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Original Research

Postpartum Obesity Is Associated With Increases in Child Adiposity in Midchildhood in a Cohort of Black and Dominican Youth

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A B S T R A C T

Background: Obesity disproportionately affects marginalized and low-income populations. Birth parent obesity from the prenatal period and childhood has been associated with child obesity. It is unknown whether prenatal or postnatal birth parent obesity has differential effects on subsequent changes in adiposity and metabolic health in children.

Objectives: We evaluated how birth parent obesity 7 y after delivery was associated with child body composition changes and cardiometabolic health in midchildhood and further assessed the influence of the perinatal and postpartum period on associations.

Methods: Black and Dominican pregnant individuals were enrolled, and dyads ($n = 319$) were followed up at child age 7 and 9 y. Measures included, height, weight, waist circumference (WC), and percent body fat (BF%). Multiple linear regression was used to relate postpartum weight status with child outcomes accounting for attrition, and a series of secondary analyses were conducted with additional adjustment for perinatal weight status, gestational weight gain (GWG), and/or long-term weight retention to evaluate how these factors influenced associations.

Results: Almost one-quarter (23%) of birth parents and 24.1% children were classified with obesity at child age 7 y, while at 9 y, 30% of children had obesity. Birth parent obesity at child age 7 y was associated with greater changes, from ages 7 to 9 y, in child BMI *z*-score (β: 0.13; 95% CI: 0.02, 0.24) and BF% (β: 1.15; 95% CI: 0.22, 2.09) but not obesity at age 9 y. All observed associations crossed the null after additional adjustment for prenatal factors.

Conclusions: Birth parent obesity at 7-y postpartum is associated with greater gains in child BMI *z*-score and BF% in midchildhood. These associations diminish after accounting for prenatal size, suggesting a lasting impact of the perinatal environment and that interventions supporting families from the prenatal period through childhood are needed.

Keywords: pediatric obesity, child health, maternal obesity, obesity, cardiometabolic risk factors, child, growth

Introduction

Obesity, defined in adults as BMI of ≥30 kg/m^2 and children as a BMI/age of ≥95th percentile [1], disproportionately affects marginalized and low-income populations [2]. Obesity prevalence varies greatly by race and ethnicity, with Hispanic adults and non-Hispanic Black adults having a higher prevalence of obesity than non-Hispanic White or Asian Americans, and similar patterns occurring in children [3–5]. Childhood obesity in the United States has more than tripled since the 1970s [6]

Abbreviations: BF%, body fat percentage; GWG, gestational weight gain; GWGz, gestational weight gain *z*-score; WC, waist circumference.

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with adults following similar trends [7]. Children who develop obesity are more likely to experience persistent adverse health complications such as metabolic syndrome, hypertension, prediabetes, and diabetes [8], throughout childhood and into adulthood, and have an increased likelihood of obesity into adulthood [3,6,9].

Research has consistently shown associations between preconception and prenatal health with child health outcomes [10]. For example, prepregnancy obesity increases risk for child metabolic syndrome or obesity at 4 and 11 y [11,12]. Gestational weight gain (GWG) above the recommended guidelines also increases risk of childhood obesity and overweight, even among individuals with a healthy prepregnancy BMI value [13–15]. A proposed mechanism for this increased risk is developmental plasticity or fetal programming where the intrauterine environment affects health outcomes during life [16]. Children of a biological parent with obesity, either birth parent or other biological parent, have more than double risk of obesity than those who do not have a parent with obesity [17]. There is evidence showing correlations between birth parent and child body size [18,19]. However, less is known about the role of birth parent weight status after the perinatal period, such as in midchildhood, with subsequent child body size and obesity risk, with few studies examining the prospective role of birth parent obesity during the offspring's childhood on subsequent growth and metabolic indicators experienced by the child [20] and none focused on a marginalized population at risk of health disparities. Moreover, it is unclear whether the birth parent's obesity status during the prenatal period or during the childhood of the offspring has a stronger association with child growth and health outcomes. Few studies to date have examined both prenatal and postpartum birth parent health in a multiethnic population in relation to child obesity and metabolic health. Previous cross-sectional work has shown a link between concurrently measured parental health behaviors and child obesity risk, suggesting that parents may strongly influence children's environments, including dietary quality and physical activity [21], thereby influencing later child obesity risk and related health outcomes [18,22]. Parents may want to eat healthy and participate in physical activity but not have the means or time to do these activities. A growing body of evidence has demonstrated that social determinants of health affect both parent and child obesity [23,24]. Marginalized communities such as the one in the Columbia Center for Children's Environmental Health (CCCEH or Children's Center) Mothers and Newborns prospective birth cohort often do not have equitable access to care, healthy food, or a built environment conducive to physical activity [23,25,26].

