Impact of parental weight status on children’s body mass index in early life: evidence from a Chinese cohort

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ABSTRACT

Objectives To understand whether parents’ weight status before conception predicts body mass index (BMI) of their offspring in early life and the differences between the mother–child and father–child associations.

Design A birth cohort study.

Setting Conducted at the Community Health Service Centre in Shenyang, Wuhan and Guangzhou.

Participants A total of 2220 live birth newborns were recruited randomly after consent of their parents, and 1178 were followed up until 2 years old.

Methods Parental demographics, maternal characteristics during pregnancy, children’s anthropometric data and feeding patterns at 1 month old were collected. BMI was calculated and BMI Z-scores (BMI_Z) were generated by referring to WHO growth standard. Parental weight status was categorised into underweight, normal weight, and overweight and obese according to the Working Group of Obesity in China. General linear models and generalised linear models were used to assess the associations between parents and offspring.

Outcome measures The primary outcomes were descriptive data on child’s sex-specific anthropometric variables. The secondary outcomes were BMI_Z and weight status of children at each time point.

Results No gender difference was observed in BMI_Z or overweight or obesity rates from birth to 24 months old, although boys were significantly heavier and had a greater length/height than girls (P<0.05). The overweight and obesity rates of children peaked at 12 months old. Maternal BMI/weight status had a significant but small effect on BMI_Z at birth, but not on the paternal side. The impact of parental BMI on child’s BMI_Z after birth was similar at each follow-up. Offspring with overweight/obese mothers tend to have reduced BMI_Z after birth while overweight/obese fathers had children with a greater BMI_Z.

Conclusions Maternal weight status had small effect on both fetal and child growth after birth. Significant but mild paternal influence was only detected after birth.

INTRODUCTION

Childhood obesity is a serious public health concern1 because of its adverse long-term health consequences2,3 and is related directly and indirectly to lifetime economic burden.4 Data from the 2014 WHO show that the overweight or obese rate among Chinese children <5 years old is about 10%,5 which is much higher than that from two decades earlier.6 As it is difficult to lose weight or to sustain successful weight loss,7,8 detecting and addressing the causes of overweight or obesity from the early years of life, especially during the critical stage of adiposity development in the prenatal period9 and early infancy,10 is of great public health importance. Both genetic factors and an obesogenic environment play roles in the development of obesity.11–13 Genetic studies have identified more than 20 genetic variations that are related to the development of childhood obesity.14,15 Meanwhile, environmental obesogenic-related factors such as a high energy diet, short sleep duration and insufficient physical activity, also contribute to childhood obesity.16,17 Parental overweight/obesity is considered as a proxy for the genetic predisposition and/or the obesogenic exposure, and should be considered when tackling the prevalence of childhood obesity. A large number of studies have confirmed a significant correlation between maternal or/and paternal weight status and the growth
measures of their school-age offspring. The parental weight status’ impact on children through similar genetic preposition and/or obesogenic environmental exposure may ‘programme’ the growth and development of children from a very early stage of life. However, the connection between parental weight status and growth of children <2 years old has been rarely discussed. The limited number of studies that have analysed the association between parental body mass index (BMI) and toddler growth, have mostly yielded inconsistent results. For example, Regnault and Knight reported a positive association between maternal BMI and their offspring’s birth weight and/or BMI, whereas Stunkard et al revealed a null association at 2 years old. Of the studies that have examined the impact of paternal BMI on offspring birth weight, only one reported any effect. Fewer studies have compared the effect of maternal and paternal weight status on postpartum growth in offspring, but with discrepant results. So far, conclusions have predominately been drawn from populations of European descent, which may not be applicable to other populations, as the parent–offspring association in BMI could vary given significant differences in social and cultural conditions. Furthermore, there is a lack of longitudinal data to examine the parental–offspring association in BMI and obesity during the early years of life, especially the father–child association in an Asian population.

Based on data from a 2-year birth cohort in three cities in China, we (1) explored the association between parental BMI/weight status and child’s BMI Z-score (BMI_Z) over the first 2 years of life and (2) compared the difference, if any, between the impacts from maternal versus paternal weight status.

