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Timing and Cumulative Exposure of Perinatal Depressive Symptoms in Relation to Infant Rapid Weight Gain: A Prospective Longitudinal Study

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Abstract

Background

Infant rapid weight gain (RWG) is an important predictor of later obesity. Although perinatal depressive symptoms have been linked to adverse offspring growth and development outcomes, evidence regarding their association with infant RWG remains limited, and existing studies have relied on single-time-point assessments.

Objectives

To examine associations of timing and cumulative exposure to perinatal depressive symptoms with infant RWG.

Methods

This prospective longitudinal study included 576 mother–infant dyads. Stage-specific perinatal depressive symptoms and cumulative depressive symptom burden were assessed using the Edinburgh Postnatal Depression Scale (EPDS) across seven waves. RWG was defined as an increase in weight-for-age Z-score (WAZ) > 0.67 from birth to 1, 3, or 6 months. Logistic regression and restricted cubic spline analyses were used to examine associations.

Results

Postnatal depressive symptoms were associated with increased odds of RWG during 0–1 month [Adjusted Odds Ratio (aOR) = 2.10, 95% confidence interval (CI): 1.05–4.17, $p = 0.035$], whereas no significant associations were observed for prenatal or persistent depressive symptoms. Regarding cumulative burden, each 1-point increase in cumulative average EPDS score was associated with a 10% increase in odds at 0–3 months (aOR = 1.10, 95% CI: 1.03–1.17, $p = 0.005$) and a 7% increase at 0–6 months (aOR = 1.07, 95% CI: 1.00–1.14, $p = 0.036$). Restricted cubic spline analyses suggested an approximately linear association for both periods.

Conclusions

Cumulative depressive symptom burden is associated with an increased risk of RWG during the first 3 and 6 months of life in a potential linear dose-response manner. The postnatal period appeared to be a critical window during which maternal depressive symptoms were associated with increased RWG risk. These findings highlight the importance of maternal mental health for early growth patterns and obesity prevention.

1. Introduction

Childhood overweight and obesity represent a serious global public health challenge. According to data from the World Health Organization (WHO), the prevalence of overweight and obesity among children and adolescents aged 5–19 years worldwide increased markedly from 8% in 1990 to 20% in 2022[1]. Childhood and adolescent overweight and obesity increase the risk of early-onset non-communicable diseases and are linked to adverse psychosocial outcomes[2–4]. The WHO highlighted that failure to identify excess weight gain at an early stage is a key contributor to overweight and obesity[1]. Infant rapid weight gain (RWG) is typically defined as a change in age-specific weight-for-age Z score (WAZ) **>** 0.67 between two time points[5–7]. Accumulating evidence consistently demonstrates that RWG in infancy strongly predicts childhood overweight and obesity and is associated with adverse metabolic outcomes later in life[8–13], with its predictive value being particularly pronounced during the first six months of life[8, 14–16]. Consequently, addressing modifiable risk factors in early infancy is essential for the effective prevention and control of childhood overweight and obesity.

Consistent with the Developmental Origins of Health and Disease (DOHaD) hypothesis, early-life developmental exposures (e.g., nutrition, hormonal milieu, and stress) exert long-term effects and thereby shape the risk of adverse health outcomes later in life[17]. Perinatal depression is a common maternal mental health condition, with a global prevalence of approximately 26.3% and a prevalence of 16.3% in China[18, 19]. Perinatal depression occurs during a critical window of opportunity for infant growth and development[20]. During this period, infants' developmental plasticity may increase their susceptibility to maternal depressive symptoms[16]. Previous studies have examined the association between maternal perinatal depressive symptoms and infant weight gain; however, the evidence remains limited and inconsistent. For example, Gaffney et al. reported that infants of mothers with depressive symptoms at two months postpartum experienced greater weight gain from birth to six months compared with those of non-depressed mothers[21]. In contrast, another study examining the direct association between maternal prenatal depressive symptoms and rapid infant weight gain did not find a significant association[22].

Meanwhile, existing studies have primarily relied on single time-point assessments of perinatal depressive symptoms and have not considered the impact of depressive exposure across different perinatal stages or cumulative depressive burden from a longitudinal perspective. Research suggests that different exposure windows of depressive symptoms, as well as cumulative depressive burden, may have differential effects on offspring growth and development. For example, studies have shown that prenatal and postnatal depressive symptoms were differentially associated with early offspring adiposity. Compared with offspring of mothers without depressive symptoms, children exposed to prenatal depressive symptoms showed no significant difference in overall adiposity at 3 years of age, whereas those exposed to postnatal depressive symptoms exhibited higher overall adiposity[23]. Moreover, previous systematic reviews have revealed that persistent or recurrent maternal depressive symptoms identified through longitudinal assessments are more strongly associated with childhood

overweight and obesity than episodic depressive symptoms assessed at a single time point[24–26]. Therefore, single time-point assessments of depressive symptoms may underestimate the true impact of perinatal depressive symptoms on infant RWG.

In summary, this study aims to examine the associations between maternal perinatal depressive symptoms and RWG in infants during the first six months of life, considering both stage-specific perinatal depressive symptoms and cumulative depressive symptom burden. By elucidating these associations, the present study seeks to provide novel insights for early-life weight management strategies and contribute to the early prevention of childhood overweight and obesity.

2. Methods

2.1 Study design and populations

This prospective mother–infant cohort study was conducted at a large tertiary (Grade A) hospital in Hunan Province, China. Between October 2021 and June 2024, 779 women in early pregnancy were recruited from the obstetric outpatient clinic (T1: 7–13 gestational weeks). Eligible participants were followed at six subsequent time points: T2 (24–27 weeks), T3 (34–37 weeks), T4 (1 week postpartum), T5 (1 month postpartum), T6 (3 months postpartum), and T7 (6 months postpartum). Infants attended routine health examinations at the hospital's child healthcare center at 1, 3, and 6 months of age. The eligible criteria included: (1) age ≥ 18 years; (2) spontaneous conception with singleton pregnancy; (3) access to and ability to use the internet and telephone; and (4) willingness to deliver at the study hospital. Exclusion criteria included: (1) current severe psychiatric disorder (suicidal ideation or other severe mental illness diagnosed by a psychiatrist, e.g., bipolar disorder or schizophrenia); and (2) severe major physical diseases.

