



Published in final edited form as:

Paediatr Perinat Epidemiol. 2010 March ; 24(2): 179–189. doi:10.1111/j.1365-3016.2010.01098.x.

Antenatal and postpartum depressive symptoms are differentially associated with early childhood weight and adiposity

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Summary

Antenatal depression is associated with small for gestational age, but few studies have examined associations with weight during childhood. Similarly few studies address whether antenatal and postpartum depression differentially affect child weight. Among 838 mother-child dyads in Project Viva, a prospective cohort study, we examined relationships of antenatal and postpartum depression with child weight and adiposity. We assessed maternal depression at mid-pregnancy and 6 months postpartum with the Edinburgh Postnatal Depression Scale (score >13 indicating probable depression). We assessed child outcomes at age 3 years: body mass index (BMI) z-score, weight-for-height z-score (WHZ), sum of subscapular (SS) and triceps (TR) skinfold thickness (SS+TR) for overall adiposity, and SS:TR ratio for central adiposity. Sixty-nine (8.2%) women experienced antenatal depression and 59 (7.0%) postpartum depression. Mean (SD) outcomes at age 3 were: BMI z-score, 0.45 (1.01); SS+TR, 16.72 (4.03) mm; SS:TR, 0.64 (0.15). In multivariable models, antenatal depression was associated with lower child BMI z-score (-0.24 [95% confidence interval: -0.49, 0.00]), but higher SS:TR (0.05 [0.01, 0.09]). There was no evidence of a dose-response relation between antenatal depression and these outcomes. Postpartum depression was associated with higher SS+TR (1.14 [0.11, 2.18]). In conclusion,

whereas antenatal depression was associated with smaller size and central adiposity at age 3 years, postpartum depression was associated with higher overall adiposity.

Introduction

Beginning with birth and continuing through childhood and adolescence, weight, adiposity, and tempo of change in these physiological parameters are predictors of health outcomes. Underweight and weight gain below expected values, often labeled failure to thrive or weight faltering, indicate poor health, but so do overweight and rapid weight gain. In resource-poor settings, underweight is often the result of under-nutrition or disease. In other settings, causal factors may include problems in mother-infant interaction and feeding or maternal psychopathology.¹ Weight faltering in infancy is associated with developmental delays in early childhood²⁻⁴ and lower IQ persisting to age 8 years.⁵

On the other hand, rapid weight gain in childhood, perhaps particularly among those born small, may increase risk of obesity, high blood pressure, type 2 diabetes, and coronary events.⁶⁻¹² In addition to weight itself, adiposity and particularly central adiposity in childhood and adolescence are associated with adverse levels of lipids, insulin, and blood pressure.^{13,14} Child overweight and obesity are also associated with increased risk of asthma, orthopedic problems, sleep apnea, and psychosocial difficulties¹⁵⁻¹⁹. Given the current epidemic of obesity and sharply increasing rates even among very young children,²⁰⁻²⁶ along with the difficulties of combating obesity once present, it is important to identify potentially modifiable risk factors that operate early in life.

Two under-studied potential risk factors for unhealthful childhood growth are antenatal and postpartum depression. These mood disorders are quite common, with a recent meta-analysis reporting period prevalences of 18% for antenatal depression and 14% in new mothers during the first 6 months after delivery.²⁷ Although antenatal depression is associated with higher rates of pregnancy complications and adverse birth outcomes, including low birth weight, preterm birth, and small for gestational age,²⁸ there has been very little study of the impact of antenatal depression on size and growth in infancy and childhood.^{29,30} Women with antenatal depression have higher levels of corticotropin-releasing hormone (CRH) than non-depressed women,³¹ and we have shown that maternal CRH levels are associated with lower body mass index (BMI) z-scores and higher central adiposity in the child.³² Thus we hypothesized that children exposed to antenatal depression, compared to those not exposed, would weigh less, but have more central adiposity at age 3 years.

Postpartum depression, through its adverse effects on breastfeeding, maternal eating behavior and physical activity, and mother-child interaction may also influence early childhood weight and weight gain. We hypothesized that children exposed to postpartum depression would weigh more and have greater adiposity at age 3 compared to children not exposed to postpartum depression.

There are several biological and behavioral pathways through which antenatal and/or postpartum depression may affect growth in children. One promising biological pathway

that may mediate the effect of antenatal depression is CRH, as discussed above. In addition, if there is an association between antenatal depression and child size at age 3, it is important to discover whether the difference is due to growth in utero or postnatal growth, thus size at birth is a potential mediator of the effect of antenatal depression. Finally, maternal behaviors that may be associated with antenatal and/or postpartum depression may act as mediators of the effect of these exposures. Particularly pertinent to weight and adiposity in young children are duration of breastfeeding and age of introduction of solid foods, both of which may mediate the effect of antenatal or postpartum depression. Subsequent depression may also play a mediating role.

