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Pre, Perinatal, and Parental Predictors of Body Mass Index Trajectory Milestones

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Data statement

Data will be made available on request

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Abstract

Objective—To assess associations of pre-, perinatal and parental factors with age and magnitude at body mass index (BMI) peak and rebound.

Study design—Among 1681 children with BMI data from birth to mid-childhood in Project Viva, we fitted individual BMI trajectories using mixed-effect models with natural cubic spline functions, and estimated age and magnitude at peak in infancy and rebound in early childhood. We used stepwise multivariable regression to identify predictors of peak and rebound in the 1354 (63.6%) children with estimable trajectory milestones.

Results—The mean (standard deviation, SD) of age at BMI peak was 8.4 (2.7) months and at rebound was 59.8 (19.6) months, and the mean (SD) of magnitude at peak was 18.0 (1.4) kg/m² and at rebound was 15.9 (1.2) kg/m². Girls had a later age at peak, earlier age at rebound, and lower magnitudes at peak and rebound than boys. Maternal isolated hyperglycemia [vs normoglycemia: β 0.7 months (95% CI 0.2, 1.2)] and preeclampsia [vs normal blood pressure: 1.6 months (0.8, 2.4)] were associated with a later peak, and impaired glucose tolerance [vs normoglycemia: -0.5 kg/m² (-0.9 , -0.1)] was associated with a lower magnitude at peak. Higher maternal first-trimester weight gain, smoking during pregnancy, no breastfeeding, parental obesity and no university education were associated with higher BMI at rebound.

Conclusion—We have identified modifiable prenatal and parental predictors of BMI peak in infancy and rebound in childhood. Early-life interventions that address these factors may be effective in changing BMI peak and rebound, and potentially preventing later obesity.

Body mass index (BMI) typically increases to a maximum during the first year of life (BMI peak), followed by a decline to a nadir (BMI rebound) between 4 and 6 years of age.⁵ A later age and higher magnitude at peak predict adiposity and cardio-metabolic outcomes later in childhood.^{6, 7} Early age at BMI rebound (<4 years) has also been linked to greater adiposity⁸, risk of obesity⁹ and metabolic dysfunction¹⁰ in adolescence. An understanding of the determinants of BMI peak and rebound is therefore important in developing strategies to prevent obesity and its associated cardiometabolic disease risk.

Genetic determinants of adult BMI also have observable effects on age and magnitude at BMI peak and rebound.¹¹ Studies by Roy et al and Wen et al have characterized ethnic differences in BMI peak and rebound, respectively.^{12, 5} The evidence surrounding the role of infant nutrition, such as breastfeeding, on BMI peak and rebound remains inconclusive.^{6, 13–16} Few reports have described prenatal determinants of BMI peak and rebound, other than maternal obesity and gestational weight gain^{6, 17–19}. Although earlier studies have reported associations of gestational exposures such as glucose intolerance²⁰, hypertensive disorders²¹ and smoking²² with childhood BMI, these factors have yet to be related to characteristics of BMI peak and rebound. Furthermore, evidence remains scant regarding the relationship of paternal factors (eg, obesity and socio-economic status) with BMI peak and rebound.

We used data from Project Viva, a longitudinal Boston-area pre-birth cohort, to assess associations of maternal, paternal and child factors with characteristics of BMI peak and rebound. We hypothesized that known risk factors of childhood adiposity, such as obesity and socio-economic status of both parents and maternal smoking, higher gestational weight gain, glucose intolerance and hypertensive disorders,^{17, 18, 20–22} would predict age at and magnitude of BMI peak and rebound.

Methods

Children were participants in Project Viva, an ongoing prospective cohort study of pre- and perinatal influences on maternal, fetal and child health. We recruited eligible pregnant women at clinical visits during the first trimester of pregnancy between 1999 and 2002, from eight obstetric offices of Atrius Harvard Vanguard Medical Associations, a multi-site group practice in Eastern Massachusetts, as detailed elsewhere.²³ Of 2128 live singleton births, we modeled BMI trajectories in 1681 (78.9%) children who had ≥ 3 BMI measurements from birth to mid-childhood (median 92.5; range 78.8–131.2 months) (Figure 1; available at www.jpeds.com), for a total of 20,598 BMI measurements (Table I; available at www.jpeds.com).

Mothers provided written informed consent at enrollment and each postnatal follow-up visit at infancy, early- and mid-childhood. The Institutional Review Board of Harvard Pilgrim Health Care approved the project in line with ethical standards established by Declaration of Helsinki.

Parental obesity

Mothers reported their pre-pregnancy weight, height, and father's weight and height via questionnaires and interviews at recruitment. We calculated pre-pregnancy and paternal BMI (in kg/m²) as self-reported pre-pregnancy or paternal weight divided by height squared respectively, and categorized obesity as follows: both non-obese (pre-pregnancy and paternal BMI <30kg/m²), only mother obese (pre-pregnancy BMI ≥ 30kg/m²), only fathers' obese (paternal BMI ≥ 30kg/m²) or both obese (pre-pregnancy and paternal BMI ≥ 30kg/m²).

Parental socio-economic status

Mothers reported their and fathers' highest education via questionnaires and interviews at recruitment. We categorized parental educational level as neither university-educated or mother, father or both university-educated. We also calculated median annual household income for census tract of residence at delivery using data from the 2000 United States Census.²⁴

Other maternal factors

Mothers reported their smoking history via questionnaires at recruitment. As detailed previously,²⁵ we used measured weights recorded in the outpatient medical records and self-reported pre-pregnancy weight to calculate trimester-specific gestational weight gain (GWG). We obtained results of a 2-stage clinical glycemic screening and used them to categorize women as having normoglycemia, isolated hyperglycemia, impaired glucose

tolerance (IGT) or gestational diabetes (GDM), based on criteria previously detailed.²⁰ We also extracted data on parity and hypertensive disorders of pregnancy (normal blood pressure, gestational hypertension, chronic hypertension or pre-eclampsia) from hospital medical records.

Child factors

We extracted data on infant sex and birth weight from hospital medical records. We calculated length of gestation in days by subtracting the date of the LMP from the date of delivery. If gestational age according to the second-trimester ultrasound differed from that according to the LMP by >10 days, we used the ultrasound result to determine gestational duration. We calculated birth weight-for-gestational-age and sex z-scores from a US national reference.²⁶ The mother reported her child's race/ethnicity, which we categorized as white, black, Hispanic, Asian, or others. We obtained information on initiation of breastfeeding at post-delivery interviews, as detailed previously.²⁷

During research examinations at birth, in infancy (median 6.3 months; range 4.9–10.6 months), early childhood (37.9; 33.6–72.5 months) and mid-childhood (92.5; 78.8–131.2 months)²³, trained research assistants measured weight and length/height using standardized protocols.^{20, 28, 29} We also obtained additional data on weight and length/height from medical records, where pediatric clinics recorded length/height and weight data at each well-child visit during infancy and childhood. As described previously, clinicians used the paper-and-pencil technique for measuring recumbent length for infants 0–2 years at pediatric clinics.³⁰ We applied a correction algorithm to account for the systematic overestimation of clinical lengths resulting from this technique.³⁰ Using both research and clinical measures, we calculated BMI as weight in kilograms divided by length or height in meters squared.

