HHS Public Access

Author manuscript

Int J Obes (Lond). Author manuscript; available in PMC 2015 September 25.

Published in final edited form as:

Int J Obes (Lond). 2012 October ; 36(10): 1312–1319. doi:10.1038/ijo.2012.143.

Associations between maternal prepregnancy body mass index and child neurodevelopment at 2 years of age

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Abstract

OBJECTIVE—Both underweight and obese mothers have an increased risk for adverse offspring outcomes. Few studies have examined the association between prepregnancy body mass index (BMI) and children's neurodevelopment.

SUBJECTS—We used data from the nationally representative Early Childhood Longitudinal Study-Birth Cohort (ECLS-B; n =6850). Children were classified according to their mother's prepregnancy BMI (kg m^{-2}) status: underweight (BMI <18.5), normal weight (BMI 18.5–24.9), overweight (BMI 25.0–29.9), obese class I (BMI 30.0–34.9), and obese class II and III (BMI ≥35.0). Children's age-adjusted mental development index (MDI) and psychomotor development index (PDI) T-scores (mean 50, s.d. 10) were obtained using a validated shortened version of the Bayley Scales of Infant Development-II at approximately 2 years of age. While adjusting for sociodemographics, we estimated the average MDI and PDI scores or the risk of delayed (< −1 s.d. vs >1 s.d.) mental or motor development, relative to children of normal weight mothers.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

DISCLAIMER

The findings and conclusions in this article are those of the authors and do not necessarily represent the official position of the Centers for Disease Control and Prevention.

RESULTS—Compared with children of normal weight mothers, MDI scores were lower among children of mothers of all other prepregnancy BMI categories, with the greatest adjusted difference among children of class II and III obese mothers (−2.13 (95% CI −3.32, −0.93)). The adjusted risk of delayed mental development was increased among children of underweight (risk ratio (RR) 1.36 (95% CI 1.04, 1.78)) and class II and III obese (RR 1.38 (95% CI 1.03, 1.84)) mothers. Children's PDI scores or motor delay did not differ by maternal prepregnancy BMI.

CONCLUSION—In this nationally representative sample of 2-year-old US children, low and very-high maternal prepregnancy BMI were associated with increased risk of delayed mental development but not motor development.

Keywords

pregnancy; neurodevelopment; mental; psychomotor; underweight; body mass index

INTRODUCTION

In the United States, only half of pregnant women have a healthy weight at the start of their pregnancy.[1] Maternal underweight is a known risk factor for preterm delivery and low birth weight,[2] both of which are predictors of adverse brain development.[3] Conversely, maternal obesity is a strong risk factor for cesarean delivery and fetal macrosomia.[4] Furthermore, prepregnancy obesity has been recently hypothesized to have a long-term impact on offspring neurodevelopment.[5] Maternal obesity is associated with an altered intrauterine environment owing to increased inflammation, metabolic stress, and lipotoxicity.[6–8] It has been suggested that this adverse intrauterine environment may directly damage the developing fetal brain causing offspring cognitive, behavioral, and motor development delays.[9–12]

Epidemiological research supporting an association between maternal prepregnancy body mass index (BMI) and children's neurodevelopment is beginning to emerge. Results from three studies suggest that prepregnancy obesity may be associated with lower general cognitive abilities in children;[13–15] however, these findings were not replicated in a study of children from two large cohorts.[16] These studies were based on data ranging from 1966 to 2008 and were limited in that they either used a small sample,[13] different measures of child neurodevelopment (that is, degree of delay determined by disability diagnosis[14] or maternal report),[16] or had limited data to examine all maternal prepregnancy BMI categories.[14,16] Only one of the previous studies has examined children's motor development and did not observe an association.[13] The purpose of our study was to examine the relationship between maternal prepregnancy BMI status and standardized measures of mental and motor development among US children at 2 years of age.

MATERIALS AND METHODS

Study participants

Our study consisted of a nationally representative sample of US children born in 2001, who were enrolled in the Early Childhood Longitudinal Study-Birth Cohort (ECLS-B).[17] As required by the Department of Education, which sponsored the ECLS-B, we reported sample

sizes rounded to the nearest 50 and presented all percentages as weighted population estimates. ECLS-B participants were selected from birth certificate data collected by the National Center for Health Statistics' Vital Statistics System. For this analysis, we used only data collected on children and their parents during the first two waves of the ECLS-B, when the children were approximately 9 months and 2 years of age.