The goal of this analysis was to examine the relationships of antenatal and postpartum depression with child weight and adiposity at age 3 and with change in weight from birth to age 3 in a well-nourished population. We also assessed several pathways through which maternal depression may influence child size: CRH (antenatal only), birthweight (antenatal only), gestational age at birth (antenatal only), postpartum depression (antenatal only), breastfeeding duration (antenatal and postpartum depression), timing of introduction of solid foods (antenatal and postpartum depression), and depression at 1 year postpartum (postpartum depression only).

Methods

Sample and data collection

Subjects were from Project Viva, a prospective cohort study of pregnant women and their singleton children. Participants were recruited at their first prenatal visit from eight obstetric practices in the greater Boston area. We excluded subjects based on the following exclusion criteria: multiple gestation, inability to answer questions in English, plans to move out of the area before delivery, and gestational age more than 22 weeks at first prenatal visit. Between 1999 and 2002, Project Viva staff enrolled 2670 pregnant women (64% of those eligible); 329 subsequently became ineligible (60% because they were no longer pregnant), 195 withdrew, and 18 were lost to follow-up; thus 2128 participants delivered a live infant. Of these, 1681 (79%) completed a mid-pregnancy questionnaire that assessed depressive symptoms and 1263 (75% of those who completed the mid-pregnancy questionnaire) completed a second questionnaire that assessed depressive symptoms at 6 months postpartum. Project Viva staff also collected data at birth, and at 1, 2, and 3 years after delivery. Among 1249 participants with depressive symptom data at mid-pregnancy and 6 months postpartum, 898 had anthropometric data for children at age 3 years. Women with antenatal depression had 3-year outcome data at the same rate as women without antenatal depression (71% vs. 72%); women with postpartum depression were slightly less likely than their non-depressed counterparts to have 3-year outcome data (67% vs. 73%). Of the 898 subjects with anthropometric data at age 3 years, we excluded 60 (6.7%) due to missing covariate information, for a final sample size of 838.

Measures

Depressive symptoms—We assessed depressive symptoms with the 10-item Edinburgh Postnatal Depression Scale (EPDS) in mid-pregnancy (mean of 28 weeks' gestation) and at

approximately 6 months after birth. We chose the EPDS because it has been validated for antenatal and postpartum use.³³ We used the cutpoint of 13 or more (on the 0-30 point scale) to indicate probable depression, consistent with previous work in our cohort^{31,34} and in other large cohorts that collected EPDS data antenatally and postnatally.³⁵ This cut-point indicates probable depression with a sensitivity of 86% and specificity of 78% in the postnatal period.³³ The optimal cut-off for probable major depression in the antenatal period may be higher (15 or more),³⁶ thus we report results for primary models using this alternate cut-off. Finally, it should be noted that the EPDS is a screening tool that measures probable depression, and is not a clinical diagnosis of depression; however, to be succinct, we refer to an EPDS score ≥ 13 as antenatal or postpartum depression.

Child anthropometric measurements—Project Viva trained research staff completed measurements at birth, 6 months, and 3 years of age. At the 3-year visit, trained research staff measured weight with a Seca scale (model 881; Seca, Hanover, MD), height with Shorr height board (Shorr Productions, Olney, MD), and subscapular (SS) and triceps (TR) skinfold thicknesses with a Holtain caliper (Holtain Ltd., Crosswell, Crymych, Dyfed Wales, UK). Research staff followed standardized techniques for all measures³⁷ and participated in biannual training to ensure measurement validity (Irwin Shorr, MPH). Interrater and intrarater measurement errors compare well with published reference ranges for all measurements.³⁸ Experienced field supervisors provided ongoing quality control by observing and correcting measurement technique every 3 months. We measured weight in kilograms, length/height in centimeters, and skinfold thickness in millimeters. We obtained anthropometric data for ages 1 and 2 years from clinical records.