Statistical analyses

To estimate BMI peak and rebound, we used statistical models, rather than direct observations and visual inspection of individual BMI-for-age curves,³¹ which is prone to large inter-observer variation.³² Furthermore, not every child had multiple measures of weight and stature at enough time points to allow us to directly observe the peak and rebound characteristics; applying the models allowed us to estimate these characteristics for a larger number of children in the cohort. We fitted individual BMI curves using mixed-effects models with natural cubic spline functions for age to capture the non-linear trend in BMI. Random effects were included in the model to account for repeated measures in the same child and to capture the non-linear trend in BMI. We applied constraints to increase stability of the curve³³ by fixing the spline to be linear prior to 0.1 months and after 131.1 months. The fixed effects component of the model was as follows:

$$BMI = \beta_{00} + \beta_{10}(\text{age}) + \sum_{j=1}^m \beta_j \{ (\text{age} - k_j)^3_+ - \lambda_j (\text{age} - k_{\min})^3_+ - (1 - \lambda_j) (\text{age} - k_{\max})^3_+ \} + e_{ij}$$

where $k_{\min}$ and $k_{\max}$ = boundary knots, k_j = interior knot point j between boundary knots; m = number of interior knots between boundary knots; $j = 1, 2, \dots, m$; e = residual and (age-

$k_j)^3_+$ is defined as age- k if age $\geq k_j$. The random effects component of the model contained random effects for the intercept, linear age slopes and spline functions.

We considered two approaches to select knot locations: at equally spaced percentiles³⁴ between birth and 131.2 months or at the median, minimum and maximum ages of each of three developmental periods: infancy and early and mid-childhood. We determined the optimum number and location of knots using the Bayesian information criterion. The optimal number was six knots for both fixed and random effects, located at 0.1, 4.9, 10.6, 37.9, 92.5, and 131.1 months of age (Table 1). We assessed model fit using a residual plot of observed and predicted BMI. As the initial residual plot (Figure 2, A; available at www.jpeds.com) revealed heteroscedasticity in residual errors over age, we accounted for it by estimating residual error parameters over categorical distributions of age (Figure 2, B; available at www.jpeds.com).

We derived sex-specific trajectories by including interactions of child sex with spline terms as fixed parameters into the model and by modelling BMI trajectories separately for boys and girls. As the trajectories derived were similar for both approaches, we present those based on the first approach (both sexes together with interaction terms). We estimated the age (in months) at peak and rebound by differentiation of the subject-specific BMI curve; the peak and rebound are located at ages where the derivative of the curve equals zero. We estimated the magnitude (kg/m^2) at peak and rebound as the highest and lowest points, respectively, of the child-specific BMI curve. This approach has been utilized in other studies.⁶

Among the 1681 children with ≥ 3 BMI measures, 1608 (75.6%) had estimable BMI peak and rebound, 42 had no BMI peak (i.e., showed no decline in BMI after the rise in infancy) and 30 had no BMI rebound (i.e., showed no rise in BMI after the decline in early childhood) (Figure 1). Figure 3 (available at www.jpeds.com) illustrates the observed and fitted BMI data of children with estimable BMI peak and rebound ($n=6$), no BMI peak ($n=2$) and no BMI rebound ($n=2$), who were randomly selected from each group using a random number generator.

We examined correlations between pairs of BMI trajectory milestones (age and magnitude at peak and rebound) using Pearson's correlations. We first screened for the following potential predictors: maternal age, median annual income for census tract of residence at delivery, parity (nulliparous, multiparous), smoking history (never smoked, smoked prior to pregnancy or smoked during pregnancy), height, trimester-specific GWG, glucose tolerance (normoglycemia, isolated hyperglycemia, IGT or GDM), and hypertensive disorders of pregnancy (normal blood pressure, gestational hypertension, chronic hypertension or pre-eclampsia); obesity and education level for both parents; and child's gestational age at delivery (<37 or ≥ 37 weeks), sex (male or female), race/ethnicity (white, black, Hispanic, Asian or others), BW-for-GA z-score, and breastfeeding initiation (no or yes) using univariable linear regression analyses. We selected factors based on $P < .25$ for further multivariable analysis; this cutoff value is supported in earlier literature^{35, 36} and allows for clinically relevant factors to be included even if statistically nonsignificant in univariable analyses.³⁶

Subsequently, we built multivariable linear regression models with factors selected in the first step, using a forward stepwise selection approach and $p < 0.25$ as an entry criterion. We forced race/ethnicity into all models to account for known associations of child race/ethnicity with BMI peak and rebound.^{5, 12} To assess the robustness of predictors identified using this approach, we repeated the analyses using backward stepwise selection, and also by simultaneously including all factors identified from the univariable analyses into regression models of age and magnitude at peak or rebound. We created visual representations of the examined predictors of BMI peak and rebound by stratifying the estimated BMI trajectories derived in stage 1 according to the following subgroups: parental obesity and education categories, maternal hypertensive disorders of pregnancy, glucose tolerance status and smoking history. We restricted all analyses to those without missing data and who had estimable peak and rebound ($n=1354$ [63.6%]; Figure 1). Analyses were performed using Stata 15 software (StataCorp LP, Texas, USA).

Results

Table 2 describes the characteristics of children with estimable peak and rebound; the mean (SD) of age at peak was 8.4 (2.7) months and at rebound was 59.8 (19.6) months, and the mean (SD) of magnitude at peak was 18.0 (1.4) kg/m² and at rebound was 15.9 (1.2). Table 3 (available at www.jpeds.com) describes differences in characteristics between children who did and did not have estimable BMI peak and rebound. Children without estimable BMI peak were more likely to be Hispanic, had parents who were obese and less likely to have university-educated parents, than children with estimable BMI peak and rebound. Ages at peak and rebound were not correlated, and magnitude at peak and rebound were strongly correlated ($r=0.72$). Age at peak showed weak correlations with magnitude at peak ($r=-0.17$) and rebound ($r=0.09$), and magnitude at peak was not correlated with age at rebound.

Table 4 (available at www.jpeds.com) describes the crude associations of parental and child characteristics with ages at peak and rebound, and lists the variables that passed the multivariable model selection criteria. Table 5 shows the results of the forward and backward stepwise multivariable analyses. Girls had a later age at peak [by 0.8 months (95% CI 0.5, 1.1)] and earlier age at rebound [−7.8 months (−9.8, −5.8)] than boys, and Asian children had an earlier age at BMI peak than white children. Maternal GDM (vs. normoglycemia) was associated with age at peak in crude analyses, although this was attenuated to non-significance in stepwise analyses. Maternal obesity (vs. neither parent obese; Figure 4, A), pre-eclampsia (vs. normal blood pressure; Figure 4, B) and isolated hyperglycemia (vs. normoglycemia; Figure 4, C) were associated with older age at BMI peak, and gestational weight gain was not associated with age at peak (Table 6; available at www.jpeds.com). Black children had an earlier age at rebound than white children in crude analyses, which was attenuated to non-significance in stepwise analyses. Maternal, paternal or biparental obesity (vs. parental non-obese; Figure 4, A) and non-initiation of breastfeeding [−3.4 months (−6.8, −0.02)] were associated with an earlier age at rebound, although parental socio-economic status, maternal age, smoking history, height, glucose tolerance and child race/ethnicity were not significantly associated with age at rebound (Table 6).