During follow-up, mother–infant dyads were excluded if any of the following occurred: (1) Obstetric complications: life-threatening conditions such as eclampsia or massive hemorrhage; (2) Clinical intervention: women diagnosed with clinical depression who initiated pharmacological treatment; (3) Adverse birth outcomes: stillbirth, fetal death, preterm birth (< 37 weeks), or abnormal birth weight (low birth weight < 2,500g or macrosomia > 4,000g); (4) Neonatal health: infants with congenital malformations, neonatal asphyxia, or other chronic diseases affecting physical development. Participants with at least three depressive symptom assessments and at least two infant weight measurements were included, yielding a final sample of 576 mother–infant dyads (Fig. 1). The study was approved by the Ethics Committee of Xiangya Nursing School of Central South University (approval number: E202159), and written informed consent was obtained from each participant. This study was conducted and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

2.2 Data assessment and definitions

2.2.1 Assessment of exposure

Perinatal depressive symptoms were measured using the Edinburgh Postnatal Depression Scale (EPDS), a widely used self-report screening instrument that has been validated in China[27, 28]. It consists of 10 items assessing depressive symptoms over the preceding 7 days, each of which is scored on a 4-point scale (range, 0–3), and the total score was 30, with a higher score showing more severe depressive symptoms. A cutoff of 10 or more points was defined as positive for perinatal depressive symptoms according to the published recommendations[29, 30]. In this study, perinatal depressive symptoms were measured at seven follow-up time points from the prenatal period to 6 months postpartum (T1–T7). Both exposure window and cumulative exposure of perinatal depressive symptoms were assessed.

Depressive symptoms exposure windows were defined based on EPDS assessments conducted during the antenatal period (T1–T3) and the postnatal period (T4–T7). The endpoint weight measurement used to define rapid infant weight gain was treated as the reference, and the most recent EPDS score prior to this time point was used to define the cutoff for depressive symptoms exposure. Participants were classified into four groups: no depressive symptoms (EPDS < 10 at all times), antenatal symptoms (EPDS ≥ 10 only during T1–T3), postnatal symptoms (EPDS ≥ 10 only during T4–T7), and persistent symptoms (EPDS ≥ 10 at any antenatal time point and EPDS ≥ 10 at any postnatal time point).

Cumulative exposure of perinatal depressive symptoms was quantified using the cumulative average EPDS score to reflect long-term depressive burden. Time-weighted EPDS values were calculated using the trapezoidal rule to estimate the area under the EPDS–time curve (AUC), from which the average exposure level was derived[31]. The cumulative average EPDS score was calculated using the following formula: $\frac{1}{t_n - t_1} \sum_{i=1}^{n-1} \left(\frac{EPDS_i + EPDS_{i+1}}{2} \right) (t_{i+1} - t_i)$.

2.2.2 Assessment of outcome

Infant RWG was defined as a change in age-specific WAZ **>** 0.67 between two time points and was treated as a binary variable (yes/no)[5–7]. WAZ were calculated using WHO Anthro software. In this study, RWG at 0–1 month was defined as a difference of > 0.67 between the WAZ at 1 month of age and the WAZ at birth; RWG at 0–3 months was defined as a difference of > 0.67 between the WAZ at 3 months of age and the WAZ at birth; and RWG at 0–6 months was defined as a difference of > 0.67 between the WAZ at 6 months of age and the WAZ at birth. Birth weight was obtained from obstetric medical records. Infant weight at 1, 3, and 6 months of age was measured by trained nurses at child healthcare centers in a private room. During measurement, infants wore only underwear, without shoes or diapers, and weight was assessed using a calibrated infant precision scale. Each measurement was performed three times, and the mean value was recorded in the child healthcare information system.

2.2.3 Assessments of covariates

To minimize participant burden, covariates were collected across different follow-up time points. First, demographics were obtained at baseline (T1), including age, pre-pregnancy body mass index (BMI),

residence, ethnicity, occupation, educational level, household income and passive smoking status, while data on pregnancy complications, mode of delivery, and infant sex were collected at 1 week postpartum (T4).

Furthermore, based on prior literature, additional potential covariates were selected a priori. Perceived social support was assessed at baseline (T1) using the Perceived Social Support Scale (PSSS), a 12-item instrument covering three dimensions (family, friends, and others), with total scores ranging from 12 to 84, where higher scores indicate greater perceived social support[32]. The Cronbach's α of PASS was 0.84. Stressful life events during pregnancy were measured in late pregnancy (T3) using the Life Events Scale for Pregnant Woman (LESPW), which includes 53 items (score range: 0–53), with higher scores indicating greater prenatal stress exposure[33]. The Cronbach's α of LESPW was 0.75. Breastfeeding knowledge was assessed in late pregnancy (T3) using the Breastfeeding Knowledge Questionnaire (BKQ), a 17-item scale evaluating knowledge of breastfeeding benefits and skills (score range: 0–17), with higher scores reflecting greater knowledge[34]. The Cronbach's α of BKQ was 0.87[35]. Infant feeding attitudes were assessed in late pregnancy (T3) using the Iowa Infant Feeding Attitude Scale (IIFAS), which consists of 17 items (score range: 17–85), with higher scores indicating more positive attitudes toward exclusive breastfeeding and a greater likelihood of longer breastfeeding duration[36]. The Cronbach's α of IIFAS was 0.635. Infant feeding patterns were assessed using the questionnaire item "Which type of feeding have you provided to your child in the past 24 hours?" at 1, 3, and 6 months postpartum (T5–T7).