From weight and height at age 3, we calculated body mass index (BMI) (kilograms per meter squared) and each child's age- and sex-specific BMI z-score according to US national reference data.³⁹ To examine the extreme ends of the distribution, we created dichotomous variables indicating BMI greater than the 85th percentile and less than the 15th percentile of the national reference standards.³⁹ We calculated age- and sex-specific weight-for-length z-score for each child at birth, 6 months, 1 year, and 2 years and weight-for-height z-score (WHZ) at 3 years. We used the sum of SS and TR (SS+TR) skinfolds as an estimate of overall adiposity and the ratio of SS to TR (SS:TR) as an estimate of central adiposity.³² In one study, the ratio of truncal to extremity skinfold thickness was correlated with intraabdominal adipose tissue ($r=0.77$), subcutaneous abdominal adipose tissue ($r=0.77$), and dual energy x-ray absorptiometry of the trunk ($r=0.69$) in children ages 3 to 8 years old.⁴⁰

Sociodemographic factors, confounders, mediators—We chose variables that have been previously linked with antenatal or postpartum depression or with child weight as covariates. Project Viva study staff collected maternal age, race/ethnicity, household income, education, partnership status, and pre-pregnancy BMI, as well as maternal report of paternal BMI at enrollment. At mid-pregnancy, we assessed history of depression before the index pregnancy (ever experienced two weeks or more of feeling depressed, down, or little interest in pleasurable activities plus either a diagnosis of depression by a doctor or prescribed medications for depression) and smoking behavior (categorized into former, never, current smoker). From clinical records, we calculated gestational weight gain (self-

reported pre-pregnancy weight subtracted from the last clinically recorded weight before delivery and categorized according to the Institute of Medicine's guidelines⁴¹), presence of gestational diabetes or impaired glucose tolerance during pregnancy, and infant birthweight. We assayed plasma concentration of CRH from maternal blood samples at mid-pregnancy (26-28 weeks), and used log maternal CRH as a marker of fetal glucocorticoid exposure.³² We calculated gestational age at delivery from the last menstrual period or 2nd trimester ultrasound if estimates differed by more than 10 days. We calculated birthweight for gestational age z-scores with use of US national reference data.⁴² We identified women who used medication for depression during pregnancy and the first six months postpartum by searching prescription data for the 90 days before the last menstrual period through six months postpartum for 29 commonly used antidepressants. All mothers in this study received their prenatal care and likely their postnatal care at the large group practice through which we recruited subjects; thus, prescription data from these practices, though not actual use data, is a suitable measure of use of antidepressants. We created two indicator variables: one for use of antidepressants during pregnancy (a potential confounder for antenatal depression) and one for use of antidepressants in the first six months postpartum (a potential confounder for postpartum depression). From questionnaires at 6 and 12 months, we assessed duration of breastfeeding and age at which solid foods were introduced to the child across the first year of the child's life. We categorized introduction of solid foods into early (before 4 months), mid (4-5 months), and late (at or after 6 months). In addition to assessing maternal depression during pregnancy and at 6 months after birth, we assessed maternal depression at 1 year postpartum with the EPDS.

Statistical analysis

Fifty-nine mothers experienced depression during pregnancy and 69 at 6 months postpartum. Only 18 mothers experienced depression at both time points. Due to the relative lack of overlap in these exposures, we examined antenatal and postpartum depression separately. We found nearly identical estimated effects for antenatal and postpartum depression whether we included these 18 subjects or not; thus they are included in both exposure categories.

We examined two sets of outcomes: BMI, WHZ, SS+TR and SS:TR at age 3, and change in WHZ from birth through age 3 years. For age 3 outcomes, we used multivariable linear regression. In the base model (Model 1) we adjusted for child sex and age in months at assessment. In Model 2, we additionally adjusted for sociodemographic and physiological factors related to maternal depression and/or child weight. To increase precision of the estimate of interest, if removal of a covariate did not substantially alter the effect estimate of the primary exposure, we did not include that covariate in the final models. Variables tested and not included for antenatal depression were maternal marital status, education, gestational diabetes or impaired glucose, smoking during pregnancy, use of antidepressant medication during pregnancy, and paternal BMI. Variables tested and not included for postpartum depression were maternal marital status, education, smoking during pregnancy, use of antidepressant medication in the first six months postpartum, and paternal BMI. To test whether incident postpartum depression showed a different relationship with the

outcomes compared to depression that occurred in both the antenatal and postpartum period, we also ran analyses restricted to women who did not have antenatal depression.

Potential mediators of antenatal depression were maternal mid-pregnancy CRH (available for 437 subjects), gestational age at birth, birthweight for gestational age, postpartum depression, breastfeeding duration, and age of introduction of solid foods. Potential mediators of postpartum depression were breastfeeding duration, age of introduction of solid foods, and maternal depression at one year postpartum. We performed statistical tests for mediation according to the product of coefficients test for mediation^{43,44} and test for significance of the mediated effect with the Sobel method.⁴⁵ We also present effect estimates for our exposures of interest after addition of the potential mediators in Tables 2 and 3.