Table 7 (available at www.jpeds.com) describes the crude associations of parental and child characteristics with magnitude at BMI peak and rebound and lists the variables that passed the multivariable model selection criteria. Table 8 describes the results of the forward and backward stepwise multivariable analyses. Girls had a lower BMI at peak [-0.6 kg/m^2 ($-0.7, -0.4$)] and rebound [-0.2 kg/m^2 ($-0.3, -0.1$)] than boys, and black children had higher BMI at peak than white children. A higher BW-for-GA z-score was associated with higher magnitude at peak [0.3 kg/m^2 ($0.2, 0.4$) per 1SD increase] and rebound [0.2 kg/m^2 ($0.2, 0.3$) per 1SD increase]. Biparental obesity (vs. neither parent obese) was associated with higher magnitude at peak (Figure 4, A), and maternal IGT (vs. normoglycemia; Figure 4, C) and biparental university education (vs. neither parent university-educated; Figure 4, D) were associated with a lower magnitude at BMI peak. Maternal age, residence income, smoking history, height, gestational weight gain and hypertensive disorders of pregnancy were not associated with magnitude at peak BMI (Table 9; available at www.jpeds.com). Biparental obesity (Figure 4, A), higher maternal 1st-trimester GWG [0.03 kg/m^2 ($0.01, 0.06$) per kg increase] and maternal smoking during pregnancy (vs. never smoked; Figure 4, E) were associated with a higher magnitude at BMI rebound, and biparental university education (vs. neither parent university-educated) was associated with a lower magnitude at rebound (Figure 4, D). Maternal height, residence-based income, glucose tolerance, hypertensive disorders of pregnancy and child race/ethnicity were not associated with magnitude at BMI rebound (Table 9).

Discussion

In this cohort of 1354 children, known risk factors of childhood BMI, such as parental obesity and socio-economic status, higher maternal gestational weight gain, and smoking during pregnancy were associated with BMI trajectory milestones. We also noted sex and ethnic differences in age and magnitude at peak and rebound. Furthermore, we demonstrated associations of other gestational exposures (glucose intolerance and hypertensive disorders) with BMI peak milestones, which have not been previously reported.

We found maternal smoking during pregnancy and gestational weight gain to be positively associated with magnitude at BMI rebound, similar to previous reports from our own cohort,^{22, 37} as well as other studies^{38, 39} showing positive associations between these risk factors and childhood BMI. Our results also suggest that children of mothers with isolated hyperglycemia and preeclampsia have different ages at BMI peak from those of children of normoglycemic and normotensive mothers. Although children of mothers with IGT had a lower magnitude at peak BMI, they appeared to have higher BMI at mid-childhood than children of normoglycemic mothers, arguing for the importance of examining the entire BMI trajectory for these children. Although we have no clear biological explanation for these observations, genetic, epigenetic and environmental mediators should be explored in future research. There is also evidence to suggest that gestational glucose intolerance and hypertensive disorders are associated with fetoplacental vascular endothelial dysfunction leading to altered postnatal growth and development,⁴⁰ a phenomenon commonly associated with future development of chronic diseases. More importantly, the predictors we have identified are potentially modifiable through behavior-change interventions,^{41–43} suggesting

that they could be targets for changing BMI peak and rebound in children and may contribute to future obesity prevention.

Our results confirm earlier findings^{5, 44} that magnitudes at BMI peak and rebound were strongly correlated with each other, even though the ages at which these milestones occurred differed across studies. These latter differences are partly due to different study populations or statistical modeling methods. We utilized a semi-parametric modeling approach using natural cubic splines to fit subject-specific BMI trajectories, a technique favored for its greater flexibility and improved data fit compared with other methods.^{11, 33} The advantages and disadvantages of other approaches in modeling BMI trajectories have been described previously.¹¹ However, our primary objective was to characterize BMI peak and rebound, and our approach sufficed for obtaining precise milestone estimates, given our model flexibility and good data fit.

Our finding of ethnic differences in BMI peak corroborates earlier studies reporting that African-American infants had higher magnitude at BMI peak¹², and Asian children had earlier age at BMI peak⁶ than white infants. Prior studies have suggested that socio-economic differences may explain different childhood obesity patterns between African-American,⁴⁵ Asian⁴⁶ and white children. In our study, however, the differences persisted even after adjusting for socio-economic status.

In line with previous studies,^{5, 6, 13, 47} we observed that birth weight is directly associated with magnitude at BMI peak and rebound. This association may be explained by tracking of body size from birth to mid-childhood.⁴⁸ Our observation that girls had a later age at BMI peak, earlier age at BMI rebound and lower magnitude at peak and rebound than boys is also consistent with previous studies.^{5, 6, 12} These sex differences may be explained by differences in sex hormones, diet, or physical activity levels. We also observed that non-initiation of breastfeeding was associated with an earlier age at rebound, which could be the result of residual confounding (breastfeeding is strongly socially patterned⁴⁹) or could be caused by prolonged breastfeeding in the group that initiated it.⁵⁰ The evidence surrounding breastfeeding duration and BMI rebound remains equivocal, however.^{5, 14, 15} Further investigation is required to ascertain the potential influence of breastfeeding on timing of adiposity rebound.

Consistent with recent studies,^{17, 19, 51} we found parental obesity to be predictive of earlier age and higher magnitude at BMI rebound. This may be partly explained by inheritance of obesity susceptibility genetic variants. Using genome-wide scans, Felix et al⁵² identified 12 known adult genetic loci that were associated with childhood BMI. The association of parental obesity with BMI rebound may also be driven by unhealthy parental lifestyles that contribute to an obesogenic family environment.⁵³ It is known that a later age and higher magnitude at BMI peak,^{6, 47} and an earlier age at BMI rebound^{9, 10} predict a higher risk of developing obesity and metabolic syndrome later in life. Other studies⁴⁴ have also reported that a later age at rebound was associated with a better cardio-metabolic profile in adulthood (lower BMI, waist circumference and odds of metabolic syndrome). Taken together, our observations provide further support for parental obesity as a useful marker of children who are likely to experience worrisome growth patterns.

In our cohort, parental university education was associated with lower magnitude at BMI peak and rebound, in line with reports of socio-economic disparities in childhood BMI.^{54, 55} Less educated parents may be less aware of healthy weight targets for their children and how those targets can be achieved. Higher parental education level may lower the risk of children attaining a high BMI through better incorporation of health education messages in the parents' daily lives, better access to healthcare services and better communication with healthcare personnel.⁵⁶ Motivational interviewing approaches to educate parents of low socio-economic status about healthy parental feeding practices can favorably influence weight trajectories in their children.⁵⁷ Our study was based on a single assessment of education level at time of recruitment and did not specifically address health literacy or knowledge of obesity risk factors. Furthermore, our findings may not be generalizable to populations of low socio-economic status, as all participants in this study were predominantly well-educated and had health insurance.

