HDL-C, high-density lipoprotein cholesterol; MetS, metabolic syndrome; TG, triglyceride.

In addition, we also evaluated those associations stratified by sex as is shown in [figure 3](#) and online supplemental table S3. Parental overweight was a potential risk factor of childhood MS both in boys and girls; maternal overweight seems to have a stronger effect on offspring MS compared with paternal overweight, and the results

were similar in boys and girls. When considering the MS components, parental overweight was identified as a risk factor for abdominal obesity, elevated BP, elevated TG, low GDL-C and elevated glucose in boys, and parental overweight was associated with a higher risk of abdominal obesity, elevated BP and low HDL-C.

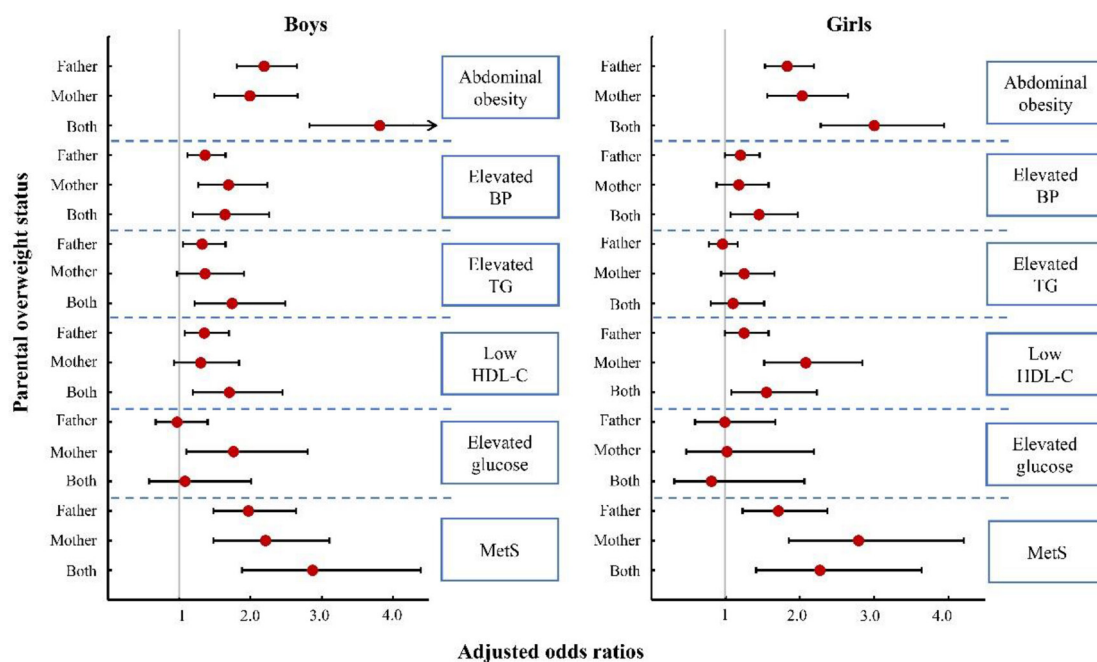


Figure 3 ORs for offspring MetS and its components with parental overweight status between boys and girls. Error bars represent 95% CIs. Crude model was a univariate model, and adjusted model was adjusted for sex, age, residence, household income, family diabetes history and family hypertension history. BP, blood pressure, HDL-C, high-density lipoprotein cholesterol; MetS, metabolic syndrome; TG, triglyceride.

DISCUSSION

In this nationally representative sample of children and their parents, we investigated the correlation of parental weight status and offspring MS and its components among two generations. The result showed that children with overweight parents had a higher prevalence and increased risk of childhood MS and abdominal obesity, elevated BP, elevated TG and low HDL-C; maternal overweight seemed to have a stronger effect on offspring MS risk compared with paternal overweight.

Using the modified ATP III criteria, we found a 7.1% prevalence of MS in Chinese children and adolescents; this prevalence was higher than the result from Chinese Nutritional and Health Surveillance conducted in 2010–2012 (4.3% using ATP III criteria modified by Cook),²⁵ and also higher than investigation conducted by Chinese Work Group of Pediatric Metabolic Syndrome (1.4% using MS-IDF2007 criteria and 2.4% using MS-CHN2012 criteria).²⁶ When compared with reports from other countries, similar prevalence has been observed in Korean adolescents (6.7% in total, 8.5% in boys and 4.5% in girls using modified ATP III criteria),²⁷ the higher prevalence was reported in studies from Italian²⁸ and Mexican²⁹ children, but there were some other studies that reported lower^{30–31} prevalence than that in our study, such as Brazilian (2.6%),³² United Arab Emirates (3.7%)³³ and Spanish (2.5%)³⁴; the reason for the difference may be racial variation and the different diagnostic criteria applied in those studies. The MS diagnosis criteria used in adolescents were different from those in adults, as there were several factors, such as pubertal development, that could influence the anthropometric and blood indices. As such, age-dependent cut-off points are needed. Currently, there was no consensus about the definition of MS in adolescents, nor its clinical value.²⁶ In this study, we choose the modified ATP III criterion because it has been widely used in previously published literature and guidelines for adolescents from China as well as other Asia countries. Among MS components, abdominal obesity was the most prevalent one in all participants, followed by elevated TG and low HDL-C; this was consistent with previous studies.^{35–36}