Additional studies are needed to disentangle the drivers of obesity in childhood, including differences in the roles of prenatal factors compared with those of postnatal factors. It is unknown whether the prenatal environment (fetal programming) or birth parent postpartum health has a stronger impact on the development of offspring obesity and cardiometabolic risk factors (Figure 1). Programs have focused on the prenatal period to demonstrate reduction in obesity risk, but there might be a need to continue programs throughout the early life course to continue to reduce risk of obesity among children. Among a unique cohort of Black and Dominican birth parent–child dyads living in New York City, we examined the longitudinal relationship between birth parent obesity and weight changes in midchildhood with subsequent child growth, body composition, and cardiometabolic health indicators. We conducted additional analyses examining for differences by prenatal (prepregnancy BMI or GWG) or postnatal factors (7-y-postpartum weight retention) to assess whether these factors impacted associations. We hypothesized that birth parent obesity and birth parent weight changes when the child is in midchildhood would be positively associated with our primary outcome of child obesity at age 9 y, as well as secondary outcomes of changes in child body size, body fat percentage (BF%), waist circumference (WC), and cardiometabolic risk profiles. We further hypothesized that the birth parent body size measures more proximal to midchildhood would more strongly influence child weight and metabolic risk than measures from the prenatal period.

Methods

Study population

This secondary analysis was conducted with data from the CCCEH Mothers and Newborns prospective birth cohort study, previously described [9,27]. In brief, individuals who self-identified as Black or Dominican and reported residence in either Northern Manhattan (Washington Heights or Central Harlem) or the South Bronx, New York, for ≥1 y were enrolled in the study from 1998 to 2006 ($N = 727$). Enrollment was limited to pregnant people who had no self-reported history of diabetes, hypertension, illicit drug use, smoking, or known HIV; received prenatal care before 20 wk gestation; were aged 18 to 35 y; and were in their third trimester of pregnancy. All enrolled adult individuals provided written informed consent. Infants were automatically enrolled in the study at birth, although children after age 7 y provided written assent. All study procedures and questionnaires were approved by the Columbia University Institutional Review Board.

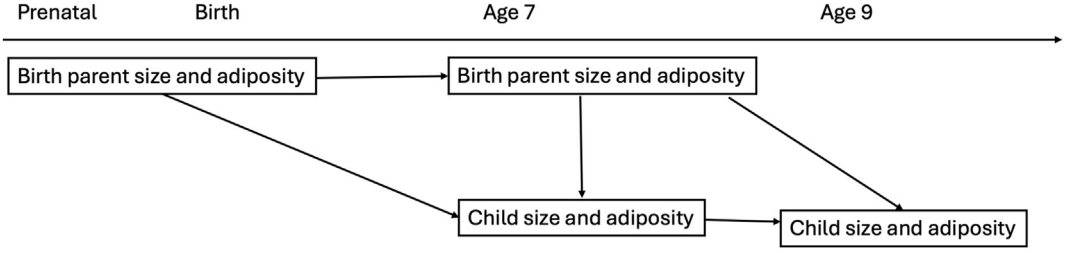

FIGURE 1. Conceptual framework. CCCEH, Columbia Center for Children's Environmental Health.

Measures

Prepregnancy weight was self-reported during the prenatal third trimester visit. After delivery, prenatal weights were abstracted from medical charts. Gestational weight gain *z*-scores (GWGz) were calculated using the method established by Hutcheon et al. [28], which allows for examining effects of GWG independent of gestational duration. Birth parent height (in centimeters) was self-reported during pregnancy and measured at the postpartum, 7- or 9-y, visit with a stadiometer (before January 2010: Cardinal Scale, Webb City, Missouri; after January 2010: Holtain, Crymych, United Kingdom). Data cleaning for birth parent height has previously been described [29]. When the child was at ages 7 and 9 y, birth parent's weight (in kilograms) was measured with the Tanita Digital Body Mass Indicator Scale BC-418 (Tanita Corp of America, Arlington Heights, Illinois). Height and weight measurements were used to calculate birth parent 7- and 9-y-postpartum BMI (in kg/m^2). Birth parent weight retention was calculated by subtracting self-reported prepregnancy weight from the weight measured at the child age 7-y visit. Birth parent obesity was defined as a BMI $\geq$ 30 kg/m^2 per Center for Disease Control guidelines and previously used in this population [30,31].

Demographic information—such as race and ethnicity, receipt of public assistance, marital status, challenges in afford food, number of pregnancies, and total number of people in a household—were self-reported at the initial prenatal visit as well as follow-up visits. Affording food and public assistance updates were assessed via self-report during postnatal follow-up visits.