One limitation of our study is that we estimated BMI peak and rebound from statistical models, rather than direct observations using visual inspection of individual BMI-for-age curves.³¹ Our models showed mean residual errors (differences between observed and predicted BMI) that were close to zero across all ages, however. Modeling the entire curve from birth is likely to provide more precise estimations of peak and rebound than visual inspection of raw BMI values, which is subject to large inter-observer variation.^{5, 32} Second, our study findings may not be applicable to other racial groups, because a majority of our participants were white. Lastly, our findings are applicable only to children with estimable BMI peak and rebound as we excluded children without estimable BMI peak or rebound from our analyses.

Many of the predictors we found are amenable to behavior-change interventions. Future studies are warranted to identify whether intervening on these factors are effective in changing BMI peak and rebound in children, and if so, whether such intervention can reduce the risk of later obesity and its long-term cardiometabolic consequences.

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Abbreviations

BW-for-GA	birthweight-for-gestational age
BMI	body mass index
CI	confidence interval
IGT	impaired glucose intolerance
GDM	gestational diabetes mellitus

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Figure 1 online.
Flowchart of study sample

Fig2A

Fig2B

Figure 2 online.

Plot of residual BMI values (observed – predicted BMI values) over age (**A**) without accounting for heteroskedasticity in residual errors, and (**B**) accounting for heteroskedasticity in residual errors

Figure 3 online.

Fitted BMI trajectories of 10 children with estimable BMI peak and rebound, without estimable BMI peak (i.e. showed no decline in BMI after the rise in infancy) and without estimable BMI rebound (i.e. showed no rise in BMI after the decline in early -childhood)

Fig_4A

Fig_4B

Fig_4C

Fig_4D

Fig_4E

Figure 4.

Estimated BMI trajectories from birth to mid-childhood according to (A) parental obesity categories, (B) maternal hypertensive disorders of pregnancy, (C) maternal glucose tolerance status, (D) parental education level and (E) maternal smoking history during pregnancy. BMI trajectories were derived using mixed-effects models with natural cubic spline functions for age

Table 1 online

Fit statistics of mixed-effects regression modelling using natural cubic splines, according to number and location of knot positions.

Model	No. of BMI measures	No. of knots	Knot positions (months) defined by percentiles of age distribution	BIC
1	20598	4	0.1, 7.4, 26.5, 87.7	71898.9
2	20598	5	0.1, 4.1, 15.0, 37.3, 87.7	64766.8
3	20598	6	0.1, 4.1, 9.9, 21.1, 43.8, 87.7	64388.27
4	20598	7	0.1, 2.1, 7.0, 15.0, 28.3, 52.5, 95.7	model did not converge
	No. of BMI measures	No. of knots	Knot positions (months) defined by developmental time periods	BIC
5	20598	4	0.1, 7.8, 37.9, 92.5	74458.32
6	20598	5	0.1, 4.9, 10.6, 37.9, 92.5	63890.75
7	20598	6	0.1, 4.9, 10.6, 37.9, 92.5, 131.1	63640.71
8	20598	7	0.1, 4.9, 10.6, 37.9, 75.6, 92.5, 131.1	model did not converge

Table 2

Characteristics of parents and children in Project Viva (n = 1354)

	Mean (SD) or n (%)
<u>Parental</u>	
Maternal age (years)	32.5 (4.7)
Median annual income for census tract of residence at delivery (US dollars)	58,886 (21,163)
Parental education	
Both not university-educated	289 (21.3)
Mother university-educated	170 (12.6)
Father university-educated	114 (8.4)
Both university-educated	781 (57.7)
Nulliparous	
No	721 (53.3)
Yes	633 (46.8)
Maternal smoking history	
Never smoked	936 (69.1)
Former smoker	275 (20.3)
Current smoker	143 (10.6)
Maternal height (m)	1.65 (0.07)
Paternal height (m)	1.79 (0.08)
1st trimester weight gain (kg)	2.9 (2.8)
2nd trimester weight gain (kg)	6.4 (2.4)
3rd trimester weight gain (kg)	6.3 (2.9)
Parental obesity	
Both non-obese	1020 (75.3)
Mother obese	128 (9.5)
Father obese	138 (10.2)
Both obese	68 (5.0)
Maternal glucose tolerance status	
Normoglycemia	1113 (82.2)
Isolated hyperglycemia	123 (9.1)
IGT	43 (3.2)
GDM	75 (5.5)
Hypertensive disorders of pregnancy	
Normal blood pressure	1194 (88.2)
Gestational hypertension	97 (7.2)
Chronic Hypertension	18 (1.3)
Pre-eclampsia	45 (3.3)
<u>Child</u>	
Sex	
Male	705 (52.1)
Female	649 (47.9)

	Mean (SD) or n (%)
Race/ethnicity	
White	930 (68.7)
Black	185 (13.7)
Hispanic	58 (4.3)
Asian	57 (4.2)
Others	124 (9.1)
Gestational age at delivery	
Term	1265 (93.4)
Preterm	89 (6.6)
Birth weight-for-gestational age z-score (SD units)	0.20 (0.96)
Breastfeeding initiation	
Yes	1215 (89.7)
No	139 (10.3)
Age at BMI peak (months)	8.4 (2.7)
Age at BMI rebound (months)	59.8 (19.6)
Magnitude at BMI peak (kg/m²)	18.0 (1.4)
Magnitude at BMI rebound (kg/m²)	15.9 (1.2)

IGT: impaired glucose tolerance; GDM: gestational diabetes mellitus

Table 3 online

Comparison of characteristics between children that did and did not have estimable peak and rebound
(n=1409^a)

	Had estimable BMI peak and rebound (n=1354)	No BMI peak (n=32)	No BMI rebound (n=23)	p value
	Mean (SD) or n (%)			
<i>Parental</i>				
Maternal age (years)	32.5 (4.7)	32.8 (4.4)	32.4 (4.5)	0.92
Median annual income for census tract of residence at delivery (US dollars)	58,886 (21163)	52,995 (15618)	58,922 (22343)	0.29
Parental college education				0.01
Both parents not university-educated	289 (21.3)	9 (28.1)	5 (21.7)	
Only mother university-educated	170 (12.6)	11 (34.4)	3 (13.0)	
Only father university-educated	114 (8.4)	2 (6.2)	2 (8.7)	
Both parents university-educated	781 (57.7)	10 (31.3)	13 (56.5)	
Nulliparous				0.11
No	721 (53.3)	23 (71.9)	12 (52.2)	
Yes	633 (46.8)	9 (28.1)	11 (47.8)	
Maternal smoking history				0.37
Never smoked	936 (69.1)	18 (56.3)	16 (69.6)	
Smoked prior to pregnancy	275 (20.3)	8 (25.0)	6 (26.1)	
Smoked during pregnancy	143 (10.6)	6 (18.7)	1 (4.3)	
Maternal height (m)	1.6 (0.07)	1.7 (0.07)	1.7 (0.07)	0.33
Paternal height (m)	1.8 (0.08)	1.8 (0.08)	1.8 (0.07)	0.13
1 st -trimester weight gain (kg)	2.9 (2.8)	3.3 (3.2)	2.7 (1.8)	0.64
2 nd -trimester weight gain (kg)	6.4 (2.4)	6.9 (2.5)	5.9 (2.7)	0.28
3 rd -trimester weight gain (kg)	6.3 (2.9)	5.3 (4.2)	5.3 (2.5)	0.05
Parental obesity				<0.01
Both parents non-obese	1020 (75.3)	12 (37.5)	18 (78.3)	
Only mother obese	128 (9.5)	7 (21.9)	2 (8.7)	
Only father obese	138 (10.2)	7 (21.9)	1 (4.4)	
Both parents obese	68 (5.0)	6 (18.8)	2 (8.7)	
Maternal glucose tolerance status				0.70
Normoglycemia	1113 (82.2)	25 (78.1)	90 (87.0)	
Isolated hyperglycemia	123 (9.1)	2 (6.3)	1 (4.3)	
IGT ^b	43 (3.2)	2 (6.3)	0 (0.0)	
GDM ^b	75 (5.5)	3 (9.4)	2 (8.7)	
Hypertensive disorders of pregnancy				0.13
Normal blood pressure	1194 (88.2)	28 (87.5)	19 (82.6)	
Gestational hypertension	97 (7.2)	2 (6.3)	1 (4.4)	
Chronic Hypertension	18 (1.3)	2 (6.2)	1 (4.3)	
Pre-eclampsia	45 (3.3)	0 (0.0)	2 (8.7)	

