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Prepregnancy obesity is associated with lower psychomotor development scores in boys at age 3 in a low income, minority birth cohort

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Abstract

Whether maternal obesity and gestational weight gain (GWG) are associated with early-childhood development in low-income, urban, minority populations, and whether effects differ by child sex remain unknown. This study examined the impact of prepregnancy BMI and GWG on early childhood neurodevelopment in the Columbia Center for Children's Environmental Health (CCCEH) Mothers and Newborns study. Maternal prepregnancy weight was obtained by self-report and GWG was assessed from participant medical charts. At child age three years, the Psychomotor Development Index (PDI) and Mental Development Index (MDI) of the Bayley Scales of Infant Intelligence (BSID-II) were completed. Sex-stratified linear regression models assessed associations between prepregnancy BMI and pregnancy weight gain z-scores with child PDI and MDI scores, adjusting for covariates. Of 382 women, 48.2% were normal weight before pregnancy, 24.1% overweight, 23.0% obese, and 4.7% underweight. At three years, mean scores on the PDI and MDI were higher among girls compared to boys [PDI: 102.3 vs. 97.2, p=0.0002; MDI: 92.8 vs. 88.3, p=0.0001]. In covariate adjusted models, maternal obesity was markedly

CONFLICTS OF INTEREST

None

ETHICAL STANDARDS

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national guidelines on human experimentation (United States Department of Health and Human Services, Protection of Human Subjects in Research (45 CFR 46), the Common Rule (45 CFR 46 Subpart A), and with the Helsinki Declaration of 1975, as revised in 2008, and has been approved by the institutional committee Columbia University Institutional Review Board.

associated with lower PDI scores in boys [b= −7.81, 95% CI: (−13.08, −2.55), p=0.004], but not girls. Maternal BMI was not associated with MDI in girls or boys, and GWG was not associated with PDI or MDI among either sex (all-p>0.05). We found that prepregnancy obesity was associated with lower PDI scores at three years in boys, but not girls. The mechanisms underlying this sex-specific association remain unclear, but due to elevated obesity exposure in urban populations, further investigation is warranted.

Keywords

pregnancy; maternal obesity; gestational weight gain; child development

INTRODUCTION

In the United States, nearly two-thirds of women of reproductive age are affected by overweight or obesity[1–3] and a majority of women gain excessive weight during pregnancy.[4–7] Maternal obesity and excessive gestational weight gain (GWG) disproportionately affect inner-city children and may detrimentally impact child development.[8–12] Early cognitive development determines, in part, a child's ability to learn and adapt, and higher childhood IQ scores are associated with higher adult IQ scores, education level, professional success, and earning potential.[13,14] The prevalence of developmental disabilities has risen in the United States, affecting 15% of children 3 to 17 years old, and disproportionately affects low-income, urban, and minority children.[15,16] Given that the brain primarily develops in utero and during early infancy, nutritional status and early life exposures are critical during this period.

Evidence suggests that maternal prepregnancy adiposity may be associated with reduced cognitive development and later cognition in children.[11,12,17–22] Additionally, pregnancy weight gain may be independently linked to developmental outcomes. Recent data show that, from six to 16 years, children of women with excessive GWG may experience negative long-term outcomes, including reduced academic achievement and deficits in executive function – a composite indicator of general cognition, social acuity, and behavioral control.[23–25] Although the precise etiology is unknown, inflammatory and hormonal mechanisms accompanying excess adipose tissue may adversely alter the placental environment leading to increased fetal brain inflammation and impaired neural circuitry related to behavior and mental health.[26–28]

Developmental milestones may be affected in a sex-specific manner by a deleterious environment in utero. Prenatal exposure to environmental stressors, such as economic hardship and environmental toxicants, alters normal fetal growth and neurocognitive function.[16,29] Responses to these exposures differ between boys and girls, as do growth, cognition, and behavior.[16,30] Male sex is an independent risk factor for adverse pregnancy outcomes and more males experience delayed growth and death during the perinatal period.[31,32] Boys grow at an accelerated rate from conception, such that they are more vulnerable to compromised nutrition status or uterine environment.[33–37] But, boys experience a slower rate of cortical development[38,39] that may increase susceptibility to detrimental prenatal

exposures, such as inflammation associated with maternal adiposity or environmental stressors. Interestingly, a nurturing home environment may remediate adverse effects of these exposures on child neurodevelopment;[29,30] previously in our cohort we observed sex-specific associations of the home environment on child development.[30] It remains unclear if sex-specific differences in neurodevelopment vary by maternal BMI and GWG, and whether a nurturing home environment mitigates these effects.

Sex-specific effects from prenatal exposures may be attributable to the divergent developmental trajectories of boys and girls.[35,36] Neurodevelopment related to maternal weight-related factors and child sex has not been examined in a low-income, urban population during early childhood. Therefore, we investigated how prepregnancy BMI and GWG influence neurodevelopment at child age three in the Columbia Center for Children's Environmental Health (CCCEH) Mothers and Newborns Study. We hypothesized that greater prepregnancy BMI, specifically maternal overweight and obesity, and GWG were negatively associated with child neurodevelopment at age three, differing by child sex.