It has been demonstrated that metabolic disorders tend to cluster within families.^{37–38} In this study we found that parental overweight was a risk factor for developing childhood MS and its components in their offspring, and this relationship remained significant after adjustments for confounding factors that are known to affect MS. To our knowledge, there were few studies investigating the correlation of parental weight status several years after fertility and offspring childhood MS risk, even though parental nutritional status during prepregnancy or gestational period and MS in offspring has been widely investigated.^{17–39} Veena *et al.*⁴⁰ conducted a birth cohort study in India adolescents and found the risk of adiposity and adverse cardiovascular outcomes increased when parents have excessive weight during pregnancy. However, the molecular biological mechanisms underlying those

transgenerational correlations remain poorly understood; evidence to date indicates that multiple mechanisms are in play in the interaction between nutritional imbalance and the transgenerational transmission of obesity and related metabolic phenotypes.^{41–42} One opinion points out that parental obesity is induced by both genetic and acquired environmental factors, which can influence the offspring innate genetic and early life nutritional variation.¹⁶ Ornellas *et al.*¹⁵ found that paternal obesity results in insulin resistance/T2D and increased levels of cortisol in umbilical cord blood, which increases the risk of cardiovascular disease. In our study, we think that parental weight status containing both shared genetic and postnatal environmental factors as BMI was obtained when their offspring was in adolescence. In addition, it also has been suggested that parental lifestyle behaviours can contribute to overweight and obesity of their own as well as metabolic health in their offspring. For example, the practice of a high-fat diet and physical inactivity could induce a higher weight status; those factors could influence the family environment shared by all members and successively influence the metabolic health in their offspring. For MS components, the highest risk in children with overweight parents was for developing abdominal obesity, followed by elevated BP, elevated TG and low HDL-C. On the contrary, no statistically significant association was identified between parental overweight and elevated glucose. We were not surprised by this because abdominal obesity was regarded as the starting point for subsequent metabolic disturbances, as demonstrated by European IDEFICS/I. Family cohort study⁴³ and Framingham Heart Study⁴⁴ while fasting glucose have been proved to be a poor predictor of glucose tolerance.^{45–46}

Our study reported that maternal overweight seems to have a stronger influence on offspring MS and elevated TG and glucose; similar results had been reported in previous studies^{35–47}; Baxi *et al.*³⁵ found that maternal MS status had a significant effect on central adiposity and MS risk in adolescents, whereas such relationships were weaker and not significant for paternal MS status. But there were also different opinions; Veena *et al.*'s study⁴⁰ found that maternal and paternal had equally effects on offspring adiposity and insulin residence.

Adolescence is a crucial period in life as many chronic diseases that occurred in adulthood have their roots in childhood. Our study adds to the current understanding of the effects of parental weight status on offspring metabolic health, which implies that children with overweight or obese parents are the vulnerable population. Future intervention taking both mother and father into account might have better benefits in MS prevention for the younger generation.

The strengths of this study include a large number of parent–offspring trios and allow us to compare correlations between father–offspring and mother–offspring pairs. Besides, in this study, we have evaluated the relationship not only in the ‘intergenerational cycle’ but also after this period. But there are also several limitations.



First, as data were from a cross-sectional survey, it limited us to make casual inferences. Second, information on parental height and weight was recalled from the questionnaire, which was not as accurate as measured directly. Furthermore, we did only collect information about parental current BMI information, but unable to obtain pregnancy BMI and other health conditions, which were regarded as potential factors affecting the health status of their offspring. Future studies based on longitudinal data are needed to further investigate those associations.

CONCLUSION

This study adds to the current body of literature by evaluating the association between parental overweight and offspring MS. The main finding of this study was that parental overweight/obesity was an independent risk factor for childhood MS in their offspring. Maternal overweight seems to have a stronger impact on childhood MS in their offspring. The result of our study support the hypothesis that parental overweight, especially with both parents overweight, can be used to be an indicator in identifying the vulnerable children with increased risk of metabolic dysfunction, and children with obese parents should be targeted to apply some intensive primary prevention.

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Contributors All authors contributed in conceiving and design of this study. ZY and YL performed the data analysis. ZY, YL, DG and BW interpreted, wrote and finalised the manuscript, BD and JM participate in the reviewing and revising the manuscript. All authors read and approved the final manuscript.

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Competing interests None declared.

Patient consent for publication Not required.

Ethics approval This study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving study participants were approved by the Medical Ethical Committee of Peking University Health Science Center (IRB No. 00001052–12072).

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Data availability statement All data relevant to the study are included in the article or uploaded as supplementary information.

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