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Authors

Rinne, Gabrielle R
Davis, Elysia Poggi
Mahrer, Nicole E
et al.

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Maternal depressive symptom trajectories from preconception through postpartum: Associations with offspring developmental outcomes in early childhood

Gabrielle R. Rinne[a,*], **Elysia Poggi Davis**[b,c], **Nicole E. Mahrer**[d], **Christine M. Guardino**[e], **Julia M. Charalel**[f], **Madeleine U. Shalowitz**[g], **Sharon L. Ramey**[h], **Christine Dunkel Schetter**[a]

[a]Department of Psychology, University of California, Los Angeles, Los Angeles, CA, United States of America

[b]Department of Psychology, University of Denver, Denver, CO, United States of America

[c]Department of Psychiatry & Human Behavior, University of California, Irvine, Irvine, CA, United States of America

[d]Psychology Department, University of La Verne, La Verne, CA, United States of America

[e]Department of Psychology, Dickinson College, Carlisle, PA, United States of America

[f]Department of Psychiatry & Behavioral Sciences, University of California, San Francisco, San Francisco, CA, United States of America

[g]Department of Pediatrics, Rush University Medical Center, Chicago, IL, United States of America

[h]Fralin Biomedical Research Institute, Department of Psychology, Departments of Psychiatry and Pediatrics, Virginia Tech, Roanoke, VA, United States of America

Abstract

Background: Two theoretical frameworks, the *cumulative stress* and *match-mismatch model*, propose that patterns of maternal depressive symptoms over early periods of offspring development predict outcomes in opposing ways. Studies have yet to test these theories across the preconception, prenatal, and early postnatal period. Study 1 identified trajectories of maternal depressive symptoms from preconception to postpartum. Study 2 examined associations of these trajectories with offspring developmental outcomes in early childhood.

[*]Corresponding author at: Department of Psychology, 1285 Franz Hall, Los Angeles, CA 90095, United States of America. gabrielle.rinne@ucla.edu (G.R. Rinne).

Declaration of competing interest

The authors declare no conflict of interests.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.jad.2022.04.116.

CRediT authorship contribution statement

GRR conceptualized the research problem with guidance from EPD, performed data analysis, interpreted results, and drafted the paper. CDS supervised the process and provided feedback at every stage. CMG and NEM were project coordinators of the child follow-up study and CMG was involved in the design and conduct of both studies. EPD and NEM provided feedback on data analysis and paper drafts. JMC provided critical review of the manuscript. MUS and SRM were principal investigators (Pis) of the CCHN and Co-PIs of the follow-up study. CDS was PI of the follow-up study and Co-PI of the CCHN. All authors contributed to and approved the final paper.

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Methods: In Study 1, women (n = 362) enrolled in a longitudinal study were assessed prior to conception and through a subsequent pregnancy and postpartum. In Study 2, a subsample of 125 mother-child pairs completed home visits in early childhood. Mothers reported on child temperament at age 4. Children completed assessments of executive function at age 5.

Results: Four trajectories of maternal depressive symptoms were identified: *low-stable, increasing, decreasing, persistent*. In controlled analyses, children of women with *decreasing* symptoms were lower in maternal ratings of effortful control at age four (β = −0.24, p = .003). Children of women with *increasing* symptoms scored lower on an inhibitory control task at age five (β = −0.35, p = .001).

Conclusions: Changes in maternal depressive symptoms, but not stable symptoms, were associated with lower maternal ratings of effortful control and poorer performance on an inhibitory control task. Results are consistent with the *match-mismatch model*. Assessment of preconception depressive symptoms in women and changes in symptoms may be beneficial for early intervention for women and children.