METHODS

Design

The design of the Columbia Center for Children's Environmental Health prospective Mothers and Newborns cohort based in Northern Manhattan and South Bronx in New York City has been previously reported.[40,41] The study was originally designed to evaluate effects of prenatal exposure to adverse environmental pollutants on offspring health. Pregnant women between the ages of 18-35 years attending prenatal visits at New York Presbyterian Medical Center, Harlem Hospital, or a satellite clinic by the 20th week of pregnancy and who lived in the area for a minimum of one year were recruited between 1998 and 2006. Women were excluded if they had positive HIV status, pre-existing diabetes or hypertension, or tobacco or drug use. Although not explicitly measured, women with gestational diabetes were likely excluded from the original cohort, since this screening would have occurred prior to study enrollment (average prenatal study visit 33.2 weeks gestation).

Prenatal interviews during the second or third trimester were administered by bilingual trained interviewers to assess basic demographic, health, lifestyle, lifetime residential, and environmental information. At this visit, weight and height prior to the beginning of this pregnancy were obtained by self-report and used to calculate prepregnancy BMI. After birth, medical charts were abstracted to evaluate prenatal medical history, including total pregnancy weight gain, gestational age at delivery, and birthweight. Gestational weight gain z-scores that allow total gestational weight gain to be classified as a standardized z-score independent of gestational duration, were calculated according to the methods developed by Hutcheon et al.[42,43] Exposure to environmental pollutant chlorpyrifos (CPF), an organophosphate insecticide, was assessed in maternal blood within 24 hours postpartum using isotope-dilution gas chromatography-high-resolution mass spectrometry previously described.[44] DNA adducts of polycyclic aromatic hydrocarbons (PAH), cigarette smoke and traffic-related combustion pollutants, were analyzed in extracted white blood cell DNA using a high performance chromatography fluorescence method.[45] At follow-up visits, maternal report of breastfeeding duration was collected at 3, 6, 9, and 12 months.

At child age three years, trained bilingual research assistants administered the Psychomotor Development Index (PDI) and Mental Development Index (MDI)[46] of the Bayley Scales of Infant Development 2nd Edition (BSID-II). The BSID-II, developed and validated for children aged one to 42 months,[47,48] is sensitive to effects of toxicants (e.g. lead) on development and moderately predicts intelligence and later school performance from scores at age three years.[47,49–51] Maternal nonverbal intelligence was measured via the Test of Non-Verbal Intelligence-Third Edition (TONI-3).[52] The nature and quality of the home environment were measured by Bradley and Caldwell's Home Observation for Measurement of the Environment (HOME), a tool commonly used in neurotoxicity studies, administered in the participant homes.[51,53,54]

This study was approved by the Institutional Review Board at Columbia University. Informed consent was obtained from all participating mothers.

Statistical Analyses

All analyses were conducted using Stata (version 14.2, Stata Corp., College Station, TX, USA) with an alpha of 0.05 for statistical tests. A complete case analysis was conducted. In order to be included in the primary analytic sample, women required exposure, outcome, and covariate data. Descriptive statistics were used to compare exposures, outcomes, and covariates by sex.

Multivariable linear regression models estimated associations between maternal-weight related exposures with child MDI and PDI scores. Primary exposures were prepregnancy BMI category (underweight <18.5 kg/m^2, normal weight 18.5-24.9 kg/m^2, overweight 25.0-29.9, obesity ≥30 kg/m^2) and gestational-age standardized weight gain (GWG z-score).[42,43] Effect modification was examined by including interaction terms between prepregnancy BMI with sex and GWG. We used an alpha <0.1 to account for the lower power associated with detecting interaction effects,[6,8,55,57] although 0.1 is more conservative than 0.15 used in similar studies.[8,56,57] Based on previous literature, potential confounders included maternal IQ, ethnicity, prenatal smoke exposure, marital or cohabitation (>7 years) status, maternal demoralization score,[58] use of public assistance, education status, and age at BSID-II. The primary model covariates included maternal IQ, ethnicity, tobacco exposure, marital/cohabitation status, and age at BSID-II testing. To assess potential confounding by total HOME score, this continuous variable was added to the primary model and results were examined for beta coefficient change. We also assessed for any bias on outcomes if HOME examination occurred after the BSID-II assessment.

We conducted separate sensitivity analyses to assess whether environmental toxicant exposures, route of delivery (cesarean section), birthweight, gestational age at delivery, breastfeeding initiation, or breastfeeding duration confounded associations by evaluating change in effect size for prepregnancy BMI category or GWG z-score of ≥10%. Inconsistences in prenatal comorbidity abstraction and child anthropometric measurement practices at the three year study visit precluded examination of effects from preeclampsia or child body size on associations. Due to small cell counts (n≤5) in some of our prepregnancy BMI groups with high toxicant values, we were unable to evaluate effect modification between prepregnancy BMI and GWG with PAH or CPF.

Finally, we used inverse probability weighting to evaluate whether associations were affected by attrition or incomplete follow-up at three years, as previously described in this cohort.[6,59,60] This method estimates and corrects for bias due to missing data by applying more weight to returning participants with characteristics similar to baseline characteristics in nonreturning participants. Logistic regression models predicted probability of follow-up at age three compared to baseline data and included BMI, GWG z-score, gestational age, parity, ethnicity, child sex, maternal age, economic hardship, and public assistance status. The inverse probability of follow-up was applied as a sampling weight in the primary linear models between maternal weight characteristics and child developmental outcomes at three years.