Child anthropometrics were taken by trained research assistants. Child WC measurements were obtained using a nonstretchable measuring tape halfway between the lower rib margin and iliac crest. WC *z*-scores were calculated using the LMS (Lambda Mu and Sigma Method) tables for children aged 5–19 y from NHANES III [32]. LMS is commonly used for standardizing growth measures among children to obtain age-specific percentiles or Z-scores by smoothing three uncorrelated curves: L (skewness of distribution at each age), M (median at each age) and the S (coefficient of variation at each age) [33,34]. Child height was measured by stadiometer (before January 2010: Detecto Cardinal 750 scale/stadiometer; Cardinal Scale; after January 2010: Holtain-Harpenden Wall Mounted Stadiometer Counter 602VR; Holtain Limited). Child weight was measured using the Tanita Digital Body Mass Indicator Scale BC-148, with children wearing light clothing and no shoes. Height and weight measurements were used to calculate age-specific and sex-specific child BMI (kg/m^2) and BMI *z*-scores (BMIz). A child BMI between the 85th and 95th percentile was classified as overweight and a child BMI at or above the 95th percentile as obesity per Centers for Disease Control and Prevention guidelines [35].

BF% was obtained using bioelectrical impedance anaylsis from the Tanita Digital Body Mass Indicator Scale BC-148. A study phlebotomist collected fasting blood samples 2 wk before an additional study visit, which occurred between 8.5 and 12 y of age (mean: 9.5 $\pm$ 1.2 y) [10]. Samples were transferred on ice to the Biomarkers Core Laboratory at the Irving Institute at Columbia University. The Immulite 1000 (Siemens Healthcare Diagnostics, Munich, Germany) was used to measure insulin levels (in microinternational units per milliliter), and the Cobas Integra 400 Plus (Roche Diagnostics, Basel, Switzerland) was used to measure glucose, triglycerides, HDL cholesterol, and LDL cholesterol (all expressed in mg/dL).

Statistical analysis

Statistical analyses were completed using Stata 14.0 (College Station, Texas), with an α of 0.05 for all models. Descriptive statistics were conducted for baseline data. Characteristics were compared between those included and excluded from the sample using *t* tests and χ^2 tests. Child outcome measures for this analysis included child risk of obesity at age 9 y and change from 7 to 9 y in the following: *1*) BMIz, *2*) WC *z*-score, and *3*) BF%. Additional child measures included cardiometabolic risk clustering groups derived from measures at the metabolic visit, previously reported in this sample [10]. The change from 7 to 9 y was intended as a measure to capture change over a period instead of a cross-sectional snapshot and allowed for testing direction of associations. Cardiometabolic risk factor patterns (or risk groups) were developed previously for our data using cluster analysis of HDL and LDL cholesterol, triglycerides, HOMA-IR, and fasting glucose using the package mclust in R [10].

Multivariable linear regression and Poisson regression models with robust variance estimators were constructed to examine the association of birth parent 7-y-postpartum obesity or birth parent weight changes from 7 to 9 y postpartum on child outcomes, including changes in child BMIz, BF%, and WC *z*-score from ages 7 to 9 y, as well as risk for obesity at child age ~9 y. Multinomial logistic regression models were created to examine associations of birth parent 7-y-postpartum obesity or birth parent weight change from 7 to 9 y postpartum on child outcomes with cardiometabolic outcomes—based on risk clustering at child age 9 y described earlier (Supplemental Figure 1) [10]. Measurement changes from 7 to 9 y were chosen to assess growth for 2 reasons: *1*) 7 y is the first parent measurement after birth of the index child; *2*) 9 y is in the prepubertal window for most of the children. During puberty, there is an expected acceleration of change of growth and body composition that could skew the results. All birth parents were not measured at child age 9 y, and 29 were missing measurements of weight changes; thus the examinations with birth parent weight changes and child outcomes had smaller sample sizes.

Covariates included in the models were selected based on previous literature and included the following: birth parent age (years), race (Dominican or Black), child sex (female/male), child age at the 7-y visit (years), child age at the 9-y visit (years), ability to afford food (yes/no), receipt of public assistance (yes/no), parental relationship status (single, divorced, married/widowed, or cohabitating for $\geq$7 y), parity (number of children), and total number of people living in the household.

A series of sensitivity analyses were conducted to determine whether prenatal or postpartum weight influenced observed associations between birth parent size at child age 7 y with child adiposity and metabolic health outcomes; analyses were conducted to evaluate whether inclusion of prepregnancy BMI, GWGz, or postpartum weight retention in our models changed the β coefficient by $\geq$10% for our primary exposures: *1*) 7-y postpartum obesity or *2*) birth parent weight change from 7 to 9 y postpartum. In addition, 7-y-postpartum obesity and weight retention were evaluated as different exposures as they reflect different aspects of weight-related health. For example, a person who started pregnancy with obesity could continue with obesity

without weight retention, that is, return to baseline weight, or a person who was low normal weight could have significant weight retention but still maintain a healthy category for BMI. To compare effects of the prenatal with those of midchildhood environment, we also present associations between prepregnancy BMI and prepregnancy obesity with child outcomes that do not included adjustment for the 7-y birth parent obesity measure.