In longitudinal models allowing a random intercept, random slope, and an unstructured covariance matrix, we modeled change in WHZ across time. We included the covariates in the fully-adjusted models (Model 2). In models with antenatal depression as the primary predictor, we modeled the change in WHZ from birth through age 3; for postpartum depression, 6 months to 3 years. We entered time as a categorical variable, with each outcome assessment (birth, 6 months, 1, 2, and 3 years) as a category and tested for an interaction between maternal depression and time. All analyses employed SAS version 9.1 (SAS Institute, Cary, NC), proc genmod and proc logistic for age 3 outcomes and proc mixed for longitudinal analyses.

Results

Mothers in this sample were predominantly white, married, and had an annual household incomes over \$70,000 (Table 1). Sixty-nine (8.2%) participants experienced antenatal depression and 59 (7.0%) experienced postpartum depression. Women with antenatal depression were less likely to be married and more likely to have lower income and inadequate weight gain during pregnancy. Their children showed lower mean WHZ, but higher SS:TR at age 3. Women with postpartum depression were less likely to be married and had lower income.

In multivariable models, antenatal depression predicted smaller body size at age 3 years, but greater central adiposity. As Table 2 displays, after controlling for potential confounders (Model 2), compared to children not exposed to antenatal depression, mean BMI z-score of exposed children was -0.24 (95% confidence interval (CI): -0.49, 0.00) lower; and WHZ was -0.24 (-0.48, -0.01) lower. Though they were lighter, children exposed to antenatal depression had more central adiposity, with SS:TR 0.05 (0.01, 0.09) higher. There was minimal difference in SS+TR according to antenatal depression. Results for Model 2 using the alternate EPDS cut-off (15 or more; n=41 subjects exposed) showed smaller effect estimates and larger confidence intervals for BMI z-score (-0.13 (-0.44, 0.18)), WHZ (-0.11, -0.41, 0.19)), and SS+TR (-0.05 (-1.30, 1.20)), but largely similar results for SS:TR (0.06 (0.01, 0.11)).

Including postpartum depression in the model did not attenuate the relationships between antenatal depression and child outcomes (Model 3, Table 2). In analysis of the extreme ends of the BMI distribution, antenatal depression was not a significant predictor of BMI greater than the 85th percentile (OR=0.64; 95% CI: 0.34, 1.22) or BMI of less than the 15th percentile (OR=1.88; 95% CI: 0.77, 4.60), but associations were in the same direction as those expected based on differences in mean BMI.

There was some evidence for mediation of the association between antenatal depression and BMI and WHZ by gestational age at birth, birthweight for gestational age, duration of breastfeeding, and age of introduction of solid foods (Models 4 and 5, Table 2), though the confidence intervals for the mediated effect crossed the null for all potential mediators. In the smaller sample (n=437) for which maternal CRH was available, there was no evidence of mediation by maternal CRH.

Postpartum depression was associated with higher 3-year SS+TR of 1.14 mm (0.11, 2.18) (Model 2, Table 3). Results for other outcomes showed very small effect estimates. We found similar estimates in models restricted to those without antenatal depression. There was evidence for mediation by depression at 1 year postpartum (mediated effect= 0.42; 95% CI: 0.08, 0.76), but no evidence of mediation by duration of breastfeeding or age of introduction of solid foods. Postpartum depression was not a predictor of BMI greater than the 85th percentile (OR=0.87; 95% CI: 0.45, 1.67) or of BMI less than the 15th percentile (OR=1.05; 95% CI: 0.34, 3.26).

BMI and WHZ are not fully independent of height in this dataset (correlation between age 3 height-for-age z-score and age 3: BMI z-score, $r=0.12$; WHZ, $r=0.24$). To examine the possibility that the relationship between maternal depression and our weight and adiposity outcomes were confounded by attained height at age 3, we added height-for-age z-score at age 3 to the fully adjusted models. This addition did not alter the observed relationships between maternal depression and the weight and adiposity outcomes.

In longitudinal modeling, the association between antenatal depression and child WHZ from birth through age 3 was similar for each age (Figure 1), thus we did not include an antenatal depression by age interaction term in the final model. The association between antenatal depression and child WHZ at each time point was -0.24 (95% CI: -0.43, -0.06), controlling for child sex and age at assessment, maternal age, race/ethnicity, household income, pre-pregnancy BMI, pregnancy weight gain, and postpartum depression. With the addition of gestational age and birthweight for gestational age, the estimated association was -0.21 (-0.40, -0.03). In longitudinal models of the association between postpartum depression and child WHZ, the estimated associations were similar for each age from 6 months to 3 years of age: 0.08 (95% CI: -0.14, 0.30), controlling for child sex and age at assessment, maternal age, race/ethnicity, household income, pre-pregnancy BMI, pregnancy weight gain, gestational diabetes or impaired glucose intolerance, gestational age at delivery, and birthweight for gestational age.