Keywords

Maternal mental health; Preconception; Pregnancy; Postpartum; Child development; Psychopathology

1. Introduction

Pregnancy and postpartum are periods of heightened susceptibility to depressive symptoms among women, with up to 20% of women experiencing clinically significant symptoms (Gavin et al., 2005). Perinatal depression is a pressing public health concern given the adverse implications for short- and long-term maternal health, child health, and family functioning (Meaney, 2018). For example, exposure to maternal depressive symptoms in the prenatal and early postnatal period is associated with increased behavior problems, emotional problems, and risk for psychopathology in offspring independently of exposure to maternal depressive symptoms later in development (Davis and Sandman, 2012; Kingston and Tough, 2014; Rogers et al., 2020; Sandman et al., 2011; Tirumalaraju et al., 2020). Emerging evidence indicates that maternal mental health *prior to* becoming pregnant (i.e., preconception) is associated with maternal perinatal health and offspring development, including both in the proximal preconception period six to twelve months before pregnancy as well as distal experiences in a mother's own childhood and adolescence (Keenan et al., 2018; Spry et al., 2020). Models of changes in depressive symptoms across preconception, pregnancy, and postpartum could elucidate how exposure to maternal depressive symptoms over time influences offspring development. However, no studies have examined trajectories of symptoms across these periods or tested associations with offspring developmental outcomes.

Maternal depressive symptoms during pregnancy and postpartum are influenced by a range of psychosocial and biological factors such as stressors, social support, and genetic vulnerability (Meltzer-Brody, 2011). Accordingly, trajectories of depressive symptoms over pregnancy and postpartum are heterogeneous (Baron et al., 2017; Santos et al., 2017). Most

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women report low stable symptoms while others report persistently high levels, increasing symptoms, or decreasing symptoms over the perinatal period. A life course perspective proposes that maternal well-being during and after pregnancy is influenced by the mental and physical health of women prior to becoming pregnant (Bernstein and Merkatz, 2010; Keenan et al., 2018). For example, a recent study found that depressive symptoms remained stable from preconception to the second trimester for most women (Kee et al., 2021). Like during the perinatal period, however, women may show individual heterogeneity in trajectories of depressive symptoms from preconception through postpartum, yet studies have not examined symptom trajectories across this period. The preconception period is an optimal time for early screening and intervention to prevent adverse maternal and infant outcomes (Atrash et al., 2006; Lu and Halfon, 2003). Therefore, future research in this area is warranted.

Trajectories of maternal depressive symptoms across critical developmental periods have implications for child development. Patterns of maternal depressive symptoms during early development may contribute to child health and well-being through various biological, affective, social, and genetic pathways (Goodman and Gotlib, 1999). To date, two dominant theoretical frameworks have guided understanding of how exposure to maternal stress and distress across early periods of offspring development influence offspring outcomes (Conradt et al., 2018; Nederhof and Schmidt, 2012). First, the *cumulative stress model* posits that offspring exposure to psychosocial stress over time, including maternal depressive symptoms, cumulatively affects and disrupts offspring development (Shonkoff et al., 2012). According to this model, persistently high maternal depressive symptoms over pregnancy and postpartum contribute to less favorable offspring development.

Second, the *match-mismatch model* puts forth that the in-utero environment guides development to be prepared for the postnatal environment. Thus, adaptive offspring development occurs through matches between the environment early in development and later in development (Chan et al., 2018; Gluckman et al., 2005; Sandman et al., 2012). Accordingly, stable maternal depressive symptoms over time would contribute to more favorable offspring development, even if symptoms are high. Conversely, significant changes in symptoms over time would result in less favorable child development because early developmental adjustments may not match the demands of the later environment. For example, high maternal depressive symptoms early in the prenatal period may alter fetal developmental trajectories to prepare the fetus for a potentially adverse postnatal environment. Such adjustments may be adaptive if maternal depressive symptoms remain stable through the postpartum period but may be less adaptive if symptoms increase or decrease (Sandman et al., 2012). Examining trajectories of maternal depressive symptoms allows for the comparison of the *cumulative stress* and *match-mismatch* models and can elucidate how exposure to maternal depressive symptoms over time influences offspring development.