A series of sensitivity analyses were conducted, including evaluation of sex differences, selection bias, and exclusion of children with obesity at age 7 y. Sensitivity analyses were conducted to examine for sex-specific effects of birth parent exposures on child growth and adiposity, by including interaction terms with sex in our models. We also conducted an additional sensitivity analysis assessing effects of excluding children who were classified with obesity at 7 y, in our analyses examining effects of birth parent exposures on child obesity risk at age 9 y as these children already had the outcome of interest. For cardiometabolic clusters, sensitivity analyses were created excluding those children who had metabolic visits before age 9 y. Finally, all models were fit with inverse probability sample weighting to account for whether associations were affected by attrition or incomplete follow-up at child age 7 y, as previously described in this cohort [29,31,36,37]. Inverse probability weighting estimates and corrects for bias due to missing data by applying more weight to participants included in the final sample who have similar characteristics to participants not included in the sample due to incomplete or missing data. Logistic regression models predicted probability of follow-up at age 7 y compared with baseline data and included birth parent age, birth parent height, marital status, ability to afford food, public assistance status, race, parity, and prepregnancy weight.

Results

Parent and household characteristics

Of the 697 participants with baseline data, 319 dyads were included in the analytic sample (Figure 2). Birth parent characteristics are summarized in Table 1. Fewer than 25% of birth parents were classified with obesity before pregnancy; but by 7-y postpartum, over 40% of birth parents had obesity. Over half of the families received public assistance throughout the study; however, few families reported difficulty affording food. Other than being younger (24.7 compared with 25.8; $P = 0.008$) and taller (161.7 compared with 162.7; $P = 0.04$), there were no differences in those included in the analytic sample compared with those not included (Supplemental Table 1). Child characteristics are summarized in Table 2. Between the 7 and 9 y visit, BF%, BMI and WC *z*-scores, and obesity prevalence increased. At the metabolic visit, a separate visit occurring between child ages 8.5 and 12 y, mean child biomarkers were within normal limits. Almost a third (30%) of children had abnormal HDL concentrations, followed by insulin (25%), LDL (24%), triglycerides (17%), and glucose (8%) at this study visit.

Birth parent obesity at child age 7 y: associations with child growth and body composition from ages 7 to 9 y

Multivariable linear regression models were used to examine the relationship between birth parent obesity when child was age 7 y with changes in child growth and body composition measures from age 7 to 9 y (Table 3). In unadjusted multivariable regression models (Table 3), having a birth parent with obesity at child age 7 y was associated with a 49% increased risk of obesity in the child at age 9 y as well as higher gains in all growth and body composition measurements, than those children with birth parents without obesity. After implementing inverse probability weighting to reduce the effects of attrition, the associations for our primary outcome, child obesity at age 9, and for WC *z*-score crossed through the null, indicating that the association was no longer significant, while the associations for birth parent obesity with child BMIz and body fat changes remained. Specifically, birth parent obesity was associated with 0.13 increase in BMIz from child age 7 to 9 y and a 1.15% increase in BF%.

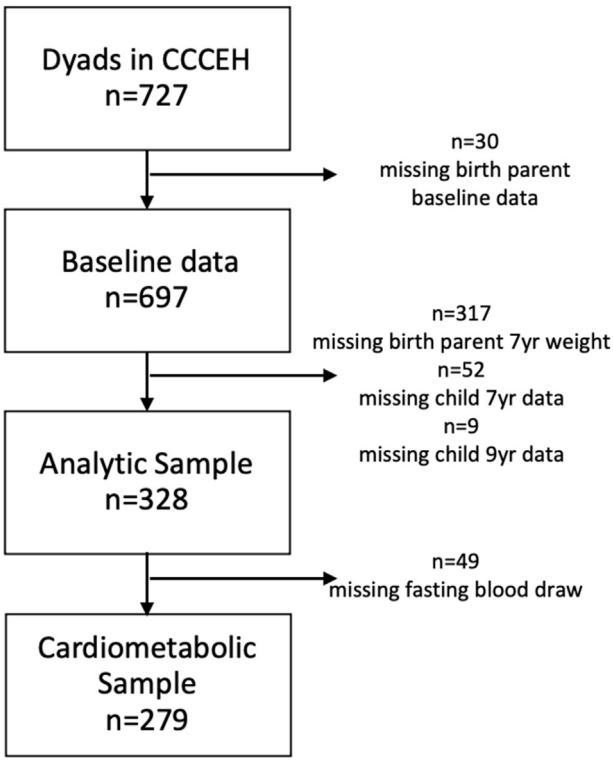

FIGURE 2. Flow diagram.

Birth parent weight changes from 7 to 9 y postpartum

Associations with child growth and body composition

In the subset of the sample who had birth parent weight change data available, no association were observed between birth parent weight changes from child age 7 to 9 y with concurrent changes in child growth and body composition from age 7 to 9 y and child obesity at age 9 (Table 3).