2.3 Statistical analysis

We used descriptive statistics to summarize participants characteristic. Continuous variables were reported as mean (standard deviation, SD) or median (interquartile range, IQR), and categorical variables as frequency (percentage). Baseline characteristics were compared across groups defined by stage-specific perinatal depressive symptoms and quartiles of cumulative average EPDS scores. Continuous variables were analyzed using one-way ANOVA test, categorical variables were compared using the chi-square test or Fisher's exact test, and ordinal variables were analyzed using the rank-sum test.

The associations between perinatal depressive symptoms and infant RWG at 0–1, 0–3, and 0–6 months were examined using binary logistic regression analysis, with adjusted odds ratios (aORs) and their 95% confidence intervals (CIs) reported. First, stage-specific perinatal depressive symptoms, categorized into four groups, was included as the independent variable, with the non-depressed group serving as the reference category in binary logistic regression models. Second, the cumulative average EPDS score was treated as a continuous variable. It was further categorized into quartiles (Quartile 1 (Q1) to Q4), with Q1 serving as the reference group in binary logistic regression analyses. Covariates were selected based on two criteria: (1) variables that showed statistically significant differences at baseline ($P < 0.05$), and (2) relevant confounders consistently reported in previous studies to influence infant RWG, including pre-pregnancy BMI, stressful life events, breastfeeding knowledge, infant sex, birth weight, and feeding patterns. Additionally, a multivariable-adjusted (fully adjusted) restricted cubic spline (RCS) logistic

regression analysis was conducted, with 3 knots selected at the 10th, 50th, and 90th percentiles, respectively, to examine the linearity and the dose–response relationship between the cumulative average EPDS score and the risk of infant RWG.

Variables with missing data and the percentage of missing observations are presented in Supplementary Figure S1. To examine the pattern of missingness, Little's Missing Completely at Random (MCAR) test was conducted. The result was not statistically significant (χ^2 (700) = 741.502, P = 0.134), suggesting that the assumption of MCAR could not be rejected. In addition, baseline demographic characteristics measured at T1 were compared between completers and non-completers (i.e., participants with any missing data at follow-up). No significant differences were observed in these variables (all $P > 0.05$), suggesting that attrition was unlikely to be systematically associated with observed demographic characteristics (see Supplementary Table S1). Ultimately, multiple imputation was used to handle the missing data. All analyses were conducted across the imputed datasets, and parameter estimates were pooled according to Rubin's rules.

All analyses were conducted using SPSS 27.0 and R version 4.5.1. Two-sided P values of 0.05 were considered statistically significant.

2.4 Sensitivity analysis

To assess the potential impact of missing data on the study findings, a complete-case analysis was conducted as a sensitivity analysis. The consistency between the complete-case results and the primary analysis was examined to evaluate the robustness of the findings.

3. Results

3.1 Characteristic of study participants

The participant characteristics are detailed in Table 1. Among the 576 participants, the mean age was 28.51 ± 3.21 years, the mean pre-pregnancy BMI was 21.11 ± 2.92 kg/m²s, 91.49% were living in urban area, 89.76% were Han Chinese, 86.98% were employed, 68.40% had a bachelor's degree or higher, and 76.22% had a household income of 5000 yuan or more. The mean scores for breastfeeding knowledge, attitude, social support and stressful life event were 9.87 ± 4.47, 53.22 ± 5.16, 69.76 ± 10.91, and 5.01 ± 3.92, respectively. During pregnancy, the passive smoking rate was 27.60% and pregnancy complications occurred in 28.30%. At delivery, 54.17% were vaginal delivery, 51.74% of the infants were male, and the mean birth weight was 3.20 ± 0.33 kg. During postpartum period, Exclusive breastfeeding rates at 1, 3, and 6 months postpartum were 48.57%, 56.87%, and 42.23%. Mean infant weights at the corresponding ages were 4.41 ± 0.51 kg, 6.45 ± 0.72 kg, and 8.15 ± 0.90 kg, and the prevalence of RWG was 27.62%, 46.19%, and 53.33%, respectively. The mean cumulative average EPDS scores were similar across time windows, with values of 6.67 ± 3.68 for T1–T4, 6.69 ± 3.62 for T1–T5, and 6.66 ± 3.66 for T1–T6. When categorized into quartiles, cumulative EPDS scores showed a clear gradient, ranging from approximately 2.3 in the lowest quartile to 11.5 in the highest quartile across all time windows.

Table 1
Characteristics of study participants.

Variables	Total Sample (N)	Statistics
Age, years, mean (SD)	576	28.51 (3.21)
Pre-pregnancy BMI, kg/m^2, mean (SD)	576	21.11 (2.92)
Residence, N (%)	576	
Urban		527 (91.49)
Rural		49 (8.51)
Ethnicity, N (%)	576	
Han Chinese		517 (89.76)
Minority ethnic groups		59 (10.24)
Occupation, N (%)	576	
Public institution staff		104 (18.06)
Healthcare worker		44 (7.64)
Self-employed		43 (7.47)
Unemployed		75 (13.02)
Other		302 (52.43)
Farmer/Worker		8 (1.39)
Educational Level, N (%)	576	
≤ High school		40 (6.94)
Junior college		142 (24.65)
Bachelor's degree		333 (57.81)
≥ Master’s degree		61 (10.59)
Household income, yuan (RMB), N (%)	576	
< 3000		17 (2.95)
3000–5000		120 (20.83)
> 5000		439 (76.22)
Passive smoking status, N (%)	576	

Note. T1, First trimester (gestational weeks 7–13); T4, 1 week postpartum; T5, 1 month postpartum; T6, 3 months postpartum. BMI = Body Mass Index; RWG = Rapid weight gain; EPDS = Edinburgh Postnatal Depression Scale; SD=Standard Deviation.