Recent studies examining trajectories of maternal depressive symptoms over pregnancy and postpartum have supported both the *cumulative stress* and *match-mismatch model* in relation to offspring socioemotional problems and internalizing and externalizing behaviors (Fransson et al., 2020; Guyon-Harris et al., 2016; Kingston et al., 2018; Park et al.,

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2018). Of note, several of these studies have reported effects of symptom trajectories independent of maternal depressive symptoms at the time offspring developmental outcomes are assessed (Fransson et al., 2020; Kingston et al., 2018). Research indicates that levels of maternal depressive symptoms during pregnancy and postpartum are associated offspring temperament and executive function (Kingston and Tough, 2014; Rogers et al., 2020). Studies have yet to test whether changes in maternal depressive symptoms over pregnancy and postpartum are associated with early childhood temperament or executive function. Temperament reflects dispositional differences in reactivity and regulation and is often classified into distinct dimensions of effortful control, negative affect, and surgency (Rothbart et al., 2000). Executive function refers to an interrelated set of top-down cognitive processes integral to self-regulation and control of goal-directed behavior, involving both cognitive flexibility and cognitive control (Zelazo, 2020). These cognitive processes are related to parent report of effortful control in temperament assessments. There is evidence that parent report of effortful control and performance on executive function tasks are related and reflect overlapping self-regulation processes (Beck et al., 2011; Bridgett et al., 2013; Davis et al., 2019; Rothbart et al., 2007; Zhou et al., 2012). Of note, high negative affect and low effortful control/poor executive function in early childhood are transdiagnostic risk factors for psychopathology across the lifespan (Nigg, 2006; Zelazo, 2020), thus it is important to elucidate factors early in development that contribute to these domains.

Despite increasing evidence that the preconception period is another important period for maternal and child health (Keenan et al., 2018), no studies have included assessment of maternal depressive symptoms prior to conception when identifying trajectories. There is growing evidence that mental health of women prior to conception is associated with uterine priming, zygote implantation, early placental development, and subsequent fetal development (Chan et al., 2018). For example, maternal stress and distress prior to conception has been associated with birth and developmental outcomes, including shorter length of gestation (Mahrer et al., 2020a), lower birth weight (Guardino et al., 2016), increased behavioral reactivity in infancy (Spry et al., 2020), and more attention problems in early childhood (Class et al., 2014). Based on this evidence, examining trajectories of maternal depressive symptoms from preconception through postpartum is warranted to extend existing tests of the *cumulative stress* and *match-mismatch model*.

1.1. Current study

The current study extends previous research by (1) identifying trajectories of maternal depressive symptoms beginning prior to conception through three months postpartum, and (2) testing associations of these trajectories with maternal ratings of child effortful control and negative affect as well as child performance on executive function assessments. A racially and ethnically diverse and mostly low income cohort of women were recruited after a birth and followed through a subsequent pregnancy and postpartum period, a subset of whom completed additional home assessments of the subsequent child in early childhood. The current study defines *preconception* as prior to conception of the subsequent child (i.e., the study child), which for all participants in the sample was also an interconception period between consecutive births.

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In Study 1, our aim was to identify trajectories of maternal depressive symptoms across preconception, pregnancy, and postpartum in the cohort of women who completed visits prior to conception through postpartum. We hypothesized that between 2 and 5 trajectories would emerge, consistent with previous research on depressive symptom trajectories in pregnancy and postpartum (Baron et al., 2017; Santos et al., 2017). In Study 2, our aim was to test associations of these trajectories with maternal ratings of effortful control and negative affect at age 4 as well as assessments of executive function (inhibitory control and cognitive flexibility) at age 5 in the subsample of mother-child pairs that completed additional visits in early childhood. Findings consistent with the *cumulative stress model* would indicate that persistently high maternal depressive symptoms from preconception through postpartum predict higher maternal ratings of negative affect, lower maternal ratings of effortful control, and poorer performance on assessments executive function. Conversely, findings in support of the *match-mismatch model* would indicate that significant changes in maternal symptoms from preconception through postpartum predict higher maternal ratings of child negative affect, lower maternal ratings of child effortful control, and poorer performance on assessments of executive function. This study provides novel data addressing these models.

As a post-hoc analysis in Study 2, we examined whether continuous depressive symptoms at each timepoint were associated with offspring developmental outcomes to test whether associations of symptom trajectories and offspring developmental outcomes were due to symptom levels at one timepoint.