Discussion

Antenatal and postpartum depression had different associations with child weight and adiposity. Compared with unexposed children, children whose mothers experienced antenatal depression, on average, weighed less for a given height (as measured by lower BMI and WHZ), but had greater central adiposity, while children whose mothers experienced postpartum depression had greater overall adiposity. These relationships were independent of maternal sociodemographic factors, BMI, and health conditions during pregnancy. Longitudinal analyses revealed that the relationship between antenatal depression and lower WHZ was present at birth, and continued through age 3. The magnitude of the association of antenatal depression with child SS:TR (0.05) was similar to that of increasing maternal log CRH by 1 unit.³² For postpartum depression, the magnitude of the association with child SS + TR (1.14 mm) was similar to the effect of gestational diabetes.⁴⁶ The extent to which these changes in 3-year adiposity outcomes translate into future cardio-metabolic risk remains to be elucidated.

On the face of it, lower BMI at age 3 is not problematic, and may even seem good, given concerns of overweight in the US. However, measures of weight-for-height, such as BMI and WHZ, include lean muscle mass as well as fat. While weight-for-height measures are often used as an indicator of body fatness, the BMI-body fat relationship varies by age, race, sex, and distribution of fat.⁴⁷ Examination of adiposity in addition to BMI, and the specific distribution of that adiposity (central versus overall), gives a more nuanced understanding of the physiology and health risks of these children. While overall adiposity is associated with many health risks, central adiposity is more strongly associated with insulin resistance and hypertension than is peripheral fat.⁴⁸

The smaller size, but higher central adiposity for children exposed to antenatal depression is consistent with fetal programming of the hypothalamic-pituitary-adrenal (HPA) axis.³² In animal models, such prenatal programming is associated with adiposity, hypertension, hyperglycemia, and altered neuroendocrine responses throughout the lifespan.^{49,50} In the cohort examined for this analysis, fetal exposure to higher levels of CRH was associated with lower body size at age 3, but greater central adiposity;³² however we did not find evidence that maternal CRH mediated weight and adiposity outcomes at age 3 for children whose mothers experienced antenatal depression. Nevertheless, our longitudinal findings support the notion that antenatal depression may have a programming effect on child size: children of antenatally depressed mothers were smaller at birth and remained smaller than their unexposed counterparts through age 3.

Children whose mothers experienced postpartum depression had similar weight-for-height but more adiposity compared to unexposed children. Postpartum depression is associated with reduced breastfeeding,^{51,52} and breastfeeding may be a protective against development of obesity;⁵³⁻⁵⁵ however, breastfeeding did not play a mediating role in our analyses. Other possible unmeasured mechanisms may be that mothers with postpartum depression may have more unhealthful behaviors, such as over-eating⁵⁶ and limited physical activity, and their children may have similar behaviors. Additionally, postpartum depression and

psychological distress may contribute to impaired mother-infant interactions and to child feeding problems,⁵⁷⁻⁶⁰ leading to unhealthy eating habits in children.⁶¹

A recent cross-sectional study in Brazil reported a finding related to ours: a positive association between maternal depression between 6 and 24 months after birth and concurrent child overweight.⁶² However, our findings contrast with studies from the UK and India, in which there was an association between postpartum depression and *lower* weight (weight faltering or underweight) in children.^{1,63-65} Other studies from the UK, South Africa, and Jamaica have reported no association with underweight in childhood.^{29,66,67} To date, studies of postpartum depression and child physical development have focused on underweight, and no study that we know of has examined body fatness directly. Additionally, this is the first study that we are aware of to examine the relationship between antenatal depression and child weight and adiposity in a US sample. There are several possible explanations for divergent findings in the literature. Differences may be due to study design and measures: many studies have used cross-sectional data or case-control sampling, while our study had a prospective design with repeated outcome measurements that allowed us to establish temporal order between exposure and outcomes as well as examine change over time in outcomes. Additionally, our study used a well-validated measure of depressive symptoms and careful measurement of outcomes. There may also be confounding by unmeasured variables; for example, in resource-poor settings, household food availability may be an important confounder, and without control for it, there may be a spurious relationship between maternal depression and child underweight. Finally, these relationships have been examined in very different food and social contexts. It is possible that maternal depression causes underweight in settings with limited food availability, but overweight or adiposity in settings with food surplus.