RESULTS

Complete data on maternal weight and pertinent covariates were available for 154 African American and 228 Dominican dyads (total n=382, Fig. 1). Descriptive characteristics were similar between the original cohort (n=727) and dyads in the analytic sample (n=382); however, in the analytic sample the proportion of African American mothers was higher (40.3%) compared to the original cohort (29.0%) and Dominican mothers was lower (59.7%) compared to the original cohort (71.0%). Baseline descriptive characteristics and child measures by sex are listed in Table 1. Maternal characteristics did not differ by child sex. Based on total gestational weight gain, 13.4% of women in our sample were below, 23.3% were within, and 63.4% were above the 2009 Institute of Medicine weight gain recommendations for respective BMI categories.[61] The majority of mothers entering pregnancy overweight (77.2%) or with obesity (71.6%) gained weight above these recommendations.

At three years among all children, unadjusted mean PDI and MDI scores did not vary by maternal prepregnancy BMI category [PDI: $F(3,378)=1.53$, p=0.21; MDI: ($F(3,378)=1.30$, p=0.27)]. However, unadjusted mean scores were higher among girls compared to boys on both PDI (102.3 ± 12.0 vs. 97.2 ± 14.5, p<0.001) and MDI (92.8 ± 10.9 vs. 88.3 ± 12.0, p<0.001) (Fig. 2). In adjusted models among all children (n=382), prepregnancy obesity (BMI ≥30 kg/m^2) was associated with a 3.8-point lower PDI score at child age three (p=0.027) compared to women with prepregnancy normal weight; however, we found no relationship between prepregnancy overweight or underweight and child PDI scores. Further, we observed no associations between GWG z-score and PDI (b= −0.31, p=0.63) or MDI (b= −0.58, p=0.29).

Effect modification by sex was examined by including interaction terms between sex and 1) prepregnancy BMI category or 2) GWG z-score. For prepregnancy BMI category, we found effect modification for both PDI and MDI using an alpha <0.1.[6,8,55] Specifically, the categorical interaction term p-values were 0.08 and 0.05 respectively for prepregnancy overweight and obesity for PDI, and 0.05 among prepregnancy normal weight for MDI. No interaction was found between GWG z-score and sex (all interaction p-values >0.1). Therefore, we sex-stratified all subsequent multivariable models but included z-score as a covariate. In multivariable models for boys, compared to women with normal prepregnancy BMI, maternal obesity was associated with lower PDI scores with an effect size of almost 8

points (Table 2; Fig. 3). Prepregnancy BMI was not associated with MDI scores for boys or girls (Fig. 4), and further, GWG z-scores were not associated with MDI or PDI scores (all $p>0.05$) in sex-stratified models (Table 2). Inverse probability weighting (IPW) was utilized to assess for effects of incomplete follow-up on our sex-stratified model results. After applying IPW for successful follow-up at age three, we found that the results were essentially the same or negligibly different from primary models (data not shown).

In our analyses with the addition of HOME score to the primary sex-stratified models, we found that PDI beta coefficient for girls and MDI beta coefficients for boys and girls were essentially unchanged (data not shown). However, the addition of the HOME variable to the model attenuated the adverse effects of obesity on the PDI beta coefficient for boys of obese women from −8.8 points ($p=0.001$) to −7.8 points ($p=0.004$, ~12% change).

Over the course of the study, some children had the HOME assessment before (n=206, 53.9%) or after (n=176, 47.3%) the three year BSID-II visit. As such, we completed a sensitivity analysis removing children with HOME assessment conducted after the BSID-II visit from the primary sex-stratified models, and found that overall results were essentially unchanged. For example, for PDI among boys with HOME assessment before the three year BSID-II (n=96), the maternal obesity beta coefficient changed from −7.8 points in the primary sample to a −7.4 ($p=0.04$) in the smaller sample. In this subsample, a positive association between GWG z-score and child PDI was observed ($b= 3.7$, $p=0.033$).

In separate sensitivity analyses, we added adjustments to the primary sex-stratified multivariate models for birthweight, gestational age at delivery, if an infant ever breastfed, duration of breastfeeding to one year, route of delivery (cesarean section), and prenatal CPF or PAH exposure to examine for potential confounding between these factors and maternal weight-related exposures. We did not observe confounding (β change <10%) on the relationship between maternal weight-related exposures and child PDI or MDI scores by most of these factors (data not shown); however, in our models with PDI outcome in boys, inclusion of both birthweight and gestational age in a subset of participants (n=167) strengthened estimated associations between boys' PDI scores and maternal prepregnancy obesity category ($b= −9.1$, $p=0.001$, 15.6% change from primary model).

DISCUSSION

We found evidence for sex-specific associations between child psychomotor development at age three and maternal prepregnancy BMI, but not gestational weight gain. In covariate adjusted models in all children, maternal obesity was associated with a decrease in psychomotor development, while maternal weight-related measures were not associated with child mental development index scores.

Our study is the first to examine the effects of maternal BMI on neurodevelopmental differences between sexes or in urban, minority, low-income children in early life. Due to previously reported sex-specific effects of adverse prenatal exposures during later childhood, [30] we evaluated for and observed effect modification by child sex. In our primary models, a 7.8-point deficit in psychomotor development was seen in boys, but not girls, indicating that

the relationship between maternal obesity and PDI scores in the overall sample was driven by the strong association between maternal obesity and boys' development. Beyond our primary models, we conducted several sensitivity analyses that were mostly consistent with our primary findings. However, we found that additional adjustment for birthweight and gestational age at delivery in a subset of participants strengthened the primary effect in boys by >10%, suggesting positive confounding by these factors.[62] Though inclusion of the HOME score slightly mediated the primary effect, an 8-unit lower score is striking and may have public health implications for boys' long-term achievement and wellbeing at the individual and population level. Indeed, ongoing examination of children in this cohort at age 7 using the Wechsler Intelligence Scale for Children IV revealed a continuance of this sex-specific neurodevelopmental deficit (Widen *et al.*, unpublished data). Maternal obesity and overweight were associated with lower full-scale intelligence quotient, perceptual reasoning, processing speed (overweight only), and verbal comprehension (obesity only) scores in boys, but not girls (Widen *et al.*, unpublished data).