2. Methods

2.1. Participants and procedure

2.1.1. Study 1: Community Child Health Network—Participants in Study 1 were 362 women enrolled in the Community Child Health Network (CCHN). Women of African American/Black, Hispanic/Latina, and non-Hispanic White identity were recruited at five study sites (Lake County, IL; Washington, D.C.; eastern North Carolina; Los Angeles County, CA; Baltimore, MD) following a birth and were followed through a subsequent pregnancy and postpartum period. CCHN study visits occurred after the earlier birth and prior to conception of a subsequent pregnancy (preconception visit), during the second trimester (M = 20.4 weeks, SD = 5.5) and third trimester (M = 33.0 weeks, SD = 4.5), and postpartum (M = 12.1 weeks, SD = 5.7). The preconception visit took place within one year prior to the date of conception of the subsequent pregnancy for most participants (84.5%). On average, the preconception visit took place 7.58 months (SD = 7.07 months) prior to conception but ranged (range = 0.47 month–42.1 months), as is consistent with prior studies in human samples (Keenan et al., 2018).

2.1.2. Study 2: child follow-up study—Participants in Study 2 were a subset of women recruited to CCHN from three study sites (Lake County, IL.; Washington, D.C.; eastern North Carolina) who enrolled in a longitudinal follow-up study of the subsequent child's development. That is, children included in the follow-up study were those whom the mother was pregnant with during the subsequent pregnancy (study child). Mother-child pairs

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(*n* = 125) enrolled in the follow-up study completed additional home visits twice in early childhood. The first was when the study child was approximately 4 years of age (M = 3.83 years, SD = 0.41) and the second was one year later (M = 5.07 years, SD = 0.53).

Trained researcher staff conducted structured in-home interviews in English or Spanish with women based on family preference. Institutional Review Boards at each site approved the study. All participants provided written and informed consent and were compensated for each visit. Further details on CCHN and the follow-up study are reported elsewhere (Mahrer et al., 2020a, 2020b; Morgan et al., 2020; Ramey et al., 2015).

2.2. Measures

2.2.1. Sociodemographic and medical variables—Women reported their racial/ethnic identity, age, and education level at the time of CCHN enrollment and reported updates to household income, household size, and their relationship with partner at each study visit. Per capita income was calculated by dividing household income by household size and adjusted for cost of living at each study site. Data on birth weight and gestational age at birth of the study child were abstracted from medical records. Women in the follow-up study reported on study child age, child sex, and child racial/ethnic identity at the first early childhood visit.

2.2.2. Depressive symptoms—Two instruments were used to assess maternal depressive symptoms that were chosen to be appropriate to the time periods when administered. First, the Edinburgh Postpartum Depression Scale (EPDS) was used to assess depressive symptoms postpartum after the first birth during the period prior to conception of the study child as well as postpartum after the subsequent birth (Cox et al., 1987). The EPDS is validated for use in the first year postpartum in Spanish and English (Garcia-Esteve et al., 2003). The EPDS consists of 10 items that assess severity of depressive symptoms over the past week on a scale of 0 to 3 (total scores 0–30). Next, the Center for Epidemiological Studies Depression Scale 9-item version (CES-D) was administered in the second and third trimester of the subsequent pregnancy and at the home visits during early childhood (Santor and Coyne, 1997). The CES-D is a widely used self-report measure in the general population and is validated for use in pregnancy (Marcus et al., 2003). The CES-D assessed frequency of symptoms in the past week on a scale of 1 (*none of the days*) to 4 (*most or all days*) (total scores 9–36). Cronbach's alpha indicated acceptable reliability for the EPDS (α = 0.82–0.83) and CES-D (α =0.72–0.83). There is substantial overlap between the EPDS and CES-D as demonstrated in prior studies in perinatal samples (Tandon et al., 2012).