**METHODS**

**Study population and design**

A total of 2220 newborns (1155 boys and 1065 girls) were recruited within 1 month after birth from April 2009 to March 2010 at randomly selected Community Health Service Centres (CHSC) in Shenyang, Wuhan and Guangzhou. The participating children were registered and routinely received health checks and follow-ups at the project’s CHSC. Details of participant selection have been published elsewhere. After recruitment, the study subjects were followed at 3, 12 and 24 months (figure 1). The permanent withdrawals included consented withdrawals and those lost to follow-up. Missing data resulted from those who could not attend all follow-ups because of parent unavailability for the regular healthcare visit, child’s sickness, inclement weather or other unknown reasons. No significant differences were detected in the socioeconomic variables, children’s birth variables or parental BMI and weight status between the initially recruited subjects and those available at each follow-up.

All protocol-required procedures were standardised across the three field sites and data collectors were trained and monitored centrally. Informed consent was obtained from the parents.

**Data collection**

**Characteristics of children and parents**

Socioeconomic and health-related variables were obtained at recruitment from the Perinatal Health Booklet (PHB) held by parents and from standardised interviews at 1-month-old and 3-month-old follow-ups. Data extracted from the PHB were gestational weight gain (GWG), health condition during pregnancy (eg, gestational hypertension and diabetes), gestational age at birth, delivery mode and the child’s sex and date of birth. Children’s birth weight and crown-heel length, as measured by nurses at birth, were also extracted from the PHB. Data collected through interviews included maternal lifestyle during pregnancy (such as maternal gestational smoking, secondhand smoking and alcohol consumption), parental age and educational level at childbirth, number of children in the household and infant feeding practices within the first month.

**Anthropic measurements**

Children’s weight and length/height were measured at 3 and 12 months old within 10 days of the exact month, and within 15 days of 24 months old, by the centrally trained child healthcare staff from the project CHSC. Body weight was measured to the nearest 50g with an electronic scale (WHS-I, Wuhan Computer Software Development), with children wearing light indoor clothes. Recumbent length was measured using an infant length measurement instrument (WHS-I) before 24 months old, and a measuring tape (S-RTY1 Healthy kid Intelligent Health Check
Wuhan Computer Software Development) attached to a wall was used for height at 24 months old, both to the nearest 0.1 cm. Both weight and length/height were taken in duplicate, and the means of the replicates were used in the analyses. Parental weights before pregnancy were self-reported, while heights were measured using a stadiometer (Leicester height measure; Invicta Plastic, Leicester, UK) during the first-month-old follow-up home visit.

**Variables**

**Outcome variables**

Children’s BMI at birth and at 3, 12 and 24 months old as well as BMI_Z were employed as the primary outcome variables. BMI was calculated as weight (kilogram) divided height (metre) squared and the age-specific and gender-specific Z-score (BMI_Z) was generated according to WHO Child Growth Standards 2006, using the SAS macro provided to assess child growth and development. Overweight and obesity for children were the secondary outcome and were defined as BMI ≥25th and 95th percentiles, respectively, according to WHO gender-specific and age-specific BMI reference values for children.

**Independent variables**

Parental BMI was set as a continuous independent variable and further categorised into weight status groups, based on BMI cut-offs for Chinese adults recommended by the Working Group of Obesity in China: BMI <18.5 kg/m² as underweight, BMI ≥24 kg/m² as overweight/obese and BMI of 18.5–24 kg/m² as normal weight.

**Potential confounders**

The originally included potential confounders were selected based on previous reports. Low birth weight and macrosomia were defined as birth weight <2500g or ≥4000g, respectively. GWG was set as a continuous variable. Delivery mode was categorised as vaginal birth or caesarean section (C-section) and preterm birth referred to gestational age at birth <37 weeks. Parental education level was classified as middle school or under, high school/technical, university/college and master’s degree or advanced. The feeding pattern at 1 month of age was categorised as exclusive breast feeding, mixed feeding and formula feeding. Exclusive breast feeding was defined according to WHO definition. Mixed-fed children were fed with both breast milk and formula milk, or other food. Formula-fed children were those fed exclusively with formula milk or other food but no breast milk. Variables such as single-child family, gestational hypertension, gestational diabetes, smoking, secondhand smoking and alcohol consumption during pregnancy were set as dichotomous variables (yes/no). The act of smoking a cigarette or cigar at least once per day on average was defined as smoking, and secondhand smoking was defined as being exposed to cigarette smoke accumulating for at least 15 min for at least 1 day per week by non-smoking pregnant women, alcohol consumption as consumption at least one alcoholic drink per day.