At the baseline interview, the ECLS-B enrolled 10,700 infants, which included an oversample of select racial-ethnic groups, twins, and infants born weighing <2500 g. We limited our analysis to singleton infants without major structural or genetic congenital anomalies, as reported on their birth certificate (n =8850). Of those meeting study inclusion criteria, 750 were lost to follow-up by 2 years, 800 were excluded because no child neurocognitive assessment was completed, and an additional 450 infants were excluded owing to missing data for other relevant variables. Our final analytical sample consisted of 6850 children, weighted to be representative of approximately 3.6 million singleton US children born in 2001.

Maternal prepregnancy BMI

When study children were approximately 9 months of age, mothers were asked their current height and their prepregnancy weight. Prepregnancy BMI (kg m $^{-2}$) was calculated and categorized according to the World Health Organization guidelines:[18] underweight (BMI <18.5), normal weight (BMI 18.5–24.9), overweight (BMI 25.0–29.9), obese class I (BMI 30.0–34.9), obese class II (BMI 35.0–39.9), and obese class III (BMI ≥40.0). Because of sample size limitations, we collapsed obese class II and III into one category representing severely obese mothers.

Child neurodevelopment

Children's mental and motor development were assessed when they were 20–38 months of age (interquartile range 24–25 months) by certified fieldworkers using a shortened version of the Bayley Scales of Infant Development-II (BSID-II), the Bayley Short Form-Research Edition (BSF-R). The BSF-R, developed specifically for the ECLS-B, was equated to the BSID-II and determined to have good reliability and discrimination parameters.[19] BSF-R results are expressed as age-standardized T-scores (mean 50, s.d. 10), were normalized to the ELCS-B population, and provide a measure of children's mental and motor abilities relative to other children of the same age. The BSF-R has two components, the mental development index (MDI), which indicates children's language and cognitive abilities, and the psychomotor development index (PDI), which indicates children's fine and gross motor skills.[20] On both indices, scores < −2 s.d. are considered indicative of severe developmental delay, and scores from −1 to −2 s.d. are considered indicative of mild developmental delay.[21] Due to sample size limitations, we were unable to study only severe delay and therefore used a cutoff of ≤1 s.d. to represent children with mild or severe delay, who may require closer monitoring and referral for additional services.[22,23]

Additional variables

Mother's age at delivery (15–19, 20–24, 25–29, 30–34, 35–50 years), race-ethnicity (non-Hispanic white, non-Hispanic black, Hispanic, Asian/Pacific Islander/Native Hawaiian,

American Indian/Alaskan Native), parity (primiparous, multiparous), marital status (married, unmarried), and schooling (<12, 12, 13–15, ≥16 years), smoking during the last trimester of pregnancy (yes, no), diabetes before or during pregnancy (yes, no), and chronic or pregnancy-inducted hypertension (yes, no); and child's sex (male, female), gestational age (weeks), and birthweight (grams) were obtained from the child's birth certificate. Birthweight-for-gestational-age status was assessed according to a national reference[24] and classified as small-(<10th percentile), adequate—(10–90th percentile), and large-for-gestational-age (>90th percentile). Poverty status was determined by maternal report of household income and size at the 2-year interview and dichotomized as <185 and ≥185% of the federal poverty limit.[20] Gestational weight gain was obtained from the child's birth certificate (82% of records) or from maternal report when the child was approximately 9 months of age. Gestational weight gain adequacy was assessed on the basis of the 2009 Institute of Medicine recommendations according to maternal prepregnancy BMI status:[25] 28–40 lbs (BMI <18.5); 25–35 lbs (BMI 18.5–24.9); 15–25 lbs (BMI 25.0–29.9); and 11–20 lbs (BMI ≥30.0). Breastfeeding status was assessed at the 9-month and 2-year parent interviews. Duration was censored at the child's age at the 2-year interview if the mother indicated that she was still breastfeeding at that time (<1, 1–5, 6–12, >12 months).

Statistical analysis

We used analysis of variance to examine the weighted mean (s.e.) MDI and PDI scores across sample characteristics. Sociodemographic covariates chosen *a priori* were maternal age, race-ethnicity, marital status, schooling, parity, and poverty. Child sex was included in all the models because of the large variation in developmental scores by child sex. Because associations related to child development vary by socioeconomic indicators, we examined a multiplicative statistical heterogeneity by household poverty status. We used linear regression to estimate the associations between children's MDI and PDI scores and maternal prepregnancy BMI category. We calculated model-adjusted risk ratios for the associations between maternal prepregnancy BMI category and children's risk of delayed mental or motor development.[26] All results are expressed as comparisons with children of normal weight mothers.