Variables	Total Sample (N)	Statistics
Yes		159 (27.60)
No		417 (72.40)
Pregnancy complications, N (%)	576	
Yes		163 (28.30)
No		413 (71.70)
Breastfeeding knowledge, mean (SD)	480	9.87 (4.47)
Infant feeding attitude, mean (SD)	480	53.22 (5.16)
Social support, mean (SD)	576	69.76 (10.91)
Stressful life events, mean (SD)	480	5.01 (3.92)
Mode of delivery, N (%)	576	
Vaginal delivery		312 (54.17)
Cesarean delivery		264 (45.83
Infant sex, N (%)	576	
Male		298 (51.74)
Female		278 (48.26)
Feeding patterns, N (%)		
1 Month	523	
Exclusive feeding		254 (48.57)
Mixed-feeding		234 (44.74)
Formula-feeding		35 (6.69)
3 Months	415	
Exclusive feeding		236 (56.87)
Mixed-feeding		120 (28.92)
Formula-feeding		59 (14.21)
6 Months	367	
Exclusive feeding		155 (42.23)

Note. T1, First trimester (gestational weeks 7–13); T4, 1 week postpartum; T5, 1 month postpartum; T6, 3 months postpartum. BMI = Body Mass Index; RWG = Rapid weight gain; EPDS = Edinburgh Postnatal Depression Scale; SD=Standard Deviation.

Variables	Total Sample (N)	Statistics
Mixed-feeding		115 (31.34)
Formula-feeding		97 (26.43)
Birth weight, kg, mean (SD)	576	3.20 (0.33)
1-month-old weight, kg, mean (SD)	529	4.41 (0.51)
3-month-old weight, kg, mean (SD)	437	6.45 (0.72)
6-month-old weight, kg, mean (SD)	401	8.15 (0.90)
RWG at 0–1 month, N(%)	529	
Yes		146 (27.60)
No		383 (72.40)
RWG at 0–3 months, N(%)	437	
Yes		205 (46.91)
No		232 (53.01)
RWG at 0–6 months, N(%)	401	
Yes		217 (54.11)
No		184 (45.89)
Cumulative average EPDS score, mean (SD)		
T1_T4	392	6.67 (3.68)
Quartile1		2.35 (1.11)
Quartile2		5.33 (0.74)
Quartile3		7.73 (0.75)
Quartile4		11.59 (2.09)
T1_T5	379	6.69 (3.62)
Quartile1		2.33 (1.13)
Quartile2		5.28 (0.83)
Quartile3		7.72 (0.97)
Quartile4		11.55 (2.22)

Note. T1, First trimester (gestational weeks 7–13); T4, 1 week postpartum; T5, 1 month postpartum; T6, 3 months postpartum. BMI = Body Mass Index; RWG = Rapid weight gain; EPDS = Edinburgh Postnatal Depression Scale; SD=Standard Deviation.

Variables	Total Sample (N)	Statistics
T1_T6	285	6.66 (3.66)
Quartile1		2.37 (1.21)
Quartile2		5.25 (1.08)
Quartile3		7.63 (1.19)
Quartile4		11.46 (2.30)

Note. T1, First trimester (gestational weeks 7–13); T4, 1 week postpartum; T5, 1 month postpartum; T6, 3 months postpartum. BMI = Body Mass Index; RWG = Rapid weight gain; EPDS = Edinburgh Postnatal Depression Scale; SD=Standard Deviation.

Characteristics of the study population after stratification by stage-specific perinatal depressive symptoms and quartiles of cumulative average EPDS scores are presented in Supplementary Tables S2–S4.

3.2 Association between stage-specific perinatal depressive symptoms and infant RWG

Figure 2 presents a comprehensive assessment of the association between stage-specific perinatal depressive symptoms and infant RWG. Compared with the no depressive symptoms group, the antenatal depressive symptoms and persistent depressive symptoms groups showed no significant association with infant RWG at 0–1 month. In the contrast, the postnatal depressive symptoms group showed a significantly higher risk of infant RWG at 0–1month (aOR = 2.10, 95% CI: 1.05–4.17). In addition, antenatal depressive symptoms and postnatal depressive symptoms groups showed no significant association with infant RWG at 0–3 and 0–6 months. The aORs (95% CI) for the persistent depressive symptoms group were 1.89 (1.13–3.18) and 1.75 (1.04–2.93) for infant RWG at 0–3 and 0–6 months, respectively.

3.3 Association between the cumulative average EPDS score and infant RWG

Figure 3 illustrates the association between cumulative perinatal depressive symptoms burden and infant RWG. The cumulative average EPDS score showed no significant associated with infant RWG at 0–1 month. In the contrast, significant associations were observed for RWG over 0–3 and 0–6 months. First, each 1-point increment in the cumulative average EPDS score was significantly associated with a 10% and 7% increase in the odds of risk of infant RWG at 0–3 and 0–6 months, respectively. Second, when analyzed by quartiles of cumulative depressive symptom burden, a significant positive trend was observed, with increasing risks of infant RWG across quartiles (*P* for trend = 0.011 and 0.013, respectively). Although the associations for the intermediate quartiles (Q2 and Q3) did not reach statistical significance, infants born to mothers in the highest quartile of cumulative average EPDS

scores (Q4) had a 139% higher risk of RWG at 0–3 months (aOR = 2.39, 95% CI: 1.24–4.61) and a 110% higher risk at 0–6 months (aOR = 2.10, 95% CI: 1.15–3.86), compared with those in the lowest quartile.

The fully adjusted RCS regression further confirmed a significant positive linear association between cumulative average EPDS scores and infant RWG at 0–3 months of age (*P* for overall = 0.006; *P* for non-linearity = 0.081). For RWG at 0–6 months, a marginally significant positive linear association was observed (*P* for overall = 0.067; *P* for non-linearity = 0.634). No significant association was observed in the RCS analysis for RWG at 0–1 month (*P* for overall = 0.647; *P* for non-linearity = 0.712). The details are presented in Fig. 4.