**Statistical analysis**

Continuous variables are presented as mean and SD except for BMI with median and IQR, which was in a skewed distribution, and categorical variables are presented as percentages or rates. A tabulated procedure was used to capture the descriptive characteristics of children and parents at recruitment and at the endpoint. Student’s t-test was used to compare differences in child weight, length/height, BMI and BMI_Z between boys and girls at birth and at 3, 12 and 24 months old; and sex-specific overweight and obesity rates of children at each age were generated using the χ² test.

General linear models were adopted to model child BMI_Z at the four follow-ups as a function of parental BMI and covariates. Covariates were selected according to previous reports and a stepwise regression analysis. The covariates of child’s sex, gestational age at birth, maternal age and GWG were included in the model for BMI_Z at birth. Delivery mode was also adjusted for in the BMI_Z models at 3, 12 and 24 months old. In addition, to examine the mediating effects of birth size on the association between parental weight status and children’s BMI_Z.

The database was set up in Epidata 3.1 with all analyses done with SAS V.9.4 software. The level of statistical significance was set at P<0.05 for all hypothesis testing.

**RESULTS**

A total of 2220 participants were recruited at birth, and 1800, 1677 and 1178 participants were successfully followed up until 3, 12 and 24 months old, respectively. At recruitment, 52% of the neonates were boys and more than 87% were the only child in the household. Low birthweight children accounted for 1.7%, whereas 6.8% of children were born with macrosomia. C-sections made up 68% of deliveries in our population. Maternal secondhand smoking during pregnancy was found in 19.9% of cases, compared with 0.7% firsthand smoking, and 1.6% of the study mothers consumed alcohol during pregnancy. More than 50% of parents had received a bachelor degree. More details about the characteristic of study subjects at recruitment and at 24 months old are presented in table 1. No significant differences were found in the parents’ or children’s characteristics between the baseline and the final stage.