	Had estimable BMI peak and rebound (n=1354)	No BMI peak (n=32)	No BMI rebound (n=23)	p value
<i>Child</i>				
Sex				0.12
Male	705 (52.1)	13 (40.6)	8 (34.8)	
Female	649 (47.9)	19 (59.4)	15 (65.2)	
Race/ethnicity				0.04
White	930 (68.7)	15 (46.9)	17 (73.9)	
Black	185 (13.7)	5 (15.6)	2 (8.7)	
Hispanic	58 (4.3)	4 (12.5)	0 (0.0)	
Asian	57 (4.2)	1 (3.1)	0 (0.0)	
Others	124 (9.1)	7 (21.9)	4 (17.4)	
Gestational age at delivery at delivery				0.67
Term	1265 (93.4)	31 (96.9)	22 (95.7)	
Preterm	89 (6.6)	1 (3.1)	1 (4.3)	
Birthweight-for-gestational age z-score	0.20 (0.96)	0.49 (0.83)	0.25 (0.79)	0.21
Breastfeeding initiation				0.53
Yes	1215 (89.7)	29 (90.6)	19 (82.6)	
No	139 (10.3)	3 (9.4)	4 (17.4)	

^aSample restricted to those without missing parental or child data

^bIGT: impaired glucose tolerance; GDM: gestational diabetes mellitus

Table 4 online
Crude associations of parental and child characteristics with age at BMI peak and rebound (n=1354)

	Age at peak				Age at rebound			
	B (months)	95% CI		p value	B (months)	95% CI		p value
		Low	High			Low	High	
<i>Parental</i>								
Maternal age (years)	0.01	-0.02	0.04	0.53	0.1	-0.1	0.4	0.25
Median annual income for census tract of residence at delivery, \$10,000	0.05	-0.02	0.1	0.16 ^a	0.6	0.1	1.1	0.01 ^a
<i>Parental college education</i>								
Both parents not university-educated	ref	—	—	—	ref	—	—	—
Only mother university-educated	-0.01	-0.5	0.5	0.98	-0.4	-4.1	3.3	0.84
Only father university-educated	-0.1	-0.7	0.5	0.68	1.2	-3.0	5.4	0.57
Both parents university-educated	0.1	-0.3	0.4	0.73	5.9	3.2	8.5	0.00 ^a
<i>Nulliparous</i>								
No	ref	—	—	—	ref	—	—	—
Yes	-0.1	-0.4	0.2	0.58	-0.6	-2.7	1.5	0.59
<i>Maternal smoking history</i>								
Never smoked	ref	—	—	—	ref	—	—	—
Smoked prior to pregnancy	-0.04	-0.4	0.3	0.82	-0.8	-3.5	1.8	0.54
Smoked during pregnancy	0.1	-0.4	0.6	0.63	-3.6	-7.0	-0.1	0.04 ^a
Maternal height (m)	0.2	-1.8	2.2	0.83	4.5	-10.3	19.3	0.55
Paternal height (m)	0.9	-0.9	2.7	0.32	4.5	-8.6	17.7	0.50
1 st -trimester weight gain (kg)	0.0	0.0	0.1	0.17 ^a	-0.2	-0.6	0.2	0.24 ^a
2 nd -trimester weight gain (kg)	-0.1	-0.1	0.004	0.07 ^a	0.3	-0.2	0.7	0.26
3 rd -trimester weight gain (kg)	-0.01	-0.1	0.04	0.76	0.2	-0.2	0.5	0.33
<i>Parental obesity</i>								
Both parents non-obese	ref	—	—	—	ref	—	—	—
Only mother obese	0.8	0.3	1.3	<0.01 ^a	-11.4	-14.9	-7.9	<0.01 ^a
Only father obese	0.2	-0.2	0.7	0.35	-7.1	-10.5	-3.7	<0.01 ^a

	Age at peak			Age at rebound		
	B (months)	95% CI		B (months)	95% CI	
		Low	High		Low	High
Both parents obese	-0.1	-0.8	0.6	0.78	-12.3	-17.0 -7.7
Maternal glucose tolerance status						
Normoglycemia	ref	—	—	—	—	—
Isolated hyperglycemia	0.9	0.4	1.4	<0.01 ^a	-2.5	-6.1 1.2
IGT ^b	-0.4	-1.2	0.4	0.31	-6.2	-12.2 -0.3
GDM ^b	0.8	0.1	1.4	0.02 ^a	-2.2	-6.8 2.4
Hypertensive disorders of pregnancy						
Normal blood pressure	ref	—	—	—	—	—
Gestational hypertension	0.1	-0.5	0.6	0.85	-1.3	-5.4 2.7
Chronic Hypertension	0.4	-0.9	1.6	0.55	-2.3	-11.4 6.9
Pre-eclampsia	1.8	1.1	2.6	<0.01 ^a	-0.7	-6.5 5.1
Child						
Sex						
Male	ref	—	—	—	—	—
Female	0.8	0.5	1.1	<0.01 ^a	-7.4	-9.4 -5.3
Race/ethnicity						
White	ref	—	—	—	—	—
Black	-0.2	-0.7	0.2	0.29	-5.4	-8.5 -2.3
Hispanic	0.2	-0.5	0.9	0.52	-0.2	-5.3 5.0
Asian	-0.9	-1.6	-0.1	0.02 ^a	0.4	-4.8 5.6
Others	0.0	-0.5	0.5	0.99	-3.3	-6.9 0.4
Gestational age at delivery						
Term	ref	—	—	—	—	—
Preterm	0.3	-0.3	0.8	0.37	-1.4	-5.6 2.8
Birthweight-for-gestational age z-score						
0.04	-0.1	0.2	0.59	0.4	-0.7	1.5
Breastfeeding initiation						
Yes	ref	—	—	—	—	—

	Age at rebound				Age at peak			
	95% CI		B (months)	p value	95% CI		B (months)	p value
	Low	High			Low	High		
No	-8.4	-1.6	-5.0	0.99	-0.5	0.5	0.0	<0.01 ^a