We performed multiple sub-analyses. Because the use of adult BMI-classification cutpoints has been found to overestimate the prevalence of overweight and obesity among adolescents,[25] we performed a sensitivity analysis in which we compared our main results for the entire study population with results of an analysis limited to the children of mothers aged ≥18 years (n =6600). In a separate sub-analysis, we were interested in examining whether the observed associations were due to downstream variables that could be acting as mediators of the association. We limited the analysis to full-term (gestational age ≥37 weeks) children born to mothers without diabetes or hypertension (n =5000). For this sub-analysis, we examined the data for a multiplicative statistical interaction between prepregnancy BMI status and gestational weight gain adequacy. We then estimated the associations between children's MDI and PDI scores or delay and maternal prepregnancy BMI category consecutively adjusted for sociodemographics, gestational weight gain, birthweight-for-gestational-age status, and breastfeeding duration.

We used Statistical Analysis Software (SAS) version 9.2 (Cary, NC, USA) with SAS-Callable SUDAAN version 10.0.1 (Research Triangle Park, NC, USA) and considered *P*-values <0.05 to be indicative of significant differences for main effects and <0.15 to be indicative of significant interactions. We chose the higher *P*-value for interactions because of the low power associated with tests of heterogeneity.[27] All analyses were weighted to account for subjects lost to follow-up and for unequal probabilities of subject selection by various demographic factors. To account for the multistage, stratified, cluster design of the ECLS-B, we used Taylor Series approximations to estimate standard errors. This analysis was reviewed by a human subject coordinator at the CDC and determined not to be research involving human subjects.

RESULTS

Compared with children retained in the analytical sample, those excluded because of incomplete data were more likely to have mothers who were Hispanic, unmarried, had less schooling, low-income, and had lower MDI and PDI scores (data not shown); no differences were observed by maternal age, parity, smoking during pregnancy, prepregnancy BMI, or child sex.

MDI scores varied across all maternal and child characteristic sociodemographcis, whereas PDI scores varied by all characteristics, except maternal age and prepregnancy BMI classification (Table 1). All maternal and child characteristics, with the exception of child sex, and proportion of children with delayed mental, but not motor development, varied significantly by maternal pre-pregnancy BMI status (Table 2). Relative to the distribution of MDI scores among children of normal weight mothers, the MDI distribution among children of mothers in all the other prepregnancy BMI categories were shifted to the left (that is, the MDI scores were lower; Figure 1).

We found no significant multiplicative heterogeneity by poverty status for MDI or PDI scores, thus we present poverty-adjusted results rather than stratified results. Compared with adjusted MDI scores of children of normal weight mothers, MDI scores were lower among children of mothers in all the other prepregnancy BMI categories, although only significantly lower among children of obese class II and III mothers (Table 3). The risk of delayed mental development was significantly higher among children born to underweight or severely obese mothers than among those born to normal weight mothers. Children's PDI scores or the risk of delayed motor development did not vary significantly by maternal prepregnancy BMI status (Table 3).

Restricting our analyses to only children of mothers aged ≥18 years did not meaningfully change our estimates, although doing so did decrease the precision of these estimates because of the reduction in sample sizes (data not shown). When we limited the analysis to full-term children born to mothers without diabetes or hypertension, with the exception of the interaction between prepregnancy BMI status and gestational weight gain adequacy for delayed motor development (P =0.10), there was no significant multiplicative heterogeneity by gestational weight gain adequacy. When we examined the interaction in detail, there were no meaningful differences in the risk of delayed motor development across the BMI groups

by gestational weight gain adequacy (data not shown) and thus we did not include the interaction in any of our sensitivity analyses. The final sensitivity analysis models were adjusted for gestational weight gain adequacy, birthweight-for-gestational-age status, and breastfeeding duration. Adjusted MDI and PDI scores and the risk of delayed development were not meaningfully different from the results based on the full sample (Table 4).

DISCUSSION

Findings from this nationally representative cohort of children approximately 2 years of age showed a significant association between maternal prepregnancy BMI status and children's mental development but no association with children's motor development. The association with children's cognitive development was non-linear, such that the most favorable outcomes were observed among children of normal weight mothers and the least favorable among children of mothers with a prepregnancy BMI at either extreme of the distribution (that is, underweight or severely obese). The mean MDI score of children of severely obese mothers was approximately a quarter of a s.d. below that of children of normal weight mothers. Although this difference may seem fairly small, it likely has a notable public health impact,[28] given the current US obesity epidemic.[29] In our sample, which was representative of >3 million children born in 2001, 5 and 6% of children were born to mothers whom were either underweight or severely obese at the start of the their pregnancy, respectively. In each of these groups of children, approximately 19% had delayed mental development at 2 years of age, compared with only 12% among children born to normal weight mothers. This excess risk equates to a non-trivial number of US children who may require social services and referral to early intervention programs and be at risk for long-term adverse financial and health consequences.[30,31]