Associations between birth parent obesity and weight status with child cardiometabolic profile

Birth parent obesity at 7-y postpartum was not associated with cardiometabolic cluster risk groups (Supplemental Table 2), whereas maternal weight changes from child age 7 to 9 were associated with a 12% increased risk in belonging to the moderate risk group showing elevated glucose and moderately low

TABLE 1
Birth parent characteristics at baseline and the 7-y postpartum follow-up visit: CCCEH[1]

	All CCCEH women with baseline data ($N = 697$[2])	Analytic sample characteristics ($n = 319$)	
		Prenatal ($n = 319$)	7-y visit ($n = 319$)
Age (y)	25.2 ± 4.9	25.7 ± 5.0	32.3 ± 5.0
Weight (kg)	67.8 ± 17.1	67.8 ± 17.2[3]	77.0 ± 20.2
Height (cm)	162.3 ± 7.1	161.6 ± 7.4	161.6 ± 7.4
BMI (kg/m^2)	25.7 ± 5.9	25.9 ± 6.0[3]	29.4 ± 7.1
First pregnancy	175 (24.2)	75 (23.5)	—
Birth parent obesity			
BMI < 30 (kg/m^2)	548 (79.1)	238 (75.6)[4]	186 (58.3)
BMI ≥ 30 (kg/m^2)	145 (20.9)	77 (24.4)[4]	133 (41.7)
Marital status[5]			
Single	514 (73.7)	231 (72.9)	167 (52.4)
Married or cohabitating	183 (26.3)	86 (27.1)	152 (47.6)
Ability to afford food			
Could not afford food	119 (17.1)	51 (16.0)[6]	27 (8.5)
Could afford food	578 (82.9)	266 (84.0)[6]	292 (91.5)
Public assistance			
Not on public assistance	293 (42.0)	126 (39.7)[6]	136 (42.6)
On public assistance	404 (58.0)	191 (60.3)[6]	183 (57.4)
Race/ethnicity			
Dominican/Dominican American	451 (64.7)	203 (63.6)	203 (63.6)
Black	246 (35.3)	116 (36.4)	116 (36.4)
Total number of people living in household (n)	—	—	4.3 ± 1.6

Abbreviations: CCCEH, Columbia Center for Children's Environmental Health.
[1] Values are n (%) or mean ± SD.
[2] 697 of original 727 had BMI data; prenatal column is the 319 individuals whose children also have data from 695 baseline.
[3] $n = 315$ for birth parents included in sample at 7 y.
[4] Data available for $n = 315$.
[5] Single = widowed, divorced, separated, never married; Married or cohabitating = married or lived with same partner for 7 + y.
[6] Data available for $n = 317$.

TABLE 2
Child demographic characteristics and anthropometric data: CCCEH[1]

	7-y visit ($n = 319$)	9-y visit ($n = 319$)	Metabolic visit[2] ($n = 279$)
Age (y)	7.1 ± 0.2	9.1 ± 0.2	9.3 ± 1.1
Weight (kg)	28.4 ± 7.1	37.2 ± 9.9	41.6 ± 12.1
Height (cm)	125.0 ± 5.4	136.3 ± 6.5	140.6 ± 9.5
Obesity[3]	77 (24.1)	95 (29.8)	96 (34.4)
Percent body fat (%)	24.3 ± 6.2	26.2 ± 6.9	—
Z-scores			
Waist circumference	0.21 ± 1.03	0.35 ± 1.06	—
BMI	0.82 ± 1.12	0.93 ± 1.05	0.99 ± 1.01
Insulin level (uIU/mL)	—	—	8.1 ± 8.2
Glucose level (mg/dL)	—	—	87.1 ± 6.1
Triglyceride level (mg/dL)	—	—	73.9 ± 39.0
HDL level (mg/dL)	—	—	51.8 ± 12.0
Waist circumference (cm)	59.1 ± 7.9	65.1 ± 9.6	65.4 ± 9.1[2]
Cardiometabolic risk score	—	—	−0.33 ± 2.60

Abbreviations: CCCEH, Columbia Center for Children's Environmental Health.
[1] Values are n (%) or mean ± SD.
[2] $n = 260$ for metabolic visit BMI z-score; metabolic visit, a separate study visit occurring between child age of 8.5 and 12 y for blood sample collection.
[3] Obesity cutoffs—CDC: BMI/age > 95th percentile.

HDL concentrations, compared with the referent healthier cardiometabolic risk cluster group (RR: 1.12; 95% CI: 1.03, 1.21) (Supplemental Table 2). There were minimal changes in the observed associations after excluding children with metabolic visits that occurred before age 9 y (data not shown).

Does prenatal period impact associations between birth parent size in midchildhood and child outcomes? Is the association between prepregnancy obesity and BMI with child outcomes stronger than those for birth parent obesity at child age 7?