2.2.3. Offspring developmental outcomes

2.2.3.1. Maternal ratings of child temperament.: Maternal ratings of child effortful control and negative affect were obtained via the Children's Behavior Questionnaire Very Brief version when children were approximately 4 years old (CBQ) (Putnam and Rothbart, 2006). The CBQ is widely used and has demonstrated validity and reliability. Subscale scores are based on an average of the 12 items for each subscale (total scores 1–7). Sixty-nine percent of mothers reported on their child's temperament in English and there were no significant differences in scores by language. Reliability in the current sample was

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consistent with reliability in other samples (effortful control α = 0.72, negative affect α = 0.64) (Putnam and Rothbart, 2006).

2.2.3.2. Assessments of executive function.: Domains of executive function were assessed with the Early Childhood version of the NIH Toolbox Cognition Battery (ages 3–6) (Weintraub et al., 2013) when children were approximately 5 years old. Cognitive flexibility and inhibitory control were assessed with the Dimensional Change Card Sort Task and Flanker Inhibitory Control and Attention Task, respectively (Morgan et al., 2020). The NIH Toolbox has excellent reliability and validity in Spanish and English (Weintraub et al., 2013). About three-quarters (77.4%) of children in the current sample completed the assessments in English and there were no significant differences in scores by assessment language. Domains are scored using fully corrected T-scores adjusting for child age, sex, race/ethnicity, and maternal education. Cognitive flexibility and inhibitory control scores were moderately associated in the current sample ($r = 0.36$, $p < .001$).

2.3. Statistical analysis

Data analyses were conducted in MPlus (Muthén and Muthén, 1998-2017) and R (R Core Team, 2021). Primary study variables were examined for non-normality (skewness greater than 3 or kurtosis greater than 7) and outliers (more than three standard deviations from mean) prior to analysis. None of the primary study variables violated normality assumptions and there were no meaningful differences in results with outliers winsorized. Therefore, models are presented with untransformed variables. Full information maximum likelihood with robust standard errors was used to handle missing data in primary analyses for Study 1 and Study 2 (Enders, 2010).

2.3.1. Study 1. Maternal depressive symptom trajectories in CCHN cohort—

We used unconditional latent curve growth analysis with latent trajectories (i.e., without covariates) to identify symptom trajectories based on standardized (*z*-scores) depressive symptoms at four timepoints: prior to conception, in the second trimester, in the third trimester, and postpartum. Measures of depressive symptoms were standardized prior to analysis to account for differences in measures of depressive symptoms across study visits. This method expands from conventional growth modeling and allows for examination of individual growth trajectories within a population (Jung and Wickrama, 2008). In contrast to conventional growth modeling in which heterogeneity is captured by random slopes and intercepts, latent curve growth analysis captures heterogeneity by identifying distinct subgroups within a population such that individuals within a group are more similar than individuals between groups. Accordingly, the variance and covariance estimates within each class are fixed at zero by default (Jung and Wickrama, 2008). Latent curve growth analysis can be conducted when measurement points are separated by different timepoints as well as vary across individuals (Andruff et al., 2009).

We compared models fitting one to five classes successively. The best fitting model was selected based on Bayesian Information Criteria (BIC; lowest preferred), entropy (higher preferred), and bootstrapped parametric likelihood ratio test, which compares whether model with *k* classes is a significant improvement of model with *k*-1 classes (Jung and

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Wickrama, 2008). Participants were assigned to trajectories based on posterior probabilities. Average posterior probabilities greater than 0.70 indicate the trajectories adequately grouped individuals with similar patterns and discriminated individuals with dissimilar patterns (Andruff et al., 2009).

After latent trajectories were identified using unconditional latent curve growth analysis, we tested whether women in each symptom trajectory differed by sociodemographic characteristics using one-way ANOVAs and Fisher's Exact Tests. We conducted follow-up pairwise comparisons of significant omnibus ANOVAs with Tukey's correction to adjust for multiple comparisons.

2.3.2. Study 2. Associations of trajectories with offspring developmental outcomes in follow-up cohort—

Using the latent trajectories from the best fitting model identified in Study 1, we ran a series of multivariate linear regression models to examine associations of trajectories with maternal ratings of child temperament at age 4 and child performance on executive function tasks at age 5. Symptom trajectories were dummy coded and simultaneously entered in the multivariate linear regression models (1 model for each offspring developmental outcome). We conducted four separate regression models for primary adjusted analyses.