Research concerning the relationship between maternal pre-pregnancy BMI and children's neurodevelopment has only begun to emerge.[13–16] Similar to our study, a small study among low-income African-American children aged 5 years showed that maternal prepregnancy obesity was associated with lower general cognitive ability (IQ), but not gross motor development, as determined by standard psychometric tests.[13] Additional evaluation of the cognitive test results suggested that the association between maternal obesity and lower test scores primarily reflected a delay in the development of non-verbal cognitive skills (that is, pattern and spatial ability) rather than verbal skills (that is, receptive language, syntax, and concepts). In our study, we were unable to examine associations among the separate cognitive domains. Further, although that study lacked the statistical power necessary to determine whether the cognitive development of children of underweight mothers was significantly lower, the authors suggested that there may be an inverted U-shaped relationship between mothers' prepregnancy BMI and children's cognitive function.[13] Results of a study from Northern Finland indicated that among children aged 11 years, born in 1966, those whose mothers were underweight had an increased risk for mild cognitive delay, and that among children born in 1985–86, those whose mothers were obese had an increased risk for both mild and severe cognitive delay.[14] Although the U-shaped relationship was not present at each time period, the authors concluded this relationship may be present. Results of another study of US children aged 5–7 years observed the highest reading and mathematics scores among children of normal weight mothers; however, the

difference was only statistically significant compared with children of obese mothers.[15] In contrast to these studies, results of a study involving two cohorts of European children approximately 3 years of age showed no association between maternal overweight/obesity and children's cognitive abilities.[16] However, that study was limited in that the authors collapsed maternal overweight and obesity into one category, the prevalence of overweight/obesity in the two study cohorts (21–22%) was much lower than in the ECLS-B (39%), and assessment of children's cognitive abilities was based on subjective maternal report rather than objective assessments.

The observed association between mother's prepregnancy BMI and children's mental development may be attributable to several mechanisms. Potential biological mechanisms may include inadequate prenatal micronutrient status,[32–35] elevated stress levels,[36–38] and an inflammatory intrauterine environment.[6,9,10] Specifically, obesity related intrauterine inflammation may have a direct effect on the cognitive function of offspring by damaging the developing fetal brain,[9,10] or by making the developing fetus more susceptible to other intrauterine environmental insults by increasing the permeability of the fetal blood–brain barrier.[12]

Other hypothesized downstream pathways include gestational weight gain, maternal metabolic conditions during pregnancy, preterm birth, birthweight, breastfeeding, or the postnatal environment. With regard to gestational weight gain, gains below the amount required for the products of conception have been hypothesized to be associated with delayed development via inadequate fetal growth or maternal ketosis.[25] By contrast, excessive gestational weight gain may exacerbate already elevated inflammatory levels associated with obesity,[39,40] thus further hindering development. Children born to mothers with diabetes, particularity uncontrolled diabetes, have been shown to have an elevated risk for adverse development, owing to hyperglycemia and other associated intrauterine factors.[41] Preterm birth and/or growth restriction is another strong risk factor for adverse neurodevelopment.[3,42] Postnatal nutritional factors, such as breastfeeding, may also affect early child development, although this pathway could be either biological or behavioral.[43] In our sensitivity analyses limited to full-term children born to non-diabetic and non-hypertensive mothers, after adjustment for sociodemographics, gestational weight gain, birthweight, and breastfeeding duration, the strength and direction of our findings were maintained. Lastly, postnatal environmental factors are extremely important for children's cognitive development; however, many enrichment and lifestyle factors that influence development are driven by household socioeconomics.[44] We have considered, and ruled out, poverty as an effect modifier and therefore adjusted all analyses by household poverty status. Nonetheless, further investigations designed to understand the biological or environmental mechanisms of this unfavorable association are critically needed.

We did not observe an association between maternal pre-pregnancy BMI status and children's motor development at 2 years of age. Although we hypothesized that there would be a U-shaped relationship, this null finding is similar to the findings of a previous study.[13] Motor development in young children is fairly robust to non-extreme influences and there is considerable variation in the age at which healthy children reach motor developmental milestones.[45] Therefore, population-level assessments of delayed motor development may

be difficult to detect using broad-based assessments.[46] Thus it is plausible that our study, nor the other study,[13] used assessment measures with high enough sensitivity and future studies should continue to examine long-term motor outcomes.