^aVariables that passed selection criteria (i.e. p<0.25) for stepwise multivariable analyses

^bIGT: impaired glucose tolerance; GDM: gestational diabetes mellitus

Table 5
Predictors of age at BMI peak and rebound (in months) from stepwise regression analyses (n=1354)

	Crude analyses			Stepwise analyses		
	B (months)	95% confidence intervals		B (months)	95% confidence intervals	
		Low	High		Low	High
<i>Age at peak</i>						
Parental obesity						
Both parents non-obese	ref	—	—	ref	—	—
Only mother obese	0.8	0.3	1.3	0.6	0.1	1.1
Only father obese	0.2	-0.2	0.7	0.1	-0.4	0.6
Both parents obese	-0.1	-0.8	0.6	-0.3	-1.0	0.3
Hypertensive disorders of pregnancy						
Normal blood pressure	ref	—	—	ref	—	—
Gestational hypertension	0.1	-0.5	0.6	-0.1	-0.6	0.5
Chronic Hypertension	0.4	-0.9	1.6	0.3	-1.0	1.5
Pre-eclampsia	1.8	1.1	2.6	1.6	0.8	2.4
Maternal glucose tolerance status						
Normoglycemia	ref	—	—	ref	—	—
Isolated hyperglycemia	0.9	0.4	1.4	0.7	0.2	1.2
IGT [†]	-0.4	-1.2	0.4	-0.4	-1.2	0.4
GDM [‡]	0.8	0.1	1.4	0.5	-0.1	1.1
Child Sex						
Male	ref	—	—	ref	—	—
Female	0.8	0.5	1.1	0.8	0.5	1.1
Child race/ethnicity						
White	ref	—	—	ref	—	—
Black	-0.2	-0.7	0.2	-0.4	-0.8	0.1
Hispanic	0.2	-0.5	0.9	0.2	-0.5	0.9
Asian	-0.9	-1.6	-0.1	-0.9	-1.6	-0.2
Others	0.0	-0.5	0.5	0.0	-0.5	0.5

	Crude analyses			Stepwise analyses		
	B (months)	95% confidence intervals		B (months)	95% confidence intervals	
		Low	High		Low	High
<u>Age at rebound</u>						
Parental obesity						
Both parents non-obese	ref	—	—	ref	—	—
Only mother obese	-11.4	-14.9	-7.9	-11.1	-14.8	-7.5
Only father obese	-7.1	-10.5	-3.7	-6.3	-9.7	-2.9
Both parents obese	-12.3	-17.0	-7.7	-12.0	-16.8	-7.3
Parental education						
Both not university-educated	ref	—	—	ref	—	—
Mother university-educated	-0.4	-4.1	3.3	-1.8	-5.5	1.9
Father university-educated	1.2	-3.0	5.4	-1.4	-5.5	2.8
Both university-educated	5.9	3.2	8.5	3.1	0.2	6.0
Child Sex						
Male	ref	—	—	ref	—	—
Female	-7.4	-9.4	-5.3	-7.8	-9.8	-5.8
Child race/ethnicity						
White	ref	—	—	ref	—	—
Black	-5.4	-8.5	-2.3	-2.1	-5.3	1.1
Hispanic	-0.2	-5.3	5.0	-0.1	-5.3	5.0
Asian	0.4	-4.8	5.6	-2.0	-7.0	3.0
Others	-3.3	-6.9	0.4	-2.1	-5.6	1.4
Breastfeeding initiation						
Yes	ref	—	—	ref	—	—
No	-5.0	-8.4	-1.6	-3.4	-6.8	-0.02

[†] IGT: impaired glucose tolerance; GDM: gestational diabetes mellitus

Table 6 online
Multivariable associations of parental and child characteristics with age at BMI peak and rebound (n=1354)

	Age at peak		Age at rebound	
	B (months)	95% CI	B (months)	95% CI
Parental				
Maternal age (years)	— ^a	—	0.1 ^c	-0.2 0.3
Median annual income for census tract of residence at delivery	0.04 ^b	-0.03 0.1	0.1 ^c	-0.4 0.6
Parental college education				
Both parents not university-educated	— ^a	—	ref ^c	—
Only mother university-educated	— ^a	—	-2.6 ^c	-6.3 1.2
Only father university-educated	— ^a	—	-1.9 ^c	-6.2 2.4
Both parents university-educated	— ^a	—	2.1 ^c	-1.1 5.2
Nulliparous				
No	— ^a	—	— ^a	—
Yes	— ^a	—	— ^a	—
Maternal smoking history				
Never smoked	— ^a	—	ref ^c	—
Smoked prior to pregnancy	— ^a	—	-1.3 ^c	-3.9 1.3
Smoked during pregnancy	— ^a	—	-2.5 ^c	-5.9 1.0
Maternal height (m)	— ^a	—	0.1 ^c	-14.9 15.0
Paternal height (m)	— ^a	—	— ^a	—
1 st -trimester weight gain (kg)	0.04 ^b	-0.01 0.1	-0.2 ^c	-0.6 0.2
2 nd -trimester weight gain (kg)	-0.04 ^b	-0.1 0.03	-0.5 ^c	-1.0 0.01
3 rd -trimester weight gain (kg)	— ^a	—	— ^a	—
Parental obesity				
Both parents non-obese	ref ^b	—	ref ^c	—
Only mother obese	0.6 ^b	0.1 1.1	-11.4 ^c	-15.2 -7.7

	Age at peak			Age at rebound		
	B (months)	95% CI		B (months)	95% CI	
		Low	High		Low	High
Only father obese	0.1 ^b	-0.4	0.6	-6.5 ^c	-9.9	-3.1
Both parents obese	-0.3 ^b	-1.0	0.4	-12.2 ^c	-17.0	-7.4
Maternal glucose tolerance status						
Normoglycemia	ref ^b	—	—	ref ^c	—	—
Isolated hyperglycemia	0.7 ^b	0.2	1.2	-1.6 ^c	-5.2	1.9
IGT ^d	-0.4 ^b	-1.2	0.4	-4.5 ^c	-10.3	1.2
GDM ^d	0.5 ^b	-0.1	1.1	-0.1 ^c	-4.6	4.4
Hypertensive disorders of pregnancy						
Normal blood pressure	ref ^b	—	—	— ^a	—	—
Gestational hypertension	-0.05 ^b	-0.6	0.5	— ^a	—	—
Chronic Hypertension	0.2 ^b	-1.0	1.4	— ^a	—	—
Pre-eclampsia	1.6 ^b	0.8	2.4	— ^a	—	—
Child						
Sex						
Male	ref ^b	—	—	ref ^c	—	—
Female	0.8 ^b	0.5	1.1	-7.8 ^c	-9.9	-5.8
Race/ethnicity						
White	ref ^b	—	—	ref ^c	—	—
Black	-0.3 ^b	-0.7	0.2	-2.1	-5.5	1.3
Hispanic	0.3 ^b	-0.4	1.0	-0.01	-5.3	5.3
Asian	-0.8 ^b	-1.5	-0.1	-1.5	-6.6	3.7
Others	0.0 ^b	-0.5	0.5	-1.9	-5.5	1.7
Gestational age at delivery						
Term	— ^a	—	—	— ^a	—	—
Preterm	— ^a	—	—	— ^a	—	—