Epidemiologic evidence suggests maternal obesity is associated with increased odds of offspring cognitive deficits or developmental delays.[8,11,19,63,64] Maternal obesity and excessive gestational weight gain are both associated with autism spectrum disorder,[64–66] but studies that investigated motor development have been inconsistent and are limited in early life (age <5y). In the Upstate KIDS study in New York (n=4901 dyads), Wylie *el al.* used maternal questionnaires to investigate the effect of prepregnancy BMI on motor development measured by time required for infants to acquire gross motor milestones between four and 24 months.[67] Despite a predominantly white (83.3%), well-educated (81.1% ≥ some college) cohort quite different from the CCCEH, findings were consistent with our results: Maternal obesity was associated with a slight delay in infant ability to sit and crawl.

Contrary to our findings, in the nationally representative US Department of Education's Early Childhood Longitudinal Study-Birth Cohort (ECLS-B, n=4750), Hinkle and colleagues showed strong associations between MDI and prepregnancy overweight and obesity, but no association between PDI and prepregnancy BMI in children at 2 years.[8] Interestingly, at kindergarten age in the same cohort, children of mothers with severe prepregnancy obesity (class II and III, BMI ≥35 kg/m^2) had a higher prevalence of low fine and gross motor function ($p<0.001$) according to the BSID-II,[9] suggesting a multifactorial influence on early development that begins in pregnancy and extends beyond the postnatal period. Conversely, in 355 mother-child dyads, Neggers *et al.* observed associations between maternal obesity with diminished IQ and nonverbal abilities, but not motor development, in children at 5 years.[63]

The mothers in these two cohorts were quite different from our sample of urban, minority mothers, which may partly explain these differential findings. The ECLS-B had fewer mothers with prepregnancy obesity (14.8%) than our sample (23.0%), while mothers in the ECLS-B with prepregnancy BMI ≥35 (6.2%) were generally of lower socioeconomic status than normal weight counterparts, with fewer years of education, more likely to live below 130% of the federal poverty threshold, and more likely to be white (54.3%). Children in Neggers' study were bom to low-income, African American mothers in Birmingham,

Alabama and were exposed to a lower rate of prepregnancy overweight (14.4% vs. 24.1% in our sample), higher rate of prepregnancy obesity (39.9% vs. 23.0% in our sample), and a lower HOME score (37.5 ± 6.9) compared to our sample (39.5 ± 6.2). The equivocal nature of these findings indicates that early development requires further investigation to identify the influences of gestational weight corollaries on early development.

To our knowledge, the sex-specific differences in motor development among children born to mothers with obesity observed in our study have not been previously reported in this age group from a similar population. Interestingly, boys in our study appeared more vulnerable than girls to an in-utero environment affected by excess maternal adiposity. The physiological mechanisms linking maternal adiposity, gestational weight gain, and early cognition are intricate and likely differ between boys and girls. Potential mechanisms associated with excess adiposity may produce an inimical prenatal environment including dysregulation of lipid, insulin and appetite or increased estrogen and inflammatory signaling;[26,68–70] specific dietary patterns, including high fat diet, excess free fatty acids or glucose, or micronutrient deficiencies;[20,26,71] altered placental transport mechanisms;[68,70,72] a lipotoxic environment for brain development;[73,74] excessive production of reactive oxygen species and reduction in placental ATP generation;[75–77] or alterations in serotonergic and dopaminergic signaling pathways.[20]

Fetal growth differs between sexes in rate and efficiency of the placental response to maternal adiposity;[37] boys grow faster, larger, require more placental efficiency to grow at this accelerated rate,[34,36,81] and male placentas are more vulnerable to placental inefficiency from inflammatory, oxidative, and nitrative stress from maternal adiposity.[75,76] The placenta is the interface of maternal-fetal nutrient exchange and its contributions to sexual dimorphism in the origins of health and disease have been well documented.[37,82,83] The sex-specific relationship between excess adiposity and placental abnormalities in key regulatory biochemical and molecular mechanisms of growth and development is currently under investigation.[76,77,84–88] Placental hypertrophy is more common among women with greater adiposity[86] and some evidence points to alterations in placental nutrient exchange in obese and overweight women.[89,90] Due to amplified growth, males in utero may be limited by a lack of reserve placental capacity and nutrient exchange during critical phases of growth, making developmental disparities more pronounced.[34,35] As a counterpart to placental efficiency, suboptimal maternal dietary intake may limit availability of nutrients at critical phases.[37] Dietary aberrations associated with overweight and obesity, such as high-fat diet or limited nutrient intake,[20,26,71] may adversely alter the placental milieu and potentially produce augmented adverse effects in boys. However, it remains unclear why only maternal obesity was associated with considerably lower psychomotor development scores in boys at age three.