A series of additional models (Table 4) were fit with additional adjustment for prenatal and/or postnatal size measures, including prepregnancy BMI, GWGz, and birth parent weight retention to the child age 7-y visit. Including prepregnancy BMI or prepregnancy BMI and GWGz attenuated all associations, and all CIs crossed the null. After removing birth parent obesity at age 7 y from the model, prepregnancy BMI, as a continuous exposure, and prepregnancy obesity, as a categorical variable, were both positively associated with BMIz and BF% changes from child age 7 to 8 y. The β coefficient for prepregnancy obesity on child outcomes was the same as the midchildhood birth parent exposure in the primary model, whereas the effect of prepregnancy obesity on child BF% was stronger.

Sensitivity analyses

In addition to examining for effects of the prenatal period, we also conducted several sensitivity analyses. After excluding children with obesity at age 7 y, the association between birth parent obesity and child risk for obesity at 9 y of age was markedly higher than observed in the main analysis; specifically, the association was more than doubled, with the incidence rate ratio increasing from 1.26 (95% CI: 0.99, 1.61) to 3.06 (95% CI: 1.45, 6.47). Finally, no differences were observed for child outcomes when examining for effect modification of parental weight-related exposures by child sex.

Discussion

Although other studies have examined both prenatal and postnatal influences of birth parent obesity on child growth, body composition, and metabolic outcomes in early childhood [20], this study is the first, to our knowledge, to be conducted during midchildhood in a marginalized population [10]. In our cohort of Black and Dominican birth parent–child dyads living in metropolitan New York City, we found that birth parent obesity at child age 7 y was associated with gains in child BMIz, WC z-score, and BF% from 7 to 9 y in unadjusted models but was not associated with our primary outcome, child obesity at age 9 y. In our adjusted models with sample weighting to account for attrition from the prenatal period to child age 7 y, birth parent obesity at child age 7 y was positively associated with changes in BMIz and BF% from 7 to 9 y but was not associated with changes

TABLE 3
Regression models with associations of birth parent weight status with changes in child body size from 7 to 9 y

	Secondary outcomes			Primary outcome
	BMI *z*-score change from 7 to 9 y, β (95% CI)	Body fat (%) change from 7 to 9 y, β (95% CI)	Waist circumference *z*-score change from 7 to 9 y, β (95% CI)	Risk of obesity at 9 y, β (95% CI)
Model 1: Birth parent obesity (*n*)	319	319	319	319
Birth parent obesity when child is 7 y				
Unadjusted model	0.17 (0.05, 0.29)**	1.15 (0.30, 2.01)**	0.15 (0.01, 0.28)*	1.49 (1.06, 2.08)*
Adjusted model	0.15 (0.03, 0.27)*	1.27 (0.36, 2.18)**	0.18 (0.03, 0.33)*	1.26 (0.99, 1.61)
IPW	0.13 (0.02, 0.24)*	1.15 (0.22, 2.09)**	0.15 (−0.004, 0.31)	1.26 (0.97, 1.62)
Model 2: Birth parent weight change from 7 to 9 y (*n*)	290	290	290	290
Birth parent weight change at child age from 7 to 9 y				
Unadjusted model	−0.01 (−0.01, 0.001)	0.003 (−0.04, 0.05)	−0.003 (−0.01, 0.01)	1.01 (0.99, 1.03)
Adjusted model	−0.003 (−0.01, 0.003)	0.01 (−0.04, 0.06)	−0.001 (−0.01, 0.01)	1.00 (0.98, 1.03)
IPW	−0.003 (−0.01, 0.002)	0.01 (−0.03, 0.05)	−0.002 (−0.01, 0.01)	1.01 (0.98, 1.03)

Covariates in adjusted models included birth parent age, race (Dominican/Black), child sex, mean age at the 7-y visit, mean age at the 9-y visit, ability to afford food, receipt of public assistance, birth parent relationship status, and number of people living in the household. β-Coefficients are from linear regression, while IRR are estimated from Poisson regression with robust variance estimators.
Abbreviations: IPW, inverse probability weighting; IRR, incidence rate ratio.
*$P < 0.05$; **$P < 0.01$.

TABLE 4
Regression models examining associations between prenatal and postnatal birth parent weight status on child body composition at age 7–9 y: CCCEH