2.3.2.1. Post-hoc multiple testing correction.: We applied a post-hoc Bonferroni correction to control for an inflated Type I error rate with multiple testing for our primary adjusted analyses in Study 2. The adjusted alpha level for the multiple testing correction was 0.013 (0.05/four total multiple linear regression model). A power analysis was conducted for the primary adjusted multiple linear regression analyses in Study 2 using G*Power 3 (Erdfelder et al., 2009). Power analyses indicated that the sample size for Study 2 was sufficient to detect the recommended minimum effect size for an effect having potentially clinical or practical significance ($\beta = 0.20$; Ferguson, 2009) at 80% power with an adjusted alpha level of 0.013. A minimum sample size of 98 would be required indicating this sample size of 125 was adequate.

2.3.2.2. Post-hoc analysis.: We ran a series of multiple linear regression models with continuous depressive symptoms at each timepoint to examine whether associations of symptom trajectories and offspring developmental outcomes were due to symptom levels at one timepoint. Maternal depressive symptoms prior to conception, in the second trimester, in the third trimester, and postpartum were entered into the same model for each developmental outcome. We applied the same post-hoc Bonferroni correction for the post-hoc analysis (adjusted alpha level = 0.05/four post-hoc multiple linear regression models = 0.013).

3. Results

3.1. Study 1: trajectories of maternal depressive symptoms in the CCHN cohort

3.1.1. Descriptive statistics—

Complete sample characteristics for women in the larger CCHN cohort appear in Table 1. Mean maternal age at enrollment was 25 years (M = 25.2, SD = 5.7). Women identified as African American/Black (44.8%), Hispanic/Latina (29.3%), and Non-Hispanic White (26.0%). Over half of the sample was below the federal

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poverty line (53.9%) and the modal level of educational attainment in both cohorts was high school diploma or GED. A majority of women were married (38.3%) or cohabiting with a partner (31.7%) one year after the earlier birth. Less than one-third of the sample was single (30.0%).

Mean levels of maternal depressive symptoms at the preconception visit (M = 4.61, SD = 4.64) were similar to but slightly higher than depressive symptoms at the postpartum visit (M = 3.96, SD = 4.48), as measured by the EPDS. Moreover, mean levels of depressive symptoms were similar in the second (M = 18.10, SD = 6.35) and third trimester of pregnancy (M = 17.83, SD = 5.59), as assessed by the CES-D. Preconception depressive symptoms were modestly to moderately associated with depressive symptoms in the second trimester ($r = 0.31$, $p = .001$), third trimester ($r = 0.28$, $p < .001$), and postpartum ($r = 0.43$, $p < .001$).

3.1.2. Trajectories of maternal depressive symptoms—We compared models fitting one to five classes successively using unconditional latent curve growth analysis. The four class model was identified as the best fitting model based on the BIC, entropy, and the bootstrapped likelihood parametric test (Table 2). Class 1 (*low-stable*; 74.3%) included participants who reported low symptoms that did not significantly change over time. Class 2 (*increasing*; 11.9%) included participants who reported low symptoms prior to conception that significantly increased over time. Class 3 (*decreasing*; 6.9%) included participants who reported high symptoms prior to conception that decreased over time. Class 4 (*persistent*; 6.9%) included participants who reported high symptoms prior to conception that remained high during pregnancy and postpartum. Trajectories for the four class model are visually presented in Fig. 1. The average posterior probability for each class indicated each trajectory adequately grouped individuals with similar patterns of symptoms and distinguished between individuals with dissimilar patterns of symptoms (Class 1 M = 0.92, SD = 0.13; Class 2 M = 0.75, SD = 0.19; Class 3 M = 0.73, SD = 0.18; Class 4 M = 0.79, SD = 0.19; Andruff et al., 2009). Descriptions and visual presentations of the two class, three class, and five class model can be found in the Supplemental Materials. Mean CES-D and EPDS scores for the full sample and by predicted trajectory membership are visually presented in the Supplemental Materials.