Strengths of our study included our assessment of the increased risk for neurodevelopment delays among children of mothers who were severely obese before their pregnancy, which to our knowledge has not been previously done. Given that about half of the obese population is severely obese,[29] our finding have important public health implications. An additional strength of our study is that our analyses were based on data collected from a large, diverse population-based sample of children whose development was objectively measured by rigorously trained fieldworkers.[19] Utilizing certified interviewers reduced the potential for maternal response bias in our study. We also examined and ruled-out heterogeneity by poverty status, which has been shown to be one of the strongest predictors of cognitive disabilities.

Study limitations included our reliance on mother's estimates of prepregnancy weight at 9-months postpartum. Because lighter women tend to overreport and heavier women tend to under-report their weight, prepregnancy BMI may have been misclassified.[47] We have no reason to suspect that reporting would be differential by developmental outcome assessed at 2 years of age and thus our findings may be biased toward the null. Because we restricted our analysis to children with complete data for all covariates, our sample may not be fully representative of all singleton US children born in 2001; however, the data were weighted to account for attrition and non-response. Furthermore, we did not have any data regarding why children who were initially enrolled in the ECLS-B may have dropped out and therefore we cannot rule out the possibility of survival bias. We cannot rule out residual confounding by lifestyle factors associated with prepregnancy BMI; however, we did adjust for many sociodemographics, including maternal schooling and household poverty, gestational weight gain, and breastfeeding status in our sensitivity analyses, all of which may be correlated with maternal/family lifestyle, and observed only small changes in estimates. Lastly, our study was based on mental and motor development measures obtained from the BSF-R, shortened version of the BSID-II. The BSID-II was previously recommended for neurodevelopmental assessments in large population-based studies;[48] however, pilot testing determined that it was too time consuming for the ECLS-B. Although the BSF-R has been shown to have good psychometric properties,[19] the predictive validity of the BSF-R has not been studied and the predictive validity of the BSID-II remains unclear.[49] Therefore, more studies of the relationship between maternal prepregnancy BMI status and neurodevelopment among older children will be necessary to clarify the implications of our findings.

In conclusion, we found that both low and high maternal prepregnancy BMI status was associated with poor cognitive development among 2-year-old children. Although the exact mechanism remains unclear, our findings suggest that children born to underweight and severely obese mothers have an increased risk of delayed mental development and may therefore require closer monitoring for developmental delays. This study also reinforces the importance of encouraging all women of reproductive age to achieve a healthy weight and lifestyle before pregnancy. Although there are barriers to such preconception weight loss,[50]

including the high proportion of unplanned pregnancies,[51] increasing the proportion of US women who are at a healthy weight when they conceive is a current public health priority.[52] Although the prevalence of prepregnancy underweight appears to be declining, prepregnancy obesity, including class II and III obesity, has continued to increase.[53] This study adds to the growing body of literature documenting the diverse and long-term adverse child outcomes that are associated with maternal obesity.

Acknowledgments

This research was performed under an appointment to the Centers for Disease Control and Prevention, administered by the Oak Ridge Institute for Science and Education under contract number DE-AC05–06OR23100 between the US Department of Energy and Oak Ridge Associated Universities.

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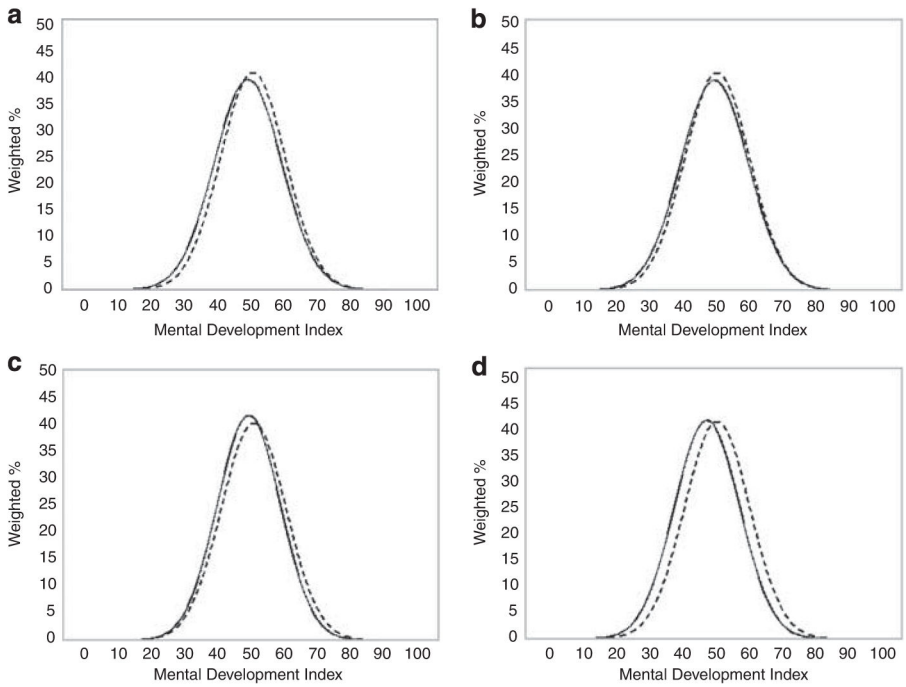

Figure 1.