3.4 Sensitivity analysis

The complete-case sensitivity analysis showed that the overall patterns of associations were generally consistent with those observed in the primary analysis. Although several associations no longer reached conventional statistical significance after restricting the analysis to complete cases, the effect estimates and directions remained largely comparable. In addition, significant associations were still observed in the highest quartile (Q4) of cumulative average EPDS scores and infant RWG at 0–3 months (aOR = 2.49, 95% CI = 1.09–5.72). As the quartiles of cumulative average EPDS scores increased, the risk of RWG in infants aged 0–3 months and 0–6 months still showed a progressively increasing trend (*P* for trend = 0.036 and 0.050, respectively). These findings, to some extent, support the robustness of our main results. The details are presented in Supplementary Table S5.

4. Discussion

To our knowledge, this is the first study to examine both the time-specific and cumulative effects of maternal perinatal depressive symptoms on RWG during early infancy. Our findings suggested a potential dose–response relationship between cumulative depressive symptom burden and the risk of RWG during the first 3 and 6 months of life, with greater cumulative exposure associated with progressively higher risk. Specifically, each one-point increase in cumulative average EPDS score was associated with a 10% and 7% higher risk of RWG during 0–3 and 0–6 months, respectively. Infants born to mothers in the highest quartile of cumulative depressive symptom burden had substantially higher risks of RWG during the same periods. Moreover, exposure window analyses showed that persistent depressive symptoms across the perinatal period, rather than prenatal or postnatal depressive symptoms alone, were associated with increased risk of RWG. These findings highlighted the potential importance of sustained maternal psychological distress in shaping early infant growth patterns. In addition, we identified the postnatal period as a potential critical window during which maternal depressive symptoms were associated with increased risk of RWG in the first month of life.

This study suggested that the risk of RWG increased with greater cumulative exposure to perinatal depressive symptoms. This finding is similar to those of Lampard et al., who reported that chronic depression has a stronger impact on childhood overweight and obesity than episodic depression[25]. The

study provides new insights by examining RWG in the first six months of life. Compared with RWG at 0–1 month occurring over the short period, RWG observed at 0–3 and 0–6 months reflects longer-term changes in growth trajectories. During this period, the effects of cumulative depressive burden may gradually emerge, particularly in metabolic patterns. Cumulative depressive burden may influence RWG at 0–3 and 0–6 months through the combined action of biological and behavioral pathways. From a biological perspective, evidence suggested that maternal perinatal depressive symptoms can disrupt the hypothalamic-pituitary-adrenal (HPA) axis, leading to increased fetal or infant cortisol exposure via placental transfer and breastfeeding[37]. Elevated cortisol levels may alter infant metabolic programming by increasing adipocyte number and size, inducing insulin resistance, and promoting visceral fat accumulation[38]. From a behavioral perspective, in addition to maternal depressive symptoms being associated with non-responsive feeding behaviors that increase infant energy intake[39], the influence of complementary feeding on weight gain may become more evident as infants grow older during 0–3 and 0–6 months. Previous studies have shown that mothers with depressive symptoms are more likely to introduce complementary foods prematurely (< 4 months) for infants[40]. Early introduction of complementary foods may increase energy intake beyond that provided by breast milk or formula and has been associated with RWG in infants[41, 42]. Together, these sustained behavioral changes may continuously provide excess caloric intake. Consequently, a high cumulative depressive burden may lead to alterations in infant metabolic patterns, which, when combined with excess energy supply, are more readily translated into fat accumulation and manifested as RWG.

This study identified the postnatal period as a potential critical window during which perinatal depressive symptoms are associated with increased risk of RWG in the first month of life. While previous studies have predominantly focused on the effects of antenatal depressive symptoms[22], the novel findings of this study highlight the important role of postnatal depressive symptoms in RWG during the neonatal period. This phenomenon may be jointly determined by depression-related changes in maternal caregiving and feeding behaviors and the unique developmental characteristics of infants during the neonatal period. Research suggested that postnatal depressive symptoms increase the likelihood of non-responsive feeding practices, such as pressuring or controlling feeding, which may override infants' hunger and satiety cues and thereby increase the risk of overfeeding[43, 44]. From a developmental perspective, neonatal energy requirements per unit body weight and the proportion devoted to growth are relatively high[45], rendering weight gain highly sensitive to feeding frequency and volume. In addition, compared with older infants, newborns exhibit less diverse and total number of hunger and satiety cues in their behavioral expressions[46]. In this context, caregiving and feeding deviations associated with maternal postnatal depressive symptoms are more likely to be amplified and rapidly reflected in infant weight gain.

This study has important clinical implications. Our findings suggest that maternal perinatal depressive symptoms should be considered in the management of early infant weight, which may be of substantial significance for the early prevention and intervention of childhood overweight and obesity. Specifically,

continuous longitudinal monitoring of perinatal depressive symptoms is warranted, and particular attention should be paid to depressive symptoms during the early postnatal period.

Several limitations of this study should be acknowledged. First, due to the inherent characteristics of the longitudinal study design, a certain degree of missing data was unavoidable. To minimize the potential impact of attrition bias, we conducted loss-to-follow-up analyses and a sensitivity analysis restricted to complete cases, the results generally supported the robustness of the primary findings. It should be noted that some associations did not reach statistical significance in sensitivity analysis, which may be attributable to reduced statistical power resulting from the smaller number of complete cases. Second, all participants in this study were recruited from a single hospital in Hunan Province, China, which may limit the representativeness of the sample. However, this hospital is a designated national-level regional center for obstetric and gynecologic care, serving a relatively broad population base, which may partially enhance the generalizability of our findings. Third, although the cumulative average measure used in this study effectively captured the overall burden of depressive symptoms over time, it did not characterize specific symptom trajectories. Different trajectories of depressive symptoms may be differentially associated with the risk of RWG in infancy, which warrants further investigation in future studies.