Table 2 lists the crude sex-specific and age-specific weight, length/height, BMI, BMI_Z, overweight and obesity rates of children at the four age points. Child’s BMI, BMI_Z, overweight and obesity rate increased during the first year but declined from 12 to 24 months.
| Variables                                    | At recruitment (N=2220) |          | At endpoint (N=1178) |          |
|----------------------------------------------|-------------------------|----------|----------------------|----------|
|                                              | n (%)  | Mean (SD) | n (%)  | Mean (SD) |          |
| Boys                                         | 1155 (52.0) |          | 611 (51.9) |          |          |
| Single-child family                          | 1937 (87.3) |          | 1032 (87.6) |          |          |
| Birth weight (g)                             |          | 3334 (419) |          | 3325 (424) |          |
| Low birth weight                             | 34 (1.5) |          | 20 (1.7)   |          |          |
| Normal birth weight                          | 2028 (91.4) |          | 1078 (91.5) |          |          |
| Macrosomia                                   | 158 (7.1) |          | 80 (6.8)   |          |          |
| Maternal age at childbirth (years)           | 2217    | 28.4 (3.8) | 1176    | 28.6 (3.8) |          |
| Paternal age at childbirth (years)           | 2211    | 31.1 (4.7) | 1172    | 31.3 (4.8) |          |
| Maternal prepregnancy BMI (kg/m²)            | 2215    | 20.4 (2.6)* | 1174 | 20.4 (2.6) |          |
| Maternal underweight                         | 541 (24.4) |          | 487 (22.8) |          |          |
| Maternal normal weight                       | 194 (66.9) |          | 1463 (68.5) |          |          |
| Maternal overweight/obesity                  | 194 (8.8) |          | 187 (8.8) |          |          |
| Paternal BMI at recruitment (kg/m²)          | 2201    | 23.5 (3.3)* | 1169 | 23.6 (3.4) |          |
| Paternal underweight                         | 87 (4.0) |          | 81 (3.8) |          |          |
| Paternal normal weight                       | 1232 (55.9) |          | 1169 (55.0) |          |          |
| Paternal overweight/obesity                  | 884 (40.1) |          | 877 (41.2) |          |          |
| Gestational weight gain                      | 2205    | 17.1 (5.4) | 1168    | 17.2 (5.6) |          |
| Gestational age at birth                     | 2206    | 39.5 (1.5) | 1162    | 39.5 (1.5) |          |
| Preterm                                      | 80 (3.6) |          | 47 (4.0) |          |          |
| Full term                                    | 2126 (96.4) |          | 1115 (96.0) |          |          |
| Delivery mode                                |          |          |          |          |          |
| Vaginal                                      | 699 (31.5) |          | 376 (31.9) |          |          |
| C-section                                    | 1521 (68.5) |          | 802 (68.1) |          |          |
| Maternal pregnancy hypertension              | 52 (2.6) |          | 30 (2.7) |          |          |
| Pregnancy smoking                            | 15 (0.7) |          | 8 (0.8) |          |          |
| Pregnancy secondhand smoking                | 435 (19.9) |          | 240 (20.7) |          |          |
| Pregnancy alcohol consumption               | 35 (1.6) |          | 20 (1.7) |          |          |
| Maternal educational level                   |          |          |          |          |          |
| Middle school or less                        | 432 (19.5) |          | 227 (19.3) |          |          |
| High school/technical                        | 584 (26.3) |          | 321 (27.3) |          |          |
| University/college                           | 1117 (50.3) |          | 582 (49.4) |          |          |
| Master degrees or advanced                   | 87 (3.9) |          | 48 (4.1) |          |          |
| Paternal educational level                   |          |          |          |          |          |
| Middle school or less                        | 343 (15.5) |          | 180 (15.3) |          |          |
| High school/technical                        | 644 (29.0) |          | 329 (27.9) |          |          |
| University/college                           | 1105 (49.8) |          | 595 (50.5) |          |          |
| Master degrees or advanced                   | 128 (5.8) |          | 74 (6.3) |          |          |
| Feeding pattern at the first month           |          |          |          |          |          |
| Exclusive breast feeding                     | 759 (36.7) |          | 418 (37.1) |          |          |
| Mixed feeding                                | 1015 (49.1) |          | 532 (48.2) |          |          |
| Formula feeding                              | 295 (14.3) |          | 153 (13.9) |          |          |

*BMI is presented as median (IQR).
BMI, body mass index; C-section, caesarean section.
Significant sex differences in the growth measures (weight, length/height and BMI) and insignificant sex difference in BMI_Z and the rate of overweight and obesity were detected at all follow-ups.

Results from the general linear models show that maternal BMI had a significant effect on the child’s BMI_Z at birth and later (3, 12 and 24 months old), and the impact of maternal BMI on child’s BMI_Z at birth was significantly larger than that on the after-birth measures (0.07 vs 0.02–0.04, P<0.05); No meaningful impact from the father’s side was detected. The generalised linear models showed that children from underweight mothers had significantly lower BMI_Z from birth to 24 months old compared with those with normal weight mothers. Similarly, overweight/obese mothers tended to have heavier children, which was only significant at birth (β=0.30, P<0.05). Consistent with the general linear models, paternal overweight/obesity had a significant effect on the children’s BMI_Z after birth but not for BMI_Z at birth (at birth: β=0.04, P>0.05; after birth: β=0.04–0.20, P<0.05). Both parents’ BMI and weight status had almost unchanged coefficients in the unadjusted and adjusted models with child’s sex, gestational age at birth, maternal age and GWG included being the major confounders (Table 3).