Distribution of age-standardized child mental development index *T*-scores at approximately 2 years of age by maternal prepregnancy BMI status: (**a**) underweight; (**b**) overweight; (**c**) obese class I; (**d**) obese class II and III, represented by the solid lines. All dashed lines indicate the distribution among children of normal-weight mothers.

Table 1

Child mental and motor development scores at 2 years of age by maternal and child characteristics, Early Childhood Longitudinal Study-Birth Cohort, 2001–2004

Sample characteristics	*n* (%)[a]	Mental development index (MDI)		Psychomotor development index (PDI)	
		Mean (s.e.)	*P*	Mean (s.e.)	*P*
Overall	6850 (100)	50.31 (0.22)		50.11 (0.24)	
Maternal age, years			<0.001		0.13
15–19	850 (11)	48.13 (0.46)		49.78 (0.45)	
20–24	1750 (25)	48.72 (0.35)		49.84 (0.34)	
25–29	1700 (27)	50.54 (0.34)		50.03 (0.46)	
30–34	1600 (23)	52.12 (0.36)		50.51 (0.31)	
35–50	1000 (14)	51.51 (0.43)		50.37 (0.42)	
Maternal raceethnicity			*<0.001*		*<0.001*
White, non-Hispanic	3200 (61)	52.36 (0.23)		50.14 (0.29)	
Black, non-Hispanic	1150 (15)	47.66 (0.40)		51.92 (0.42)	
Hispanic	1100 (20)	46.08 (0.40)		48.82 (0.40)	
Asian/Pacific Islander/Native Hawaiian	1050 (4)	49.84 (0.33)		49.42 (0.33)	
American Indian/Alaskan Native	350 (1)	48.11 (0.54)		49.76 (0.61)	
Maternal marital status			*<0.001*		*0.007*
Married	4550 (68)	51.48 (0.25)		50.31 (0.28)	
Unmarried	2300 (32)	47.85 (0.28)		49.70 (0.32)	
Maternal schooling, years			*<0.001*		*<0.001*
<12	1350 (20)	46.10 (0.40)		48.81 (0.42)	
12	2100 (32)	49.25 (0.27)		50.04 (0.35)	
13–15	1500 (22)	51.07 (0.39)		50.26 (0.42)	
≥16	1900 (26)	54.32 (0.30)		51.11 (0.37)	
Maternal parity			*<0.001*		*0.01*
Primiparous	2850 (41)	51.06 (0.27)		50.43 (0.30)	
Multiparous	4000 (59)	49.80 (0.26)		49.89 (0.28)	
Maternal prenatal smoking			*<0.001*		*0.002*
No	800 (88)	50.48 (0.23)		50.23 (0.24)	

Sample characteristics	*n* (%)[a]	Mental development index (MDI)		Psychomotor development index (PDI)	
		Mean (s.e.)	*P*	Mean (s.e.)	*P*
Yes	6050 (12)	49.01 (0.41)		49.21 (0.46)	
Household poverty			*<0.001*		*<0.001*
<185%	3150 (45)	47.53 (0.27)		49.47 (0.32)	
≥185%	3700 (55)	52.59 (0.20)		50.64 (0.28)	
Child sex			*<0.001*		*<.001*
Male	3550 (51)	48.63 (0.26)		49.38 (0.26)	
Female	3350 (49)	52.08 (0.27)		50.88 (0.30)	
Maternal prepregnancy BMI			*<0.001*		*0.13*
Underweight	500 (5)	49.49 (0.64)		48.98 (0.63)	
Normal weight	3850 (56)	50.83 (0.24)		50.17 (0.26)	
Overweight	1550 (25)	50.16 (0.38)		50.19 (0.36)	
Obese class I	600 (8)	49.52 (0.56)		50.32 (0.51)	
Obese class II and III	400 (6)	47.91 (0.67)		49.94 (0.69)	

Abbreviation: BMI, body mass index.