5. Conclusion

This study demonstrated that perinatal depressive symptoms were significantly associated with an increased risk of RWG during the first 6 months of life. A potential linear dose–response relationship was observed between cumulative depressive symptom burden and infant RWG. Specifically, each one-point increase in cumulative average EPDS score was associated with a 10% and 7% higher risk of RWG during 0–3 and 0–6 months, respectively. Infants born to mothers with the highest cumulative depressive burden had a 139% and 110% greater risk of RWG over the same periods. In addition, the postnatal period appeared to represent a critical window during which maternal depressive symptoms were associated with increased risk of RWG in the first month of life. These findings support the inclusion of maternal mental health in early-life weight management and childhood obesity prevention strategies. They also underscore the importance of continuous monitoring of depressive symptoms throughout the perinatal period, particularly during the early postnatal stage. Further studies are needed to determine whether alleviating perinatal depressive symptoms may help prevent RWG in infancy.

Declarations

Competing Interests statement

The authors declare no conflicts of interest.

Author Contributions

The authors' responsibilities were as follows – SF, LL, YW, MZ: designed research; LL, MZ, JZ: conducted research; WH, JL, CQ: provided essential reagents, or provided essential materials; SF, YW: analyzed data or performed statistical analysis; SF: wrote paper and had primary responsibility for final content; and all authors: reviewed and contributed to the final manuscript.

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Data availability statement

Data described in the manuscript will be made available upon request pending application and approval.

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Figures

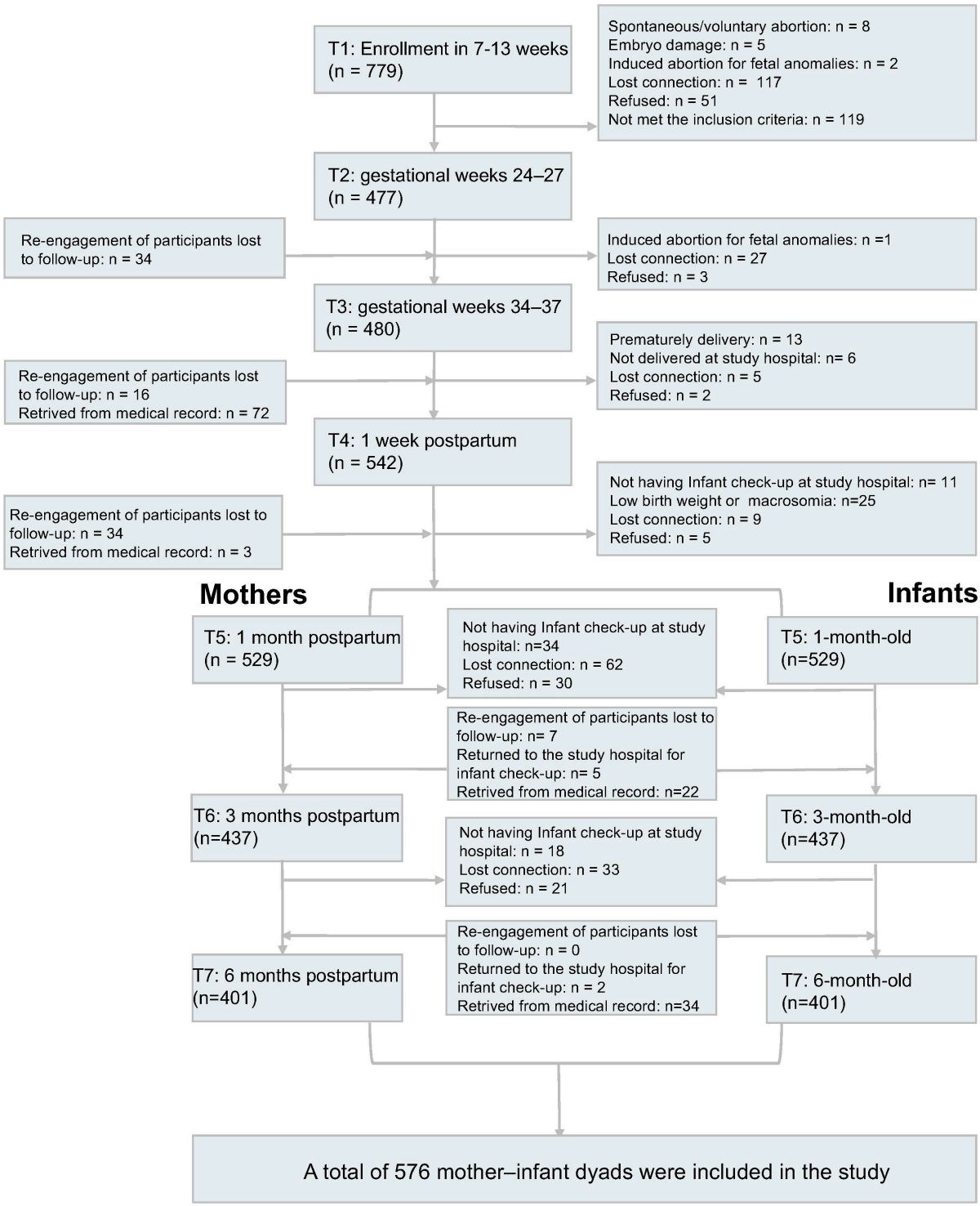


Figure 1

Inclusion and exclusion process of participants.