	Secondary outcomes			Primary Outcome
	Child change from age 7 to 9 y			Child risk of obesity at age 9 y
	BMI, *z*-score	Waist circumference, *z*-score	Body fat percentage	
	β (95% CI); % change from primary model (*n* = 310)	β (95% CI); % change from primary model (*n* = 292)	β (95% CI); % change from primary model (*n* = 304)	IRR (95% CI); % change from primary model (*n* = 308)
Birth parent obesity				
Primary model (IPW from Table 3)	0.21 (0.1, 0.32)***; —	0.13 (−0.03, 0.3); —	1.19 (0.24, 2.14)*; —	1.28 (0.97, 1.69); —
+Prepregnancy BMI	0.11 (−0.02, 0.25); −47.6	0.10 (−0.08, 0.3); −23.1	0.29 (−0.88, 1.45); −75.6	1.18 (0.86, 1.63); −7.8
+Prepregnancy BMI, GWGz	0.11(−0.03, 0.24); −47.6	0.07 (−0.11, 0.25); −46.1	0.20 (−0.95, 1.35); −83.2	1.16 (0.85, 1.58); −9.4
+Prepregnancy BMI, PPWR	0.11(−0.04,0.28); −47.6	0.19 (−0.05, 0.42); +46.1	0.78 (−0.76, 2.32); −34.4	1.18 (0.82,1.69); −7.8
Prepregnancy BMI (kg/m^2)	0.02 (0.01, 0.02)***	0.01 (−0.001, 0.02)	0.15 (0.08, 0.23)***	1.01 (0.996, 1.03)
Prepregnancy obesity	0.21 (0.11, 0.32)***	0.09 (−0.08, 0.25)	1.79 (0.66, 2.92)**	1.22 (0.95, 1.55)

Covariates included birth parent age, race (Dominican/Black), child sex, average age at the 7-y visit, average age at the 9-y visit, ability to afford food, receipt of public assistance, birth parent relationship status, and number of people living in the household. β-Coefficients are from linear regression, while incidence rate ratio (IRR) are estimated from Poisson regression with robust variance estimators. Child risk of obesity at age 9 y was defined by BMI/age ≥ 95th percentile.
Abbreviations: CCCEH, Columbia Center for Children's Environmental Health; GWGz, gestational weight gain *z*-score; PPWR, postpartum weight retention.
*$P < 0.05$; **$P < 0.01$; ***$P < 0.001$.

in child WC *z*-score and child obesity at age 9 y. This lack of association between birth parent obesity with child WC *z*-score and obesity at age 9 suggests effects of attrition and confounding were present in our unadjusted models. Interestingly birth parent weight changes, but not obesity, were associated with child metabolic indicators. After inclusion of prenatal factors in the model to disentangle prenatal compared with postpartum influences, associations between birth parent obesity at 7 y with child growth and body composition change were not present. When comparing prenatal compared with postnatal associations, the effect size between prenatal and birth parent obesity at child age 7 y were the same for child BMIz changes and stronger for the child BF%, suggesting that the prenatal period has similar effects for relative size measures and a stronger effect for sensitive measures of adiposity.

In our study population, obesity was common among birth parents and was positively associated with changes in continuous anthropometric and adiposity measures in midchildhood

but not with risk of obesity at age 9 y. Similar to our results, in a LatinX cohort from San Francisco, birth parents with obesity had children more likely to develop obesity, per Centers for Disease Control and Prevention definition, at 9 y of age; however, body fat was not assessed, and the study did not evaluate for effects of the prenatal period [38]. Likewise, in a household-based United States cohort ($n = 3284$) conducted from 1997 to 2017, parental obesity was associated with steep increases in BMI from ages 5 to 12 y in data from 1997 children [39]. In a Chinese cohort of 2066 parent–child trios, in children aged 6–14 y, birth parent obesity measured at the beginning of the study was associated with both changes in child BF% (assessed with bioelectrical impedance anaylsis) and BMIz over the subsequent 2 y [40], which supports our findings.

As developmental programming of excess adiposity may have lasting effects, we sought to evaluate whether prenatal factors impacted associations observed in our primary models [41]. Inclusion of prenatal measures—prepregnancy BMI and GWGz—in our models attenuated the association between birth parent obesity at 7 y postpartum with child BMIz, WC, and body fat changes from ages 7 to 9 y, indicating that prenatal factors may play a role in child growth and body composition well into childhood. After adding prenatal factors to the model, the effect size for BMIz decreased but they remained significant, suggesting that current birth parent weight status exerts a strong impact on child relative body size. Risk of child obesity at age 9 y neared but did not reach statistical significance; with adjustments for prenatal exposures, the influence of postpartum obesity on risk of obesity decreased and the CI widened, suggesting effects of prenatal factors persist into midchildhood. Research has shown the long-term influence of the prenatal period may be related to inflammation and insulin resistance that high prepregnancy BMI and GWG confer in pregnant people, which could impact the intrauterine environment and result in epigenetic programming for adiposity in the fetus [42]. This type of epigenetic programming has been proposed as a potential mechanism relating health during and around pregnancy to offspring obesity risk by impacting the development of metabolic and neural signaling pathways that relate to eating behaviors [42,43].