[a]Presented an unweighted sample size rounded to the nearest 50 and a weighted percentage per data agreement with the Department of Education. Percentages may not add to 100 due to rounding.

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Table 2

Sample maternal and child characteristics according to maternal prepregnancy BMI status, Early Childhood Longitudinal Study-Birth Cohort, United States, 2001–2004

Sample characteristics	Underweight (*n* =500) *n* (%)[a]	Normal weight (*n* =3850) *n* (%)[a]	Overweight (*n* =1550) *n* (%)[a]	Obese class I (*n* =600) *n* (%)[a]	Obese class II and III (*n* =400) *n* (%)[a]	*P*
Maternal age, y						*<0.001*
15–19	100 (20)	550 (13)	150 (8)	<50 (6)	<50 (7)	
20–24	150 (31)	900 (24)	450 (26)	150 (25)	100 (25)	
25–29	100 (23)	950 (27)	350 (26)	150 (33)	100 (27)	
30–34	100 (21)	900 (23)	350 (25)	150 (23)	100 (28)	
35–50	<50 (5)	600 (13)	250 (16)	100 (12)	50 (14)	
Maternal race-ethnicity						*<0.001*
White, non-Hispanic	200 (58)	1800 (63)	750 (61)	250 (56)	200 (56)	
Black, non-Hispanic	50 (14)	550 (13)	300 (14)	150 (19)	100 (25)	
Hispanic	50 (19)	550 (18)	300 (22)	100 (24)	50 (17)	
Asian/Pacific Islander/Native Hawaiian	150 (8)	750 (5)	150 (2)	<50 (1)	<50 (1)	
American Indian/Alaskan Native	<50 (1)	150 (1)	100 (1)	<50 (1)	<50 (1)	
Maternal marital status						*0.002*
Married	300 (59)	2650 (69)	1000 (70)	350 (66)	250 (61)	
Unmarried	200 (41)	1250 (31)	500 (30)	200 (34)	150 (39)	
Maternal schooling, y						*<0.001*
<12	100 (26)	750 (20)	300 (21)	100 (20)	50 (20)	
12	150 (40)	1100 (30)	500 (32)	200 (34)	150 (38)	
13–15	100 (20)	800 (21)	350 (23)	150 (26)	100 (29)	
≥16	100 (15)	1250 (30)	350 (25)	100 (19)	50 (13)	
Maternal parity						*<0.001*
Primiparous	250 (52)	1700 (43)	600 (36)	200 (33)	150 (33)	
Multiparous	250 (48)	2150 (57)	950 (64)	400 (68)	250 (67)	
Maternal prenatal smoking						*<0.001*
No	400 (81)	3450 (89)	1350 (91)	500 (86)	350 (88)	
Yes	100 (19)	400 (11)	200 (9)	100 (14)	50 (12)	
Household poverty						*<0.001*

Sample characteristics	Underweight (*n* =500) *n* (%)[a]	Normal weight (*n* =3850) *n* (%)[a]	Overweight (*n* =1550) *n* (%)[a]	Obese class I (*n* =600) *n* (%)[a]	Obese class II and III (*n* =400) *n* (%)[a]	*P*
<185%	250 (56)	1600 (41)	800 (47)	300 (49)	200 (57)	
≥185%	250 (45)	2300 (59)	750 (53)	300 (51)	150 (43)	
Child sex						*0.76*
Male	250 (50)	2000 (51)	800 (53)	300 (50)	200 (47)	
Female	250 (50)	1850 (49)	750 (47)	300 (50)	200 (53)	
Delayed mental development						*0.006*
No	400 (81)	3350 (88)	1300 (86)	500 (87)	300 (81)	
Yes	100 (19)	550 (12)	250 (14)	100 (13)	50 (19)	
Delayed motor development						*0.87*
No	400 (85)	3300 (86)	1300 (85)	500 (86)	350 (84)	
Yes	100 (15)	600 (14)	250 (15)	100 (14)	50 (17)	

[a]Presented an unweighted sample size rounded to the nearest 50 and a weighted percentage per data agreement with the Department of Education. Percentages may not add to 100 due to rounding.