This study has several limitations. These results may not be fully generalizable to the current US population of women of childbearing age, since our population was specific to low-income African American and Dominican women in an urban region of the US. Despite important roles in development, we were not able to account for paternal characteristics, rate or pattern of weight gain, placental size, or prenatal comorbidities (e.g. preeclampsia) as these were not routinely measured per the original study design. Although we were not able

to account for paternal characteristics, evidence indicates that preconception paternal stressors, including nutrition status, metabolic dysregulation, psychosocial distress, and environmental contaminants, transform the preconception paternal epigenome and are reflected in the germline and offspring phenotype.[91–95] Intergenerational transmission of the preconception environment may play a fundamental role in neurodevelopment[95–97] and should be examined in future investigations. Additionally, breastfeeding data were limited and breastfeeding continuation was not recorded after child age one. Further, small cell sizes hindered any investigation of differences between extreme BMI classes and child development. These characteristics may be linked to adverse outcomes and are important to consider in future work. Likewise, offspring body size may be on the causal pathway to adverse neurodevelopmental outcomes[96,97] and is strongly correlated with maternal BMI and GWG;[98] however, we were unable to examine for effects of child body size on associations at this age. There was potential for selection bias due to inclusion restrictions in our analyses; to examine for this potential bias, we conducted several sensitivity analyses, including inverse probability weighting, and the results did not change meaningfully. Finally, although developed specifically for this age group (1-42 months), the BSID-II measures are not direct measures of cognition or the richness of development, and an updated assessment of mental development in the third edition of the BSID became available after this study.[98]

Despite these limitations, our results are similar to some reports examining the association of adiposity with developmental and academic outcomes in childhood, although in different populations.[63,67] Strengths of this work include investigation of a diverse, low-income urban population with robust measures. We were able to examine critical cognitive outcomes, psychomotor development and mental development, with a relatively large sample size and with adjustment for a range of confounders, including maternal IQ, influence of a nurturing home environment, and toxicant exposure.

In our low-income, minority cohort, prepregnancy obesity was associated with lower PDI scores at three years only in boys. These sex-specific differences in psychomotor development in an understudied population are interesting, but the mechanisms underlying this association remain unclear. Our results are consistent with previous analyses in that prepregnancy BMI affected early child development, although in different populations and ages. Due to the elevated obesity exposure in women entering pregnancy, particularly in low-income women, further investigation to disentangle modifiable elements and inform practical guidelines and interventions is warranted.

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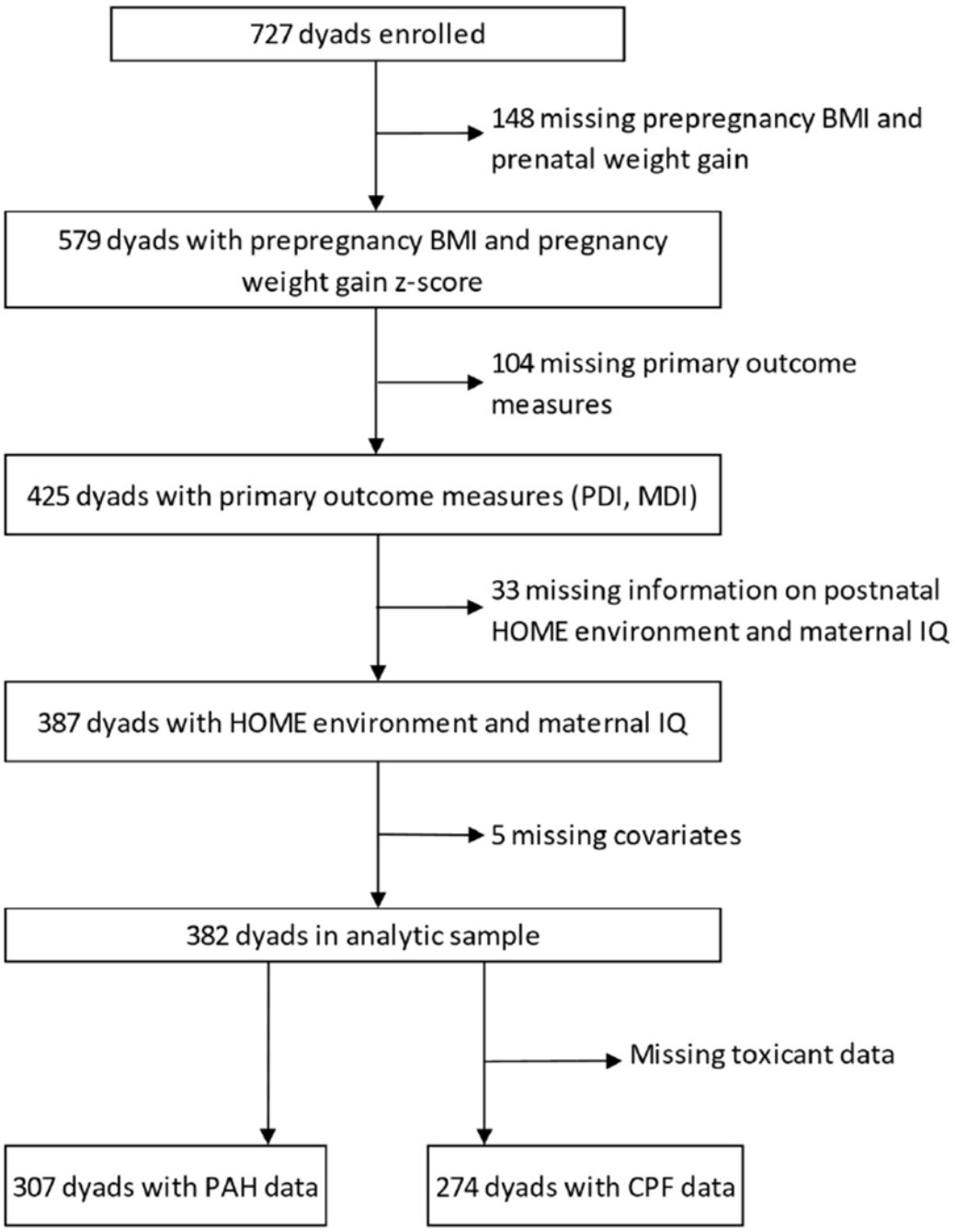