One limitation of this study is that we did not use a clinical definition of depression, but relied on self-report of depressive symptoms. Use of a more stringent cut-off for antenatal depression (EPDS \geq 15 instead of 13) revealed smaller estimated effects on BMI and WHZ. This could indicate that depressive symptoms were not the true causal agent or that these cut-offs capture different disorders that have different relationships with child weight. We are unable to determine if either of these explanations is valid, but future research on the topic could help elucidate these issues. In addition, we assessed postpartum depression at 6 months, whereas the prevalence of postpartum depression is highest in the 2nd and 3rd months postpartum.²⁷ Women identified as depressed at 6 months postpartum are likely to have begun experiencing depression within a couple months of childbirth,⁶⁸ thus, the women we identify as depressed may represent those with a relatively long course. Misclassification of our exposure is likely nondifferential with respect to the outcomes, however, which would attenuate results towards the null. Women with postpartum depression were slightly less likely to have outcome data than their non-depressed counterparts; if the more severely depressed were less likely to have outcome data and there is a dose-response relationship between exposure and outcome, our estimated associations may under-estimate the relationship between postpartum depression and child weight and adiposity. Another limitation is that the mothers in Project Viva have relatively high socioeconomic status and the prevalences of antenatal and postpartum depression were

relatively low. Thus, our results may not be generalizable to populations at higher risk of perinatal depression. Finally, due to the relative independence of antenatal and postpartum depression, our results generalize primarily to women who have either antenatal or postpartum depression, although inclusion of women with both did not alter the estimate effects in this cohort of women.

In summary, we found that antenatal and postpartum depression had different associations with weight and adiposity in 3-year-old children: whereas antenatal depression was associated with smaller child size and central adiposity, postpartum depression was associated with higher overall adiposity. As one of the first studies to examine these relationships in a US sample, these findings should be replicated in other samples in the US and other developed nations. Our results raise the possibility that treating perinatal depression may reduce obesity in early childhood and potentially benefit the long-term health of children.

Acknowledgments

Supported by grants from the National Institutes of Health (HD 34568, HL 64925, HL 68041) and by Harvard Medical School and the Harvard Pilgrim Health Care Foundation.

The authors would like to thank Sheryl Rifas-Shiman and Dr. Ken Kleinman, and Dr. Lisa Berkman for their contributions to this study.

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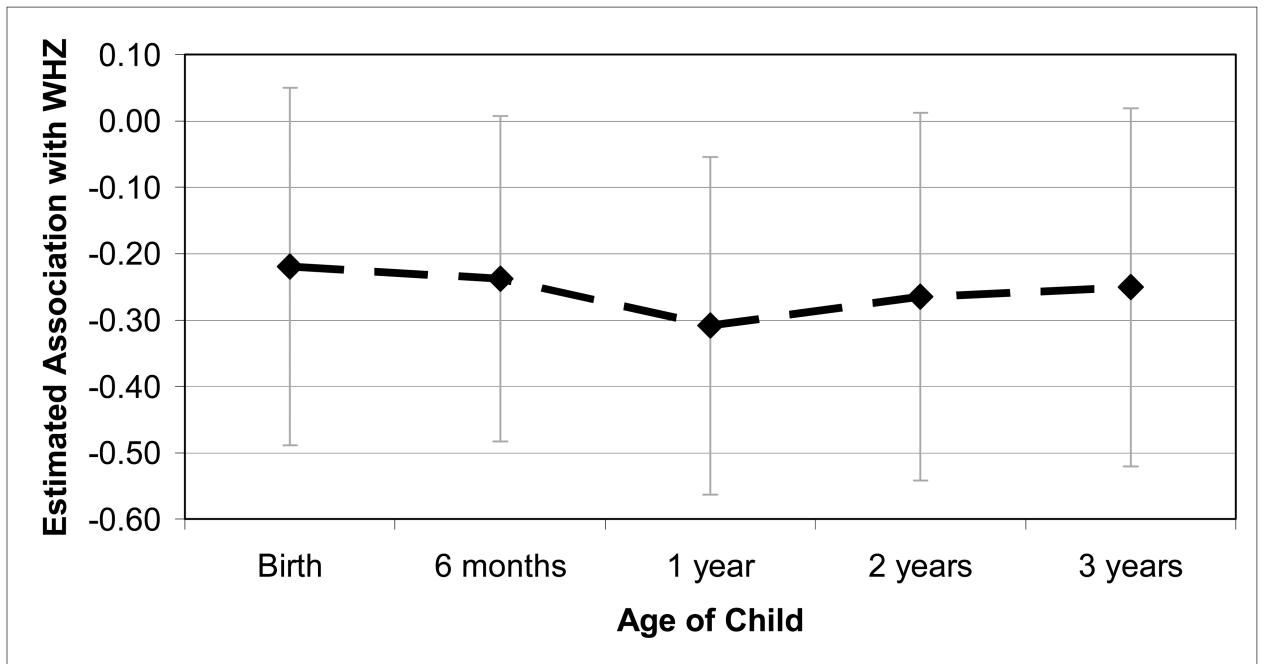