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Table 3

Associations between maternal prepregnancy BMI status and child mental and motor development at approximately 2 years of age, Early Childhood Longitudinal Study-Birth Cohort, United States, 2001–2004

	Underweight (*n* =500)	Normalweight (*n* =3850)	Overweight (*n* =1550)	Obese class I (*n* =600)	Obese class II and III (*n* =400)
MDI score, β (95% CI)					
Unadjusted	−1.34 (−2.71, 0.02)	0.00 (referent)	−0.68 (−1.38, 0.02)	−1.32 (−2.58, −0.06)	−2.92 (−4.19, −1.65)
Adjusted[a]	−0.47 (−1.77, 0.84)	0.00 (referent)	−0.21 (−0.88, 0.46)	−0.57 (−1.63, 0.48)	−2.13 (−3.32, −0.93)
MDI <40 vs ≥40, RR (95% CI)					
Unadjusted	1.51 (1.12, 2.04)	1.00 (referent)	1.12 (0.94, 1.33)	1.05 (0.79, 1.38)	1.50 (1.14, 1.98)
Adjusted[a]	1.36 (1.04, 1.78)	1.00 (referent)	1.03 (0.86, 1.22)	0.94 (0.73, 1.23)	1.38 (1.03, 1.84)
PDI score, β (95% CI)					
Unadjusted	−1.20 (−2.44, 0.05)	0.00 (referent)	0.02 (−0.61, 0.65)	0.15 (−0.90, 1.20)	−0.24 (−1.61, 1.13)
Adjusted[a]	−0.93 (−2.16, 0.31)	0.00 (referent)	0.13 (−0.52, 0.78)	0.22 (−0.82, 1.26)	−0.30 (−1.69, 1.09)
PDI <40 vs ≥40, RR (95% CI)					
Unadjusted	1.06 (0.77, 1.48)	1.00 (referent)	1.05 (0.89, 1.24)	0.99 (0.75, 1.31)	1.17 (0.85, 1.61)
Adjusted[a]	1.02 (0.73, 1.42)	1.00 (referent)	1.02 (0.86, 1.22)	0.97 (0.74, 1.29)	1.17 (0.85, 1.62)

Abbreviations: BMI, body mass index; CI, confidence interval; MDI, mental development index; PDI, psychomotor development index; RR, risk ratio.

[a]Adjusted for maternal age (continuous), race-ethnicity, marital status, parity, schooling (continuous), smoking during pregnancy, household poverty status and child sex.

Table 4

Associations between maternal prepregnancy BMI status and child mental and motor development at approximately 2 years of age, based on a sensitivity analysis limited to full-term children born to non-diabetic and non-hypertensive mothers, Early Childhood Longitudinal Study-Birth Cohort, United States, 2001–2004

	Underweight (*n* =350)	Normal weight (*n* =2950)	Overweight (*n* =1100)	Obese class I (*n* =400)	Obese class II and III (*n* =250)
MDI score, β (95% CI)					
Unadjusted	− 1.10 (−2.67, 0.47)	0.00 (referent)	−0.91 (−1.74, −0.08)	−1.04 (−2.43, 0.36)	−3.72 (−5.21, −2.22)
Adjusted[a]	− 0.19 (−1.65, 1.27)	0.00 (referent)	−0.18 (−0.97, 0.61)	−0.04 (−1.29, 1.22)	−2.30 (−3.66, −0.93)
MDI <40 vs ≥40, RR (95% CI)					
Unadjusted	1.52 (1.09, 2.12)	1.00 (referent)	1.20 (0.98, 1.46)	0.98 (0.69, 1.39)	1.78 (1.31, 2.44)
Adjusted[a]	1.32 (0.97, 1.79)	1.00 (referent)	1.09 (0.89, 1.35)	0.88 (0.62, 1.24)	1.54 (1.14, 2.10)
PDI score, β (95% CI)					
Unadjusted	− 0.85 (−2.21, 0.51)	0.00 (referent)	0.29 (−0.40, 0.98)	0.74 (−0.53, 2.00)	−0.33 (−2.04, 1.39)
Adjusted[a]	− 0.45 (−1.80, 0.91)	0.00 (referent)	0.40 (−0.33, 1.13)	0.75 (−0.51, 2.01)	−0.50 (−2.24, 1.23)
PDI <40 vs ≥40, RR (95% CI)					
Unadjusted	0.97 (0.66, 1.42)	1.00 (referent)	0.96 (0.78, 1.20)	0.95 (0.68, 1.33)	1.31 (0.92, 1.87)
Adjusted[a]	0.88 (0.59, 1.31)	1.00 (referent)	0.95 (0.75, 1.20)	0.95 (0.67, 1.34)	1.30 (0.90, 1.89)

Abbreviations: BMI, body mass index; CI, confidence interval; MDI, mental development index; PDI, psychomotor development index; RR, risk ratio.

[a]Adjusted model: maternal age (continuous), race-ethnicity, marital status, parity, schooling (continuous), smoking during pregnancy, household poverty status, child sex, gestational weight gain adequacy, birthweight-for-gestational-age status and breastfeeding duration.