Figure 1.
Participant flow diagram

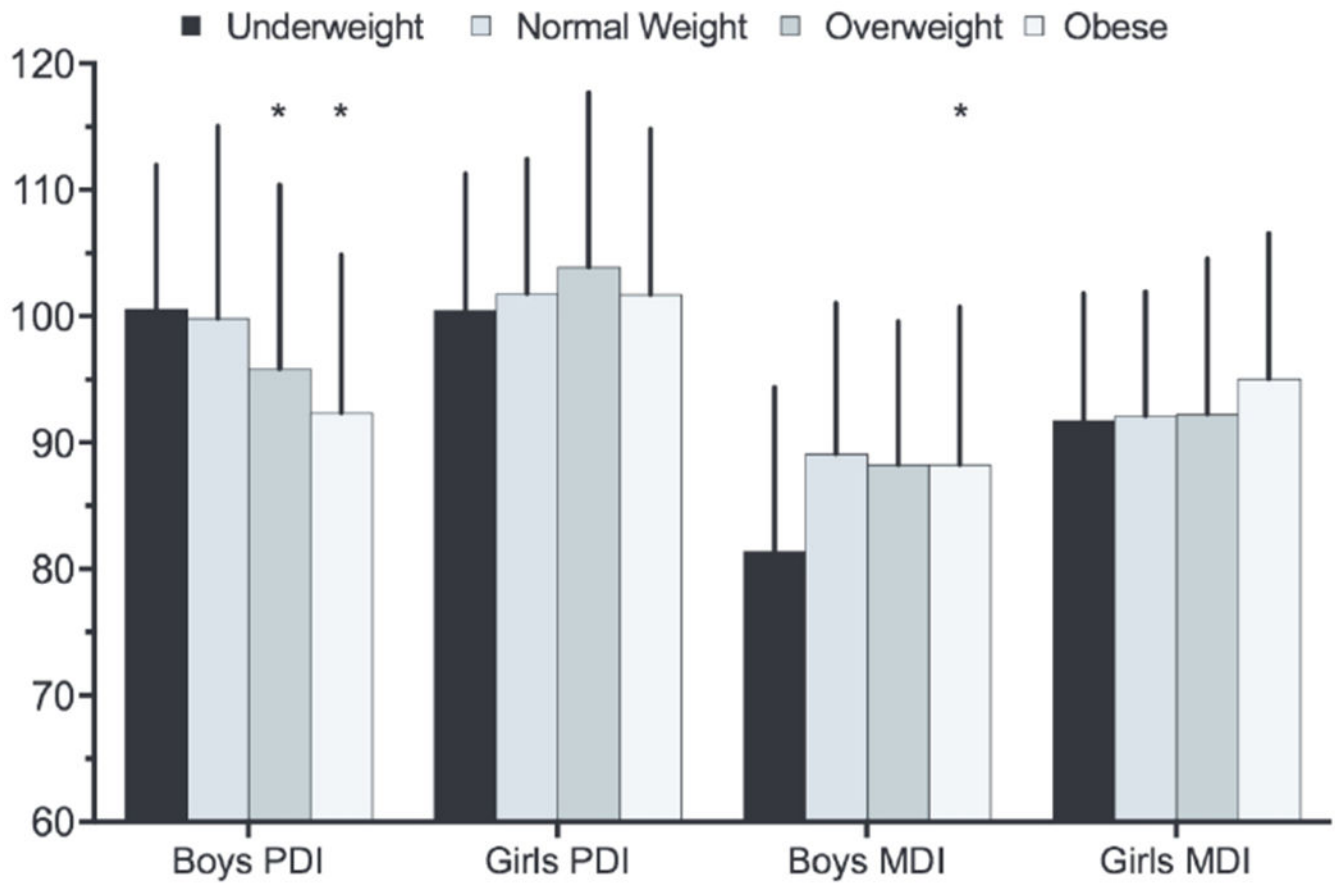

Figure 2.

Unadjusted mean PDI and MDI scores (±SD) by child sex (n=382) at age 3 PDI psychomotor development index; MDI mental development index