### DISCUSSION
In the present study, we examined the impacts of maternal and paternal weight status on offspring growth during their early ages. Our results show that maternal weight status was mildly but significantly associated with fetal growth, and this association persisted until after the child’s birth. In contrast to normal weight mothers, significant sex differences in the growth measures (weight, length/height and BMI) and insignificant sex difference in BMI_Z and the rate of overweight and obesity were detected at all follow-ups.

| Age (month) | Maternal BMI | Paternal BMI | Underweight | Overweight/obesity | Underweight | Overweight/obesity |
|-------------|--------------|--------------|-------------|--------------------|-------------|--------------------|
| 0           | 0.07*        | 0.01         | –0.22*      | 0.30*              | –0.12       | 0.04               |
| 3 Unadjusted| 0.03*        | 0.03*        | –0.17*      | 0.11               | –0.31*      | 0.11*              |
|             | Adjusted     |              | –0.14*      | 0.05               | –0.29*      | 0.10*              |
| 12 Unadjusted| 0.03*       | 0.03*        | –0.13*      | 0.12               | –0.18       | 0.16*              |
|             | Adjusted     |              | –0.11*      | 0.10               | –0.17       | 0.15*              |
| 24 Unadjusted| 0.04*       | 0.03*        | –0.16*      | 0.12               | 0.03        | 0.20*              |
|             | Adjusted     |              | –0.15*      | 0.11               | 0.20*       | 0.04               |

Variables controlled for were the same in the general linear models and generalised linear models; in the birth models, child’s sex, gestational age, maternal age, educational level and gestational weight gain were controlled for; in the unadjusted models for BMI_Z at aged 3–24 months old, delivery mode was also added to the factors controlled for in the birth models; in the adjusted models, BMI_Z at birth was also controlled for apart from the factors controlled for in the unadjusted models.

*Refers to significant effect, P<0.05; variables controlled for were the same in the general linear models and generalised linear models.

BMI, body mass index; BMI_Z, BMI Z-score.
underweight mothers had children with significantly lower BMI_Z from birth to 24 months old, whereas overweight/obesity mothers were more likely to have children with significantly higher BMI_Z at birth. The impacts from the father’s side were found to be mild and only significant after children were born.

Previous longitudinal studies reported inconsistent results regarding parental–offspring BMI associations. Both Adegbeye et al.⁴⁰ and Linabery et al.¹⁷ reported a significantly greater association between maternal weight and offspring BMI than for the paternal side. Li et al. indicated that obesity-related genes inherited from parents may play a role during the fetal period,⁴¹ and results from a large Norwegian population-based study showed a similar parental–offspring BMI association when children were 3 years old.⁴² In our study, we found an association between maternal BMI and offspring’s BMI_Z from birth to 24 months old, while the significant association between paternal BMI and offspring’s BMI_Z only became apparent after birth. Our results seemed to suggest that the genetic influence from the father side, if any, might be overshadowed by the impact of maternal weight status through an intrauterine nutritional environment and/or a genetic influence from the mother’s side.

The association between parental weight status and child’s BMI_Z has been reported previously.¹¹ In our study, when parental prepregnancy BMI value was further stratified into three weight status groups (underweight, normal weight and overweight/obesity), maternal underweight was significantly associated with a reduced child’s BMI_Z after birth, while the expected positive associations between parental overweight/obese and the outcome measure only existed in obese paternal group. The arguable results of the non-significant associations between maternal overweight/obesity and children’s BMI_Z after birth could be attributed to several factors. First, it may be due to the small number of overweight/obese mothers (8.7%) and relatively large number of underweight mothers (24.4%), which may reduce the power to detect the mother–offspring association. Second, it could be attributed to the termination of the mother’s role as the direct and only nutrition provider, the involvement of more environmental factors (e.g., feeding practices) and the probability of interactions between environmental factors and genetic predispositions if stimulated.⁴³⁻⁴⁵

Interestingly, in our study, only about 10% of children had a BMI >85th percentile at birth and this figure increased rapidly and reached nearly 30% at both 12 and 24 months old, indicating that at least some of the children may have experienced rapid growth during infancy. According to a study by Wells et al. on a group of Brazilian boys, early rapid weight gain remained the dominant risk factor for later obesity, which may weaken the effect from the maternal side.⁴⁶