Figure 1. Estimated Longitudinal Association of Antenatal Depression with Child Weight-for-Height Z-Score (WHZ). Data from 838 mother-infant pairs participating in Project Viva. The zero line represents the effect among participants not exposed to antenatal depression. Multivariable regression estimates adjusted for age, race/ethnicity, household income, pre-pregnancy BMI, pregnancy weight gain, postpartum depression and child sex and age at assessment. Bars indicate 95% confidence intervals.

Table 1
 Characteristics of Project Viva Participants According to Maternal Antenatal and Postpartum Depression. Data from 838 mother-infant pairs participating in Project Viva.

	Overall (n=838)			Antenatal Depression			Postpartum Depression			p-value**	
	mean/n	SD/%		No (n=769)	Yes (n=69)		No (n=779)	Yes (n=59)			
				mean/n	SD/%	mean/n	mean/n	SD/%	mean/n	SD/%	
Maternal characteristics											
Age	32.97	4.46	33.06	4.41	31.96	4.87	33.08	4.35	31.48	5.49	0.03
Race/ethnicity							0.09				0.13
Black	65	7.76	56	7.28	9	13.04	57	7.32	8	13.56	
Other	98	11.69	87	11.31	11	15.94	89	11.42	9	15.25	
White	675	80.55	626	81.40	49	71.01	633	81.26	42	71.19	
Education							0.43				0.11
Less than BA/BS	179	21.36	160	20.81	19	27.54	160	20.54	19	32.20	
BA/BS	342	40.81	316	41.09	26	37.68	322	41.34	20	33.90	
Graduate degree	317	37.83	293	38.10	24	34.78	297	38.13	20	33.90	
Marital status *							<0.01				<0.01
Unpartnered/cohabitating	84	10.04	70	9.11	14	20.29	71	9.13	13	22.03	
Married	753	89.96	698	90.89	55	79.71	707	90.87	46	77.97	
Household income							<0.01				<0.01
<=\$40,000	72	8.59	57	7.41	15	21.74	59	1.57	13	22.03	
40,001 to \$70,000	190	22.67	170	22.11	20	28.99	171	21.95	19	32.20	
>=\$70,001	576	68.74	542	70.48	34	49.28	549	70.47	27	45.76	
Pre-pregnancy BMI							0.13				0.09
<25	566	67.54	525	68.27	41	59.42	532	68.29	34	57.63	
>=25	272	32.46	244	31.73	28	40.58	247	31.71	25	42.37	
Pregnancy weight gain (IOM)							0.02				0.86
Inadequate	122	14.56	104	13.52	18	26.09	113	14.51	9	15.25	
Adequate	297	35.44	274	35.63	23	33.33	278	35.69	19	32.20	
Excessive	419	50.00	391	50.85	28	40.58	388	49.81	31	52.54	
Gestational diabetes or IGT							0.01				0.83

	Overall (n=838)				Antenatal Depression				Postpartum Depression			
	mean/n	SD/%	mean/n	SD/%	No (n=769)		Yes (n=69)		No (n=779)		Yes (n=59)	
No	702	83.77	652	84.79	50	72.46	652	83.70	50	84.75		
Yes	136	16.23	117	15.21	19	27.54	127	16.30	9	15.25		
Mid-pregnancy log CRH *	4.98	0.64	4.98	0.64	5.05	0.65	5.00	0.64	4.74	0.56	0.03	
Breastfeeding duration (months) *	6.58	4.47	6.54	4.46	7.00	4.66	6.61	4.50	6.05	4.27	0.35	
Infant characteristics												
Sex												0.45
girl	437	52.15	399	51.89	38	55.07	409	52.50	28	47.46		
boy	401	47.85	370	48.11	31	44.93	370	47.5	31	52.54		
GA at delivery (weeks)	39.60	1.69	39.60	1.70	39.51	1.54	39.61	1.69	39.48	1.71	0.59	
BW for GA (z value)	0.24	0.94	0.25	0.93	0.05	0.97	0.24	0.92	0.16	1.11	0.57	
Child characteristics at 3-year visit												
Age (months)	38.96	3.23	38.97	3.27	38.88	2.81	38.98	3.27	38.79	2.69	0.56	
BMI z-score	0.45	1.01	0.46	1.00	0.24	1.11	0.45	0.99	0.46	1.19	0.94	
WHZ	0.43	0.97	0.45	0.96	0.23	1.07	0.43	0.96	0.49	1.10	0.66	
SS + TR (mm)	16.72	4.03	16.74	4.05	16.55	3.91	16.65	3.94	17.66	5.00	0.14	
SS:TR	0.64	0.15	0.63	0.15	0.69	0.18	0.64	0.16	0.63	0.15	0.66	