*difference between sexes by BMI p<0.01

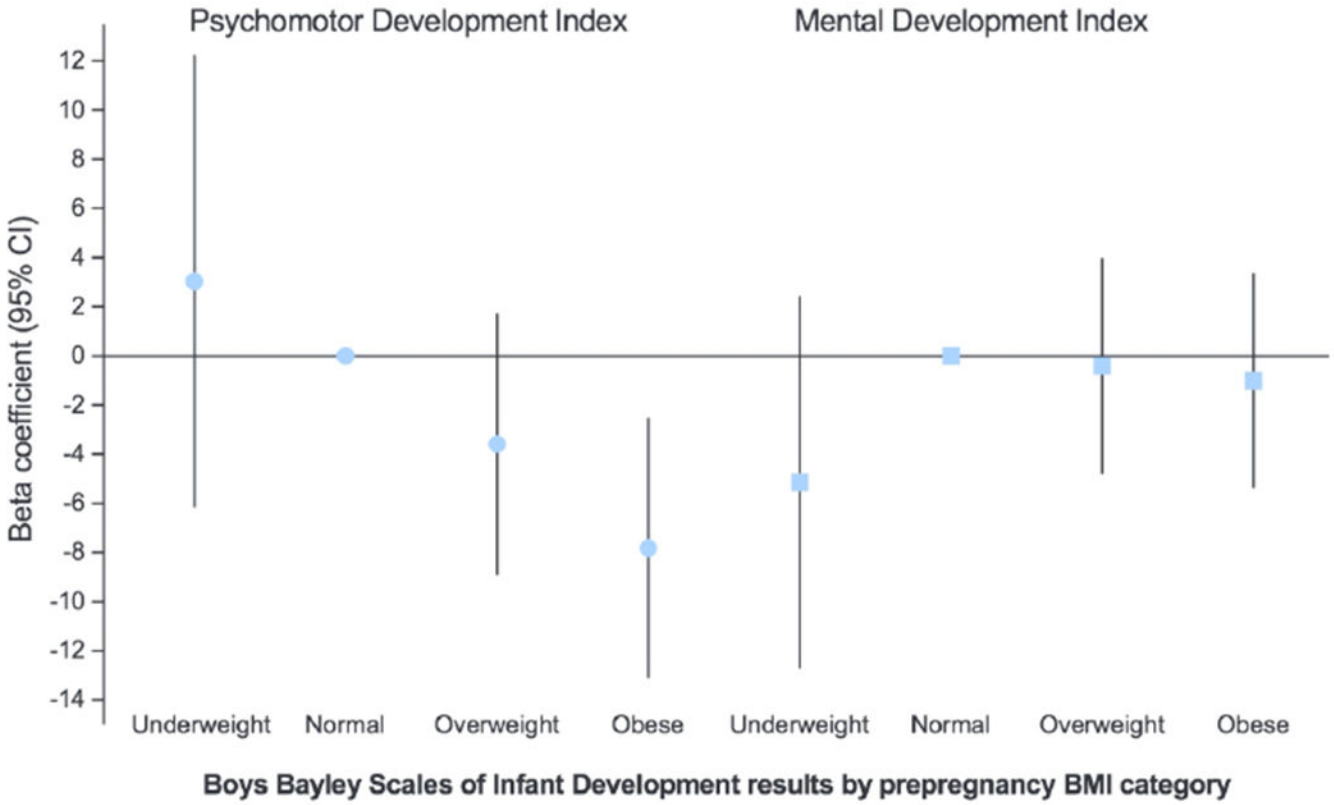

Figure 3.
Bayley Scales of Infant Development results for boys by prepregnancy BMI category (n=170)