There were several sociodemographic differences between symptom trajectories based on results of one-way ANOVAs and Fisher's Exact Tests (Table 1). Women with *persistent* symptoms were significantly less likely to be married, more likely to be cohabiting, and less likely to identify as Non-Hispanic White than would be expected by chance. Women with *decreasing* symptoms were also significantly less likely to be married than would be expected by chance and completed fewer years of education than women with *low-stable* symptoms (M difference = −1.32, $p = .09$).

3.2. Study 2: associations with offspring developmental outcomes in child follow-up study

3.2.1. Descriptive statistics—Complete sample characteristics of women included in the Study 2 analyses are presented in Table 1. Like the full CCHN cohort, women in

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the follow-up study were racially and ethnically diverse and mostly low income. Mean maternal age at the first early childhood visit was 33 years (SD = 6.33). Half of the study children were female (53.6%) and half were the second-born child (50.4%). Women reported their children's racial/ethnic identity as Hispanic or Latino (45.5%), non-Hispanic White or Caucasian (26.8%), African American or Black (18.7%), and multi-racial (8.9%).

Table 3 presents descriptive statistics and bivariate Pearson's correlation coefficients between levels of maternal depressive symptoms at each study visit and offspring developmental outcomes in early childhood. Consistent with prior studies (e.g., Davis et al., 2019), maternal ratings of child effortful control at age 4 and child performance on executive function tasks at age 5 were positively associated. Greater maternal depressive symptoms in the second trimester, postpartum, and at the first early childhood visit were associated with higher maternal ratings of negative affect at age 4. Greater maternal postpartum depressive symptoms were associated with poorer performance on the inhibitory control task at age 5.

In the subsample of participants enrolled in the follow-up study, women with *low-stable* symptoms from preconception through postpartum reported significantly lower depressive symptoms when children were age 4 ($F(3,119) = 11.37$, $p < .001$) compared to women with *decreasing* symptoms (M difference = −5.92, adjusted $p = .002$) and women with *persistent* symptoms (M difference = −7.65, adjusted $p < .001$). The pattern of results remained the same for depressive symptoms when children were age 5.

3.2.2. Covariates—Analyses for Study 2 adjusted for covariates. Maternal depressive symptoms at the early childhood visit when child effortful control and negative affect were assessed were included as a covariate for models examining maternal ratings of negative affect and effortful control as outcomes. A composite of maternal depressive symptoms at both early childhood visits was included as a covariate for models examining child performance on executive function assessments as outcomes. These were included as covariates to test whether associations between symptom trajectories and offspring developmental outcomes were independent of maternal depressive symptoms at the time developmental outcomes were assessed, consistent with past research (Fransson et al., 2020; Kingston et al., 2018).

Table 4 presents additional variables evaluated as covariates for Study 2. Negative affect and effortful control models retained covariates that were significantly associated ($\alpha = 0.05$) with maternal ratings of child temperament at age 4. Child sex, maternal racial/ethnic identity, and per capita income were included as additional covariates in models examining maternal ratings of child temperament as an outcome. None of the additional covariates evaluated met inclusion criteria for assessments of inhibitory control or cognitive flexibility.

3.2.3. Tests of cumulative stress and match-mismatch models of early development—Table 5 presents complete results for Study 2. Unadjusted models are presented only for comparison to adjusted models. The *low-stable* trajectory was used as the reference group for linear regression analyses. When adjusting for covariates, children of women with *decreasing* symptoms were significantly lower in maternal ratings of effortful control at age 4 and scored significantly lower on the inhibitory control task at age 5

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compared to children of women with low-stable symptoms. Children of women with *increasing* symptoms also scored significantly lower on the inhibitory control task at age 5 compared to women with *low-stable* symptoms. There were no significant associations between maternal depressive symptom trajectories and maternal ratings of child negative affect or child performance on the cognitive flexibility task. These results provide evidence in support of the *match-mismatch model* because changes were associated with lower maternal ratings of effortful control and scores on an inhibitory control task whereas stable symptoms were not.