According to the National Report on Nutrition and Chronic Diseases in Chinese Residents in 2015, the prevalence of overweight and obesity rate in Chinese adults were up to 30.1% and 11.9% in 2012.⁴⁷ The relatively lower overweight/obesity rate in the current maternal population (7.39% and 1.35%, respectively, with the mean prepregnancy BMI being 20.4 kg/m²) and high underweight rate (24.4%) compared with the national figure might be attributable to different characteristics such as population age (about 28 years old) and the study area (mostly central and southern China). The large proportion of thin women made the current cohort distinguishable from previous studies mainly performed in high-income counties where the average BMI was heavily shifted to the right.⁴⁸ This allowed the detection of the association between maternal undernourishment and the reduced toddler BMI. Thinness (wasting) in early childhood has been significantly related to reduced cognitive development, and increased risk for impaired immune system, poor school performance and a higher risk for mortality.⁴⁹⁻⁵¹ As a country that has been experiencing nutrition transition, China is now facing a double burden of nutrition problems. While a great deal of attention has been attached to addressing the emerging obesity issue, undernourishment, due to an unfavourable nutrition status from the early years of life or as a result of a mindset of ‘thinner is prettier’, appears to be neglected in China especially in the affluent urban area. More attention should be redirected to the group of thin women who are at their childbearing age given the health implication of maternal nutrition in both mother and child.

The current study had some strengths. First, this was a longitudinal study with a relatively large sample size. Previous studies were mostly cross-sectional in design,⁴²⁻⁴³ whereas others were longitudinal studies but with a smaller sample size.⁴⁶ Second, we confirmed the significant association between parental BMI and child’s BMI_Z at first, then classified the parental BMI as underweight or overweight/obesity to better introduce the clinical implications of our findings. This made our results more convenient for identifying high-risk individuals based on their parents’ weight status, either overweight/obesity or underweight. Furthermore, this is among the first studies to compare the parental influence on the BMI_Z of children at an early age in an Asian population.

The use of parental BMI based on their self-reported weight may be one of our weaknesses, as it may be less precise compared with anthropometric data collected under a more carefully controlled research setting. Low maternal overweight/obesity rate and high paternal overweight/obesity rate in our study may limit the generalisability of our finding. Parental overweight and obesity is considered to be due to both genetic predisposition and obesogenic environmental exposure. Using parental weight status, for example, overweight/obesity or underweight as exposure, could not distinguish the impacts of genetic predisposition from that of environmental exposure or the interaction of the two. More genetic studies are needed to explore the genetic impact on the growth of children from an early stage of life.

Research on body composition in infancy suggests that most of the variance in BMI is explained by variance in
lean mass. In our study, BMI was used as the proxy of obesity in children, which is correlated with both lean and fat mass. There was no measurement of body composition of infants available. Because of the comparatively short follow-up durations, we could not verify the association between birth weight, rapid weight gain with body composition of children later in life; and we could not detect whether this presents any inherent similarity in term of maternal and paternal impacts on offspring’s body composition. More studies should include precisely measured body composition with longer observation duration to clarify the clinical implications of BMI and the variation in the development of body composition in early childhood. Last, the comparatively high lost to follow-up rate may limit the generalisability of our findings and reduce the power to detect associations. However, no significant differences were detected in the sociodemographic or birth variables, parental BMI or weight status between the initially recruited and the remaining participants indicating the randomness of those lost to follow-ups. We believe the chance of random lost to follow-up errors affecting our conclusions was slim. Furthermore, the consistency between our findings and those of other related studies supports the generalisability of the findings.

CONCLUSIONS

Our results suggest that the parents’ BMI and weight status affected the growth of their offspring in different ways from the intrauterine period through the first 24 months of life. Maternal BMI and weight status appeared to influence intrauterine growth, and this mild effect persisted during the child’s first 24 months of life. Paternal weight status had less of a significant impact on fetal growth, but showed a small but significant and continuous impact on child’s postpartum growth, although the precise mechanism remains unknown. Nevertheless, our results stress that maintaining a healthy preganancy weight for both mother and father is critical for a healthy prenatal and postnatal growth of children. Further studies are needed to more precisely evaluate the genetic and environmental impacts and their interactions on fetal and child growth, and to clarify the underlying mechanisms of the different maternal–offspring and paternal–offspring associations in BMI.

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Contributors MH collected the data, performed the statistical analyses and the interpretation of data, and drafted, revised and finalised the article. SG contributed to data collecting, writing the background, results and discussions sections of the article. HL, YP, WM and BZ helped finalise the drafted and revised manuscripts. JZ was responsible for the design and implementation of the study, and supervised the data collection, data analysis and manuscript writing.

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