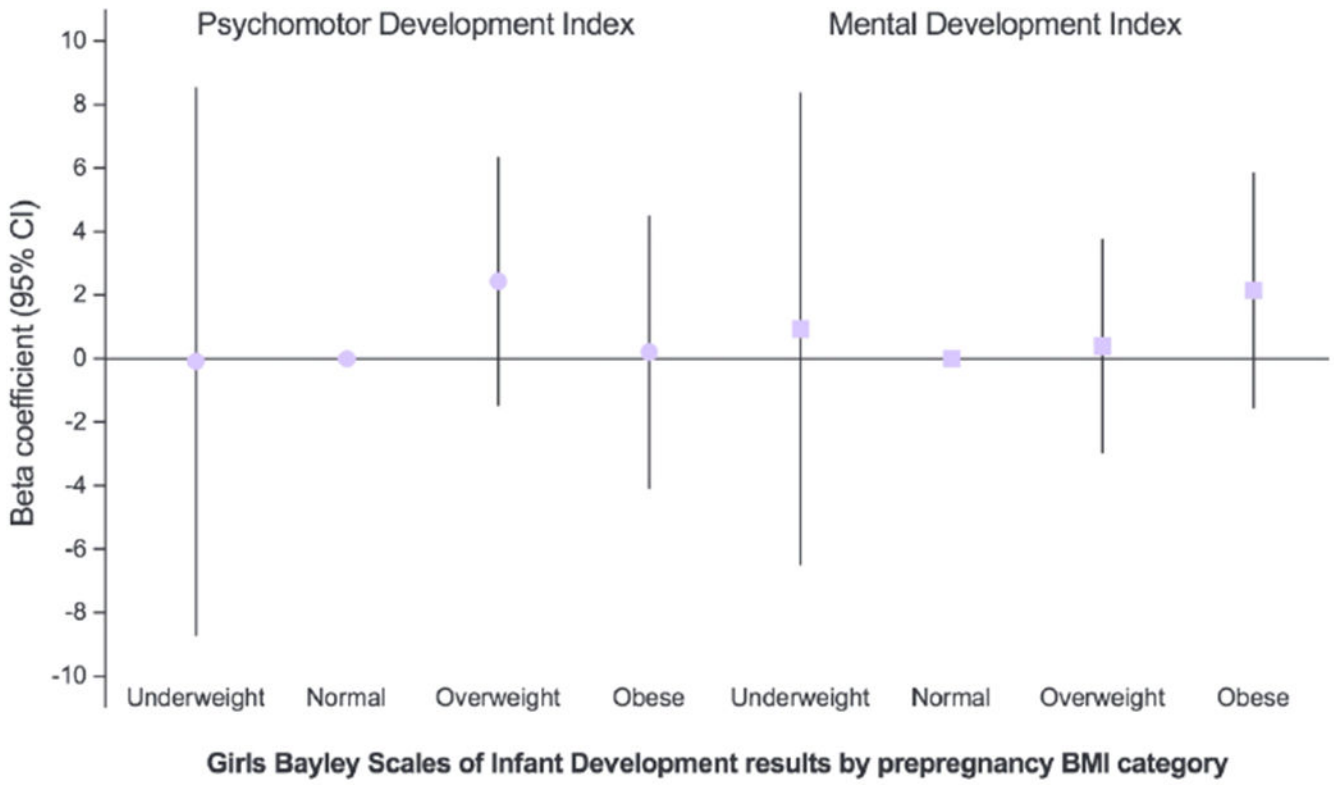

Figure 4.
Bayley Scales of Infant Development results for girls by prepregnancy BMI category (n=212)

Table 1.

Participant demographics and outcome values by child sex (n=382)

	All (n=382)	Boys (n=170)	Girls (n=212)	p-value
Maternal				
Prepregnancy BMI Category, %				0.53
Underweight	4.7	5.9	3.8	
Normal	48.2	48.2	48.1	
Overweight	24.1	21.8	25.9	
Obese	23.0	24.1	22.2	
Dominican Ethnicity, %	59.7	58.8	60.4	0.76
Maternal Education < High School, %	34.2	29.3	38.2	0.07
Receipt of public assistance[a], %	40.9	39.6	41.9	0.66
Never Married, %	67.8	65.9	69.3	0.47
HOME Score[b]	39.5 ± 6.2	39.0 ± 6.5	39.8 ± 5.9	0.17
GWG Z-score[c]	0.14 ± 1.02	0.24 ± 0.93	0.05 ± 1.08	0.06
Maternal IQ score	85.5 ± 13.5	85.0 ± 13.5	86.0 ± 13.6	0.46
Detectable PAH[d], n (%)	121 (39.4)	51 (37.5)	70 (40.9)	0.54
High chlorpyrifos[e] (>6.17 pg/g), n (%)	39 (14.2)	21 (17.5)	18 (11.7)	0.17
Child				
Gestational age at delivery, weeks	39.4 ± 1.3	39.4 ± 1.3	39.3 ± 1.2	0.84
Birthweight[f], g	3367.5 ± 469	3416.6 ± 490	3328.7 ± 448	0.07
Ever breastfed[g], n (%)	255 (69.9)	128 (77.1)	127 (63.8)	<0.01
Breastfeeding duration[g], weeks	14.5 ± 14.1	13.7 ± 13.4	15.3 ± 14.8	0.36
Age at 3y Bayley test, months	36.7 ± 2.6	36.7 ± 1.9	36.7 ± 3.0	0.99
Psychomotor Development Index score	100.0 ± 13.4	97.2 ± 14.5	102.3 ± 12.0	<0.001
Mental Development Index score	90.8 ± 11.6	88.3 ± 12.0	92.8 ± 10.9	<0.001

Values are means ± SD or percentages.

[a]Data available on 169 boys and 210 girls;

[b]HOME, Home Observation for Measurement of the Environment;

[c]GWG, Gestational weight gain;

[d-g]Data available on:

[d]136 boys and 171 girls;

[e]120 boys and 154 girls;

[f]167 boys and 211 girls;

[g]166 boys and 199 girls.

Table 2.

Adjusted sex-specific associations between maternal prepregnancy BMI, pregnancy weight gain and child cognitive test scores at age 3, Columbia Center for Children's Environmental Health enrolled from 1998 to 2006 (n=382).

	Psychomotor Development Index (PDI)		**Mental Development Index[46]**	
	β (95% CI)	**p-value**	**β (95% CI)**	**p-value**
Boys (n=170)				
Prepregnancy BMI (n)				
Underweight (10)	3.0 (−6.1,12.2)	0.51	−5.1 (−12.7,2.4)	0.18
Normal Weight (82)	Referent		Referent	
Overweight (37)	−3.6 (−8.9,1.7)	0.18	−0.4 (−4.8,4.0)	0.86
Obese (41)	−7.8 (−13.1,−2.5)	<0.005	−1.0 (−5.3,3.3)	0.65
GWG Z-score (170)	−0.1 (−2.4,2.2)	0.94	−0.1 (−2.0,2.4)	0.93
Girls (n=212)				
Prepregnancy BMI (n)				
Underweight (8)	−0.1 (−8.7,8.5)	0.98	−0.9 (−6.5,8.4)	0.80
Normal Weight (102)	Referent		Referent	
Overweight (55)	2.4 (−1.5,6.3)	0.22	0.4 (−3.0,3.7)	0.82
Obese (47)	0.2 (−4.1,4.5)	0.92	2.2 (−1.5,5.8)	0.25
GWG Z-score (212)	−0.5 (−2.0,1.0)	0.51	−0.8 (−2.1,0.5)	0.21

Results are estimated β-coefficients for PDI and MDI scores from multivariate linear regression models, controlling for covariates. Normal weight prepregnancy BMI is the reference group. The adjustment set included maternal race/ethnicity, marital/cohabitation status, HOME score, maternal IQ, home smoking status, and child Bayley age.