	Age at peak		Age at rebound	
	95% CI		95% CI	
	Low	High	Low	High
B (months)				
Birthweight-for-gestational age z-score	— ^a	—	0.9 ^c	2.0
Breastfeeding initiation				
Yes	— ^a	—	ref ^c	—
No	— ^a	—	−3.4 ^c	−0.03

^aVariables not included in the model because it did not meet inclusion criteria for multivariable analyses (i.e. $p > 0.25$ in crude analyses)

^bPredictor variables which passed inclusion criteria that are simultaneously included in the regression model for age at peak

^cPredictor variables which passed inclusion criteria that are simultaneously included in the regression model for age at rebound

^dIGT: impaired glucose tolerance; GDM: gestational diabetes mellitus

Table 7 online
Crude associations of parental and child characteristics with magnitude at BMI peak and rebound (n=1354)

	Magnitude at peak				Magnitude at rebound			
	B (kg/m ²)	95% CI		p value	B (kg/m ²)	95% CI		p value
		Low	High			Low	High	
<i>Parental</i>								
Maternal age (years)	-0.01	-0.02	0.01	0.26	-0.01	-0.02	0.01	0.26
Median annual income for census tract of residence at delivery	-0.04	-0.1	-0.002	0.04 ^a	-0.04	-0.1	-0.01	0.01 ^a
<i>Parental college education</i>								
Both parents not university-educated	ref	—	—	—	ref	—	—	—
Only mother university-educated	-0.1	-0.4	0.2	0.50	0.0	-0.2	0.2	0.99
Only father university-educated	-0.2	-0.5	0.1	0.17 ^a	-0.2	-0.4	0.1	0.20
Both parents university-educated	-0.4	-0.5	-0.2	<0.01 ^a	-0.4	-0.6	-0.2	<0.01 ^a
<i>Nulliparous</i>								
No	ref	—	—	—	ref	—	—	—
Yes	-0.1	-0.3	0.01	0.06 ^a	-0.1	-0.2	0.1	0.22 ^a
<i>Maternal smoking history</i>								
Never smoked	ref	—	—	—	ref	—	—	—
Smoked prior to pregnancy	0.04	-0.1	0.2	0.66	0.1	-0.1	0.2	0.53
Smoked during pregnancy	0.3	0.01	0.5	0.04 ^a	0.4	0.2	0.6	<0.01 ^a
Maternal height (m)	0.9	-0.1	2.0	0.08 ^a	0.7	-0.2	1.6	0.11 ^a
Paternal height (m)	-0.8	-1.7	0.1	0.09 ^a	-0.5	-1.3	0.3	0.25
1 st -trimester weight gain (kg)	0.01	-0.01	0.04	0.30	0.04	0.0	0.1	<0.01 ^a
2 nd -trimester weight gain (kg)	0.02	-0.01	0.1	0.21 ^a	0.0	-0.02	0.03	0.79
3 rd -trimester weight gain (kg)	0.02	0.0	0.05	0.07 ^a	0.01	-0.01	0.04	0.23 ^a
<i>Parental obesity</i>								
Both parents non-obese	ref	—	—	—	ref	—	—	—
Only mother obese	0.2	-0.04	0.5	0.11 ^a	0.6	0.4	0.8	<0.01 ^a
Only father obese	0.3	0.03	0.5	0.03 ^a	0.4	0.2	0.6	<0.01 ^a

	Magnitude at peak			Magnitude at rebound		
	B (kg/m ²)	95% CI		B (kg/m ²)	95% CI	
		Low	High		Low	High
Both parents obese	0.8	0.4	1.1	1.0	0.7	1.3
Maternal glucose tolerance status						
Normoglycemia	ref	—	—	ref	—	—
Isolated hyperglycemia	-0.1	-0.3	0.2	0.2	0.0	0.4
IGT ^b	-0.3	-0.8	0.1	-0.02	-0.4	0.3
GDM ^b	0.0	-0.3	0.4	0.3	-0.03	0.5
Hypertensive disorders of pregnancy						
Normal blood pressure	ref	—	—	ref	—	—
Gestational hypertension	0.03	-0.3	0.3	0.1	-0.2	0.3
Chronic Hypertension	-0.1	-0.7	0.6	-0.01	-0.6	0.6
Pre-eclampsia	0.3	-0.1	0.7	0.3	0.0	0.7
Child						
Sex						
Male	ref	—	—	ref	—	—
Female	-0.6	-0.7	-0.4	-0.2	-0.4	-0.1
Race/ethnicity						
White	ref	—	—	ref	—	—
Black	0.4	0.2	0.6	0.3	0.1	0.5
Hispanic	0.1	-0.3	0.5	0.1	-0.3	0.4
Asian	-0.5	-0.8	-0.1	-0.5	-0.9	-0.2
Others	0.03	-0.2	0.3	-0.1	-0.3	0.1
Gestational age at delivery						
Term	ref	—	—	ref	—	—
Preterm	-0.01	-0.3	0.3	-0.1	-0.3	0.2
Birthweight-for-gestational age z-score						
Breastfeeding initiation	0.3	0.2	0.4	0.2	0.2	0.3
Yes	ref	—	—	ref	—	—

No	Magnitude at peak				Magnitude at rebound			
	B (kg/m ²)	95% CI		p value	B (kg/m ²)	95% CI		p value
		Low	High			Low	High	
	0.1	-0.1	0.4	0.31	0.3	0.1	0.5	0.01 ^a

^aVariables that passed selection criteria (i.e. p<0.25) for stepwise multivariable analyses

^bIGT: impaired glucose tolerance; GDM: gestational diabetes mellitus

Table 8
Predictors of magnitude at BMI peak and rebound from stepwise regression analyses (n=1354)

	Crude analyses			Stepwise analyses		
	95% confidence intervals			95% confidence intervals		
	B (kg/m ²)	Low	High	B (kg/m ²)	Low	High
<i>Magnitude at peak</i>						
Parental obesity						
Both parents non-obese	ref	—	—	ref	—	—
Only mother obese	0.2	−0.04	0.5	0.02	−0.2	0.3
Only father obese	0.3	0.03	0.5	0.1	−0.1	0.4
Both parents obese	0.8	0.4	1.1	0.6	0.3	0.9
Parental education						
Both not university-educated	ref	—	—	ref	—	—
Mother university-educated	−0.1	−0.4	0.2	−0.03	−0.3	0.2
Father university-educated	−0.2	−0.5	0.1	−0.2	−0.5	0.1
Both university-educated	−0.4	−0.5	−0.2	−0.2	−0.4	−0.02
Maternal glucose tolerance status						
Normoglycemia	ref	—	—	ref	—	—
Isolated hyperglycemia	−0.1	−0.3	0.2	−0.04	−0.3	0.2
IGT [†]	−0.3	−0.8	0.1	−0.5	−0.9	−0.1
GDM [‡]	0.0	−0.3	0.4	−0.1	−0.4	0.2
Child Sex						
Male	ref	—	—	ref	—	—
Female	−0.6	−0.7	−0.4	−0.6	−0.7	−0.4
Child race/ethnicity						
White	ref	—	—	ref	—	—
Black	0.4	0.2	0.6	0.3	0.1	0.6
Hispanic	0.1	−0.3	0.5	−0.01	−0.4	0.4
Asian	−0.5	−0.8	−0.1	−0.2	−0.6	0.1
Others	0.03	−0.2	0.3	0.01	−0.2	0.3