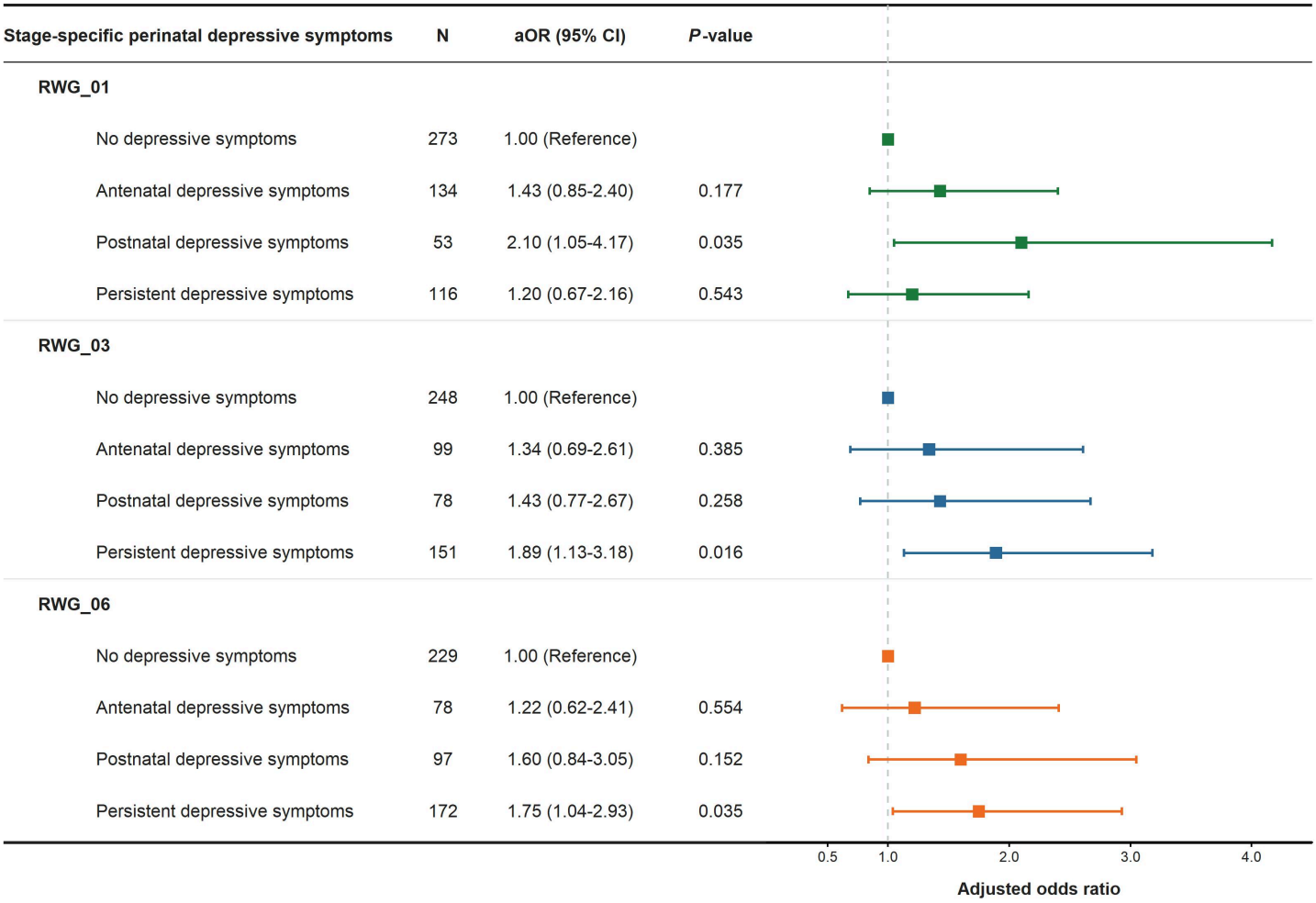


Figure 2

Associations of stage-specific perinatal depressive symptoms with infant rapid weight gain.

RWG_01, RWG_03, and RWG_06 refer to rapid weight gain during the first month, first 3 months, and first 6 months of life, respectively. The model for RWG_01 was adjusted for pregnancy BMI, passive smoking status, breastfeeding knowledge, social support, stressful life events, mode of delivery, birth weight, infant sex, feeding patterns. The model for RWG_03 was adjusted for pregnancy BMI, educational level, passive smoking status, breastfeeding knowledge, social support, stressful life events, mode of delivery, birth weight, infant sex, feeding patterns. The model for RWG_06 was adjusted for pregnancy BMI, breastfeeding knowledge, social support, stressful life events, birth weight, infant sex, feeding patterns. aOR = Adjusted Odds Ratio, CI = Confidence Interval.