BA/BS, Bachelor of Arts or Science; BMI, body mass index (kg/m²); IOM, Institute of Medicine guidelines; IGT, impaired glucose tolerance; CRH, corticotropin-releasing hormone; GA, gestational age; BW, birth weight; WHZ, weight-for-height z-score; SS, subscapular skinfold thickness; TR, triceps skinfold thickness.

* Sample sizes are slightly different due to missing data: maternal log CRH n=437; breastfeeding duration n=835; marital status n=837.

** Chi-square p-value for categorical variables and T-test p-value for continuous variables.

Table 2

Differences in 3-year Weight and Adiposity Outcomes Among Children Exposed to *Antenatal* Depression v. Not Exposed. Data from 838 mother-infant pairs participating in Project Viva.

	Estimate (95% confidence interval)			
	BMI z-score	WHZ	SS+TR (mm)	SS:TR
Model 1	-0.22 (-0.47, 0.03)	-0.22 (-0.45, 0.02)	-0.21 (-1.19, 0.76)	0.05 (0.02, 0.09)
Model 2	-0.24 (-0.49, 0.00)	-0.24 (-0.48, -0.01)	-0.14 (-1.12, 0.84)	0.05 (0.01, 0.09)
<i>Including potential mediators:</i>				
Model 3	-0.24 (-0.49, 0.01)	-0.25 (-0.49, -0.01)	-0.37 (-1.36, 0.63)	0.05 (0.02, 0.09)
Model 4	-0.21 (-0.46, 0.03)	-0.22 (-0.45, 0.01)	-0.34 (-1.33, 0.66)	0.05 (0.02, 0.09)
Model 5	-0.17 (-0.42, 0.07)	-0.18 (-0.41, 0.05)	-0.17 (-1.16, 0.82)	0.06 (0.02, 0.10)
Model 1 covariates: child sex and age at 3-year assessment				
Model 2: Model 1 + maternal: age, race/ethnicity, household income, pre-pregnancy BMI, pregnancy weight gain				
Model 3: Model 2 + postpartum depression				
Model 4: Model 3 + GA at birth and BW for GA z-value				
Model 5: Model 4 + breastfeeding duration and age of introduction of solid foods (n=835)				

BMI, body mass index (kg/m²); WHZ, weight-for-height z-score; SS, subscapular skinfold thickness; TR, triceps skinfold thickness; GA, gestational age; BW, birth weight

Table 3

Differences in 3-year Weight and Adiposity Outcomes Among Children Exposed to *Postpartum* Depression v. Not Exposed. Data from 838 mother-infant pairs participating in Project Viva.

	Estimate (95% confidence interval)			
	BMI z-score	WHZ	SS+TR (mm)	SS:TR
Model 1	0.002 (-0.26, 0.27)	0.04 (-0.21, 0.30)	1.10 (0.05, 2.14)	-0.01 (-0.05, 0.03)
Model 2	-0.05 (-0.31, 0.21)	-0.01 (-0.25, 0.23)	1.14 (0.11, 2.18)	-0.02 (-0.06, 0.02)
<i>Including potential mediators:</i>				
Model 3	-0.04 (-0.30, 0.21)	-0.01 (-0.25, 0.24)	1.15 (0.11, 2.18)	-0.02 (-0.06, 0.02)
Model 4	-0.17 (-0.46, 0.11)	-0.13 (-0.40, 0.13)	1.07 (-0.06, 2.20)	-0.04 (-0.08, 0.01)
Model 1 covariates: child sex and age at 3-year assessment				
Model 2: Model 1 + maternal: age, race/ethnicity, household income, pre-pregnancy BMI, pregnancy weight gain, gestational diabetes or glucose intolerance during pregnancy, GA at birth, BW for GA z-value				
Model 3: Model 2 + breastfeeding duration and age of introduction of solid foods (n=835)				
Model 4: Model 3 + depression at 1 year postpartum (n=773)				

BMI, body mass index (kg/m²); WHZ, weight-for-height z-score; SS, subscapular skinfold thickness; TR, triceps skinfold thickness; GA, gestational age; BW, birth weight