Attenuation of the outcomes at 7 y by prenatal body composition with GWG and prepregnancy body size adds to the growing body of evidence that fetal programming has effects into early childhood [44]. However, our data also reveal associations with child body size and birth parent weight after 7 y, indicating the surrounding environment is important to consider. The complex relationship between prenatal and environmental mechanisms illustrates the need to focus on a family-centered health approach that starts before conception and continues as children turn into adults themselves. In children's first 5 y, their home environment is highly influenced by parental choices including diet, physical activity, screen time, sleep time, and more, with children sharing similar characteristics of these habits as their parents [45]. Other studies have also consistently shown that parental food behaviors and intake patterns influence their children's behaviors and patterns and higher postpartum weight retention may be indicative of more obesogenic eating behaviors and home environment [46]. Although the present study did not evaluate dietary intake, research indicates that dietary intake impacts body composition in children and should be evaluated in future studies [47,48].

We found that birth parent weight changes from child age 7 to 9 y were associated with increased risk of having moderately adverse cardiometabolic health profiles in our sample, compared with the healthy profile, but, interestingly, did not observe an association between birth parent obesity with child metabolic profiles. One study of Brazilian children in an urban environment reported associations of postpartum obesity with increased risk of poor cardiometabolic health profiles in their offspring at age 8-9 y [49], which is in contrast to our lack of findings for birth parent obesity at 7-y postpartum. We do not understand why we observed associations for birth parent weight changes and not for obesity for child metabolic health profiles, and there is no literature on this topic for comparison. These findings indicate that potential environmental factors such as dietary and family exercise practices may be influencing metabolic indicators rather than child adiposity directly. Additionally, few children in our sample had out-of-range laboratory values for metabolic indicators, while more children had obesity.

Our sensitivity analysis demonstrating a marked increase in child risk of obesity at 9 y after exclusion of children with obesity at 7 y highlights the association of birth parent obesity with their offspring developing obesity. Programs aimed at childhood obesity should be supported throughout the life course [20]. Although some research has demonstrated female children are more at risk for obesity and cardiometabolic risk factors [50,51], we did not find a statistically significant interaction for sex on the association between birth parent obesity and child growth. Similar to our findings, other studies have found no difference between child sex and risk for obesity, but this may be due to differences in populations [52].

This study has many strengths, including our unique study population of Black and Dominican dyads and the repeated measurements from this longitudinal birth cohort study since 1998. Several limitations include study participant involvement fluctuation between visits, for example, missing 9 y but coming to the 7-y or the additional 8.5–12-y visit (metabolic visit); however, when comparing baseline data in the analytic sample in this study with those of all persons in the cohort, the characteristics were comparable. Another limitation is potential for bias due to attrition from birth to 7 y; to reduce effects of attrition on our findings, we used inverse probability weighting in our primary analyses. Our sample size also reduced our ability to assess for interactions, particularly by sex. We also did not collect sufficient information on pubertal timing during body composition measurement visits; thus, we were unable to adjust for pubertal staging. Another limitation of the study is the use of self-report data, such as prepregnancy weight, although it is still considered a reliable measure [53].

In conclusion, childhood obesity remains a pervasive public health problem throughout the United States, especially among lower socioeconomic status and non-White children. Investigating the relationships between birth parent health and childhood obesity may lead to a better understanding of the factors driving obesity. The results of this study suggest that in marginalized communities, birth parent obesity in midchildhood is prospectively associated with greater gains in child adiposity and BMI and that birth parent weight changes are associated with child metabolic health profiles. These results also suggest that the family environment plays a major role in contributing to the development of adiposity, and further study is needed to

understand how social determinants of health such as the built environment, job stability, and equitable access to care influence pathways between birth parent size and child obesity. Future research is also needed to assess the effects of birth parent weight changes along with the household and social environment on child cardiometabolic health. Based on this evidence, interventions aimed at addressing childhood obesity should begin with improving birth parent health during and after pregnancy and emphasize early life exposures. These interventions should focus on both birth parent and child health into early childhood, allowing for access to healthy foods and adequate health care. Initiatives that address both parental and childhood obesity, and incorporate early prevention options for the entire family, may have the potential to improve child metabolic outcomes and not only be more likely to have lasting positive effects but will also need positive changes in social determinants of health for families to be able to make lasting and significant changes.

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Author contributions

The authors' responsibilities were as follows – FPP, AGR: had oversight over data collection; JRC, AH: collected data; LAH: managed the study database; SFF, IT, EMW, AGR, LAH: conducted data cleaning; SFF, IT, NRB, EMW: analyzed data; SFF, EMW, AGR, ARN, SEO, JMG, NRB, MRA: interpreted the data; and all authors: contributed to the manuscript during drafting and editing and approved the final manuscript.

Conflict of interest

The authors report no conflicts of interest.

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Data availability

The data for this research were used under a limited use data use agreement between Columbia University and the University of Texas at Austin. The data that support the findings of this study are available from Columbia University; but restriction apply to the availability of these data because of the need to maintain participant confidentiality. Data are available on request and review by the Columbia Center for Children's Environmental Health through an institutional data use agreement.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.cdnut.2024.103770.

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