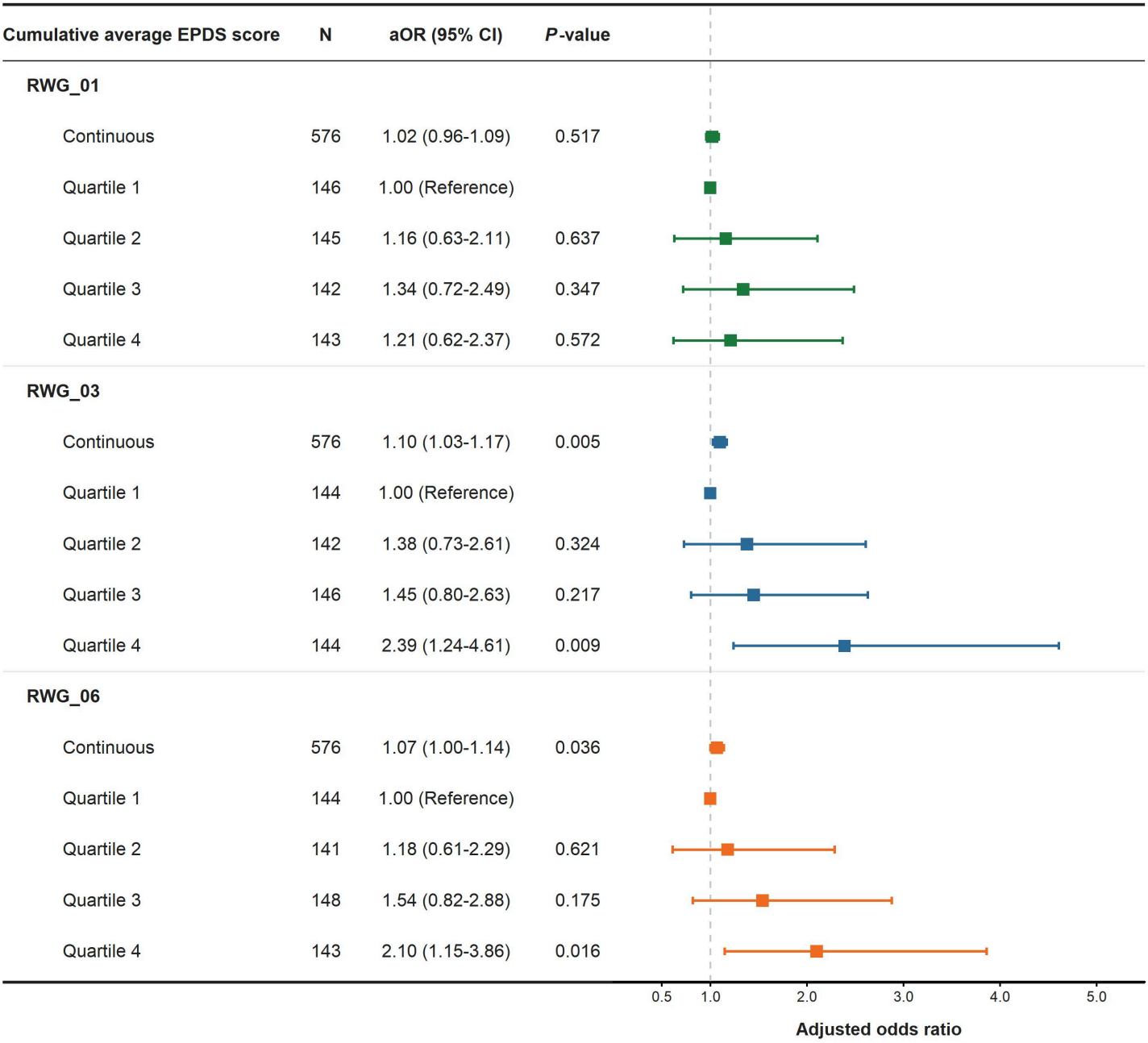


Figure 3

Associations of cumulative depressive symptom burden with infant rapid weight gain.

RWG_01, RWG_03, and RWG_06 refer to rapid weight gain during the first month, first 3 months, and first 6 months of life, respectively. The model for RWG_01 was adjusted for pregnancy BMI, passive smoking status, breastfeeding knowledge, social support, stressful life events, mode of delivery, birth weight, infant sex, feeding patterns. The model for RWG_03 was adjusted for pregnancy BMI, educational level, passive smoking status, breastfeeding knowledge, social support, stressful life events, mode of delivery, birth weight, infant sex, feeding patterns. The model for RWG_06 was adjusted for pregnancy BMI, breastfeeding knowledge, social support, stressful life events, birth weight, infant sex, feeding patterns. EPDS = Edinburgh Postnatal Depression Scale, aOR = Adjusted Odds Ratio, CI = Confidence Interval.

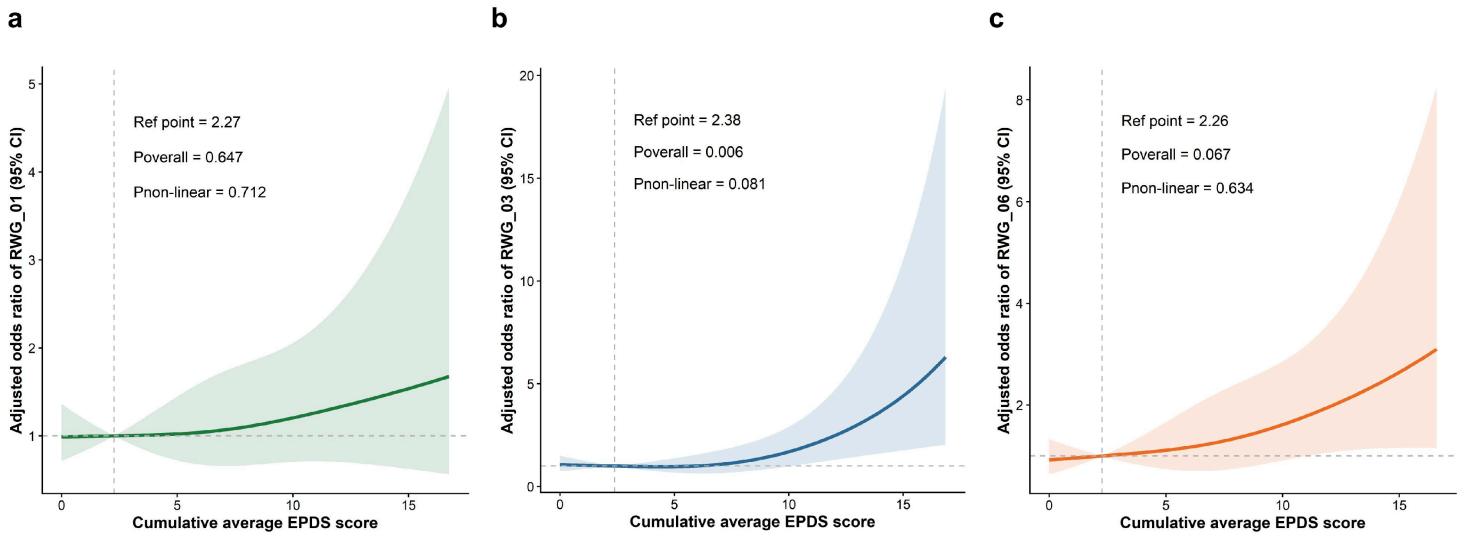


Figure 4

Restricted cubic spline analysis of the association between cumulative average EPDS score and infant rapid weight gain at 0–1 (A), 0–3 (B), and 0–6 (C) months.

RWG_01, RWG_03, and RWG_06 refer to rapid weight gain during the first month, first 3 months, and first 6 months of life, respectively. EPDS = Edinburgh Postnatal Depression Scale, CI = Confidence Interval.

Supplementary Files

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