	Crude analyses			Stepwise analyses		
	95% confidence intervals			95% confidence intervals		
	B (kg/m ²)	Low	High	B (kg/m ²)	Low	High
Birthweight-for-gestational age z-score	0.3	0.2	0.4	0.3	0.2	0.4
<i>Magnitude at rebound</i>						
Parental obesity						
Both parents non-obese	ref	—	—	ref	—	—
Only mother obese	0.6	0.4	0.8	0.4	0.2	0.7
Only father obese	0.4	0.2	0.6	0.3	0.1	0.5
Both parents obese	1.0	0.8	1.3	0.9	0.6	1.2
Parental college education						
Both not university-educated	ref	—	—	ref	—	—
Mother university-educated	-0.002	-0.2	0.2	0.1	-0.1	0.3
Father university-educated	-0.2	-0.4	0.1	-0.1	-0.3	0.2
Both university-educated	-0.4	-0.6	-0.2	-0.2	-0.4	-0.002
1st trimester weight gain	0.04	0.02	0.1	0.03	0.01	0.06
Maternal smoking history						
Never smoked	ref	—	—	ref	—	—
Smoked prior to pregnancy	0.1	-0.1	0.2	0.03	-0.1	0.2
Smoked during pregnancy	0.4	0.2	0.6	0.3	0.1	0.5
Child Sex						
Male	ref	—	—	ref	—	—
Female	-0.2	-0.4	-0.1	-0.2	-0.3	-0.1
Child race/ethnicity						
White	ref	—	—	ref	—	—
Black	0.3	0.1	0.5	0.2	-0.04	0.4
Hispanic	0.1	-0.3	0.4	-0.02	-0.3	0.3
Asian	-0.5	-0.9	-0.2	-0.3	-0.6	0.04
Others	-0.1	-0.3	0.1	-0.1	-0.4	0.1
Birthweight-for-gestational age z-score	0.2	0.2	0.3	0.2	0.2	0.3

[†]IGT: impaired glucose tolerance; GDM: gestational diabetes mellitus

Table 9 online

Multivariable associations of parental and child characteristics with magnitude at BMI peak and rebound (n=1354)

	Magnitude at peak			Magnitude at rebound		
	B (kg/m ²)	95% CI		B (kg/m ²)	95% CI	
		Low	High		Low	High
<i>Parental</i>						
Maternal age (years)	— ^a	—	—	— ^a	—	—
Median annual income for census tract of residence at delivery	−0.01 ^b	−0.05	0.03	−0.01 ^c	−0.04	0.02
Parental college education						
Both parents not college-educated	ref ^b	—	—	ref ^c	—	—
Only mother college-educated	−0.02 ^b	−0.3	0.2	0.1 ^c	−0.1	0.3
Only father college-educated	−0.2 ^b	−0.5	0.1	−0.1 ^c	−0.3	0.2
Both parents college-educated	−0.2 ^b	−0.4	0.04	−0.2 ^c	−0.4	0.01
Nulliparous						
No	ref ^b	—	—	ref ^c	—	—
Yes	−0.01	−0.2	0.1	0.1 ^c	−0.1	0.2
Maternal smoking history						
Never smoked	ref ^b	—	—	ref ^c	—	—
Smoked prior to pregnancy	0.02 ^b	−0.2	0.2	0.02 ^c	−0.1	0.2
Smoked during pregnancy	0.2 ^b	−0.1	0.4	0.2 ^c	0.04	0.5
Maternal height (m)	−0.2 ^b	−1.2	0.9	−0.1 ^c	−1.0	0.8
Paternal height (m)	— ^a	—	—	— ^a	—	—
1 st -trimester weight gain (kg)	— ^a	—	—	0.03 ^c	0.01	0.05
2 nd -trimester weight gain (kg)	0.02 ^b	0.0	0.1	— ^a	—	—
3 rd -trimester weight gain (kg)	−0.002 ^b	−0.03	0.02	0.01 ^c	−0.02	0.03
Parental obesity						
Both parents non-obese	ref ^b	—	—	ref ^c	—	—
Only mother obese	0.1 ^b	−0.2	0.3	0.4 ^c	0.2	0.7

	Magnitude at peak		Magnitude at rebound	
	B (kg/m ²)	95% CI Low High	B (kg/m ²)	95% CI Low High
Only father obese	0.1 ^b	-0.1 0.4	0.3 ^c	0.1 0.5
Both parents obese	0.6 ^b	0.3 1.0	0.9 ^c	0.6 1.2
Maternal glucose tolerance status				
Normoglycemia	ref ^b	— —	ref ^c	— —
Isolated hyperglycemia	-0.05 ^b	-0.3 0.2	0.2 ^c	-0.04 0.4
IGT ^d	-0.5 ^b	-0.9 -0.1	-0.2 ^c	-0.5 0.2
GDM ^d	-0.1 ^b	-0.5 0.2	0.1 ^c	-0.2 0.3
Hypertensive disorders of pregnancy				
Normal blood pressure	ref ^b	— —	ref ^c	— —
Gestational hypertension	0.1 ^b	-0.2 0.3	0.0 ^c	-0.2 0.2
Chronic Hypertension	0.1 ^b	-0.6 0.7	-0.03 ^c	-0.6 0.5
Pre-eclampsia	0.3 ^b	-0.1 0.7	0.09 ^c	-0.3 0.4
Child				
Sex				
Male	ref ^b	— —	ref ^c	— —
Female	-0.6 ^b	-0.7 -0.4	-0.2 ^c	-0.3 -0.1
Race/ethnicity				
White	ref ^b	— —	ref ^c	— —
Black	0.3 ^b	0.1 0.6	0.2 ^c	-0.05 0.4
Hispanic	-0.01 ^b	-0.4 0.4	-0.04 ^c	-0.4 0.3
Asian	-0.2 ^b	-0.6 0.2	-0.3 ^c	-0.6 0.05
Others	0.02 ^b	-0.2 0.3	-0.1 ^c	-0.3 0.1
Gestational age at delivery				
Term	— ^a	— —	— ^a	— —
Preterm	— ^a	— —	— ^a	— —

	Magnitude at peak		Magnitude at rebound	
	B (kg/m ²)	95% CI Low High	B (kg/m ²)	95% CI Low High
Birthweight-for-gestational age z-score				
	0.3 ^b	0.2 0.4	0.2 ^c	0.2 0.3
Breastfeeding initiation				
Yes	— ^a	— —	ref ^c	— —
No	— ^a	— —	0.1 ^c	-0.1 0.3

^aVariables not included in the model because it did not meet inclusion criteria for multivariable analyses (i.e. p>0.25 in crude analyses)

^bPredictor variables which passed inclusion criteria that are simultaneously included in the regression model for magnitude at peak

^cPredictor variables which passed inclusion criteria that are simultaneously included in the regression model for magnitude at rebound

^dGTT: impaired glucose tolerance; GDM: gestational diabetes mellitus