3.2.3.1. Post-hoc multiple testing correction.: When applying a post-hoc family-wise Bonferroni correction for multiple testing in primary adjusted analyses (corrected alpha = 0.013), results of Study 2 remained the same except the association between *decreasing* symptoms and offspring performance on the inhibitory control task at age 5 was no longer statistically significant.

3.2.3.2. Results of post-hoc analysis.: The post-hoc analysis retained the same covariates as in the Study 2 models (child sex, maternal racial/ethnic identity, per capita income, maternal depressive symptoms in early childhood for temperament; maternal depressive symptoms in early childhood for domains of executive function). Multiple linear regression models examining associations of depressive symptom levels with developmental outcomes revealed that greater preconception depressive symptoms were associated with lower maternal ratings of child effortful control at age 4 independently of symptoms at all other study timepoints (B = −0.04 [95% CI —0.07, 0.00], β = −0.19, $p = .07$). This association was not statistically significant. Furthermore, greater postpartum depressive symptoms were associated with lower scores on a child inhibitory control task at age 5 independently of symptoms at all other timepoints (B = −0.81 [95% CI −1.48, −0.14], β = −0.35, $p = .02$). However, this association was not statistically significant when adjusting for multiple tests (adjusted alpha = 0.013). Depressive symptom levels were not associated with child negative affect or cognitive flexibility.

4. Discussion

The time before conception has been recognized as a critical period for maternal and child health. Preconception mental health has implications for the mood and well-being of mothers during pregnancy and postpartum and for offspring development (Keenan et al., 2018). However, studies rarely assess maternal preconception mental health symptoms, and none to our knowledge have examined patterns of maternal depressive symptoms from preconception through postpartum. The current study builds on previous research by identifying trajectories of maternal depressive symptoms from preconception through postpartum and testing associations of these trajectories with offspring developmental outcomes.

We identified four distinct trajectories of depressive symptoms over this period labeled as *low-stable, persistent, increasing*, and *decreasing*. Most women reported low, stable symptoms from preconception through postpartum, providing further evidence of the value of a life course approach to maternal mental health. Trajectories of symptoms

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characterized by significant changes were associated with maternal ratings of temperament and performance on an executive function task in early childhood. Children of women who reported changes in symptoms from preconception through postpartum were lower in domains reflecting ability to regulate behavior, emotion, and attentionage 4 and 5 measured by both maternal report and behavioral assessment. Specifically, children of women with *decreasing* symptoms were lower in maternal ratings of effortful control at age 4. Children of women with *increasing* symptoms were lower in performance on an inhibitory control at age 5. In a post-hoc analysis, levels of maternal depressive symptoms prior to conception and postpartum were associated with lower maternal ratings of effortful control at age 4 and lower scores on the inhibitory control task at age 5, respectively. These findings support the *match-mismatch model* of early development because changes in depressive symptoms from preconception through postpartum were associated with offspring development, but stable symptoms were not (Gluckman et al., 2005). The present results have implications for offspring given associations of low effortful control/poor executive function in early childhood with risk for psychopathology across development (Nigg, 2006; Zelazo, 2020). Findings indicate possible value of researchers conducting future assessment of changes in maternal depressive symptoms over early developmental periods.

Identifying trajectories of maternal depressive symptoms elucidates how exposure to maternal depressive symptoms over time influences child development. The current findings suggest that changes in maternal depressive symptoms from preconception through three months postpartum influence child self-regulation assessed by both maternal report of effortful control and child performance on an inhibitory control task measured one year apart. Effortful control and inhibitory control are overlapping domains that reflect self-regulation processes and support goal-directed behavior, including response inhibition, attentional control, and regulation of emotional and behavioral reactivity (Beck et al., 2011; Rothbart et al., 2007; Zhou et al., 2012). Low effortful control/poor executive function in early childhood can portend risk for psychopathology across the lifespan but may also be modified through early intervention (Zelazo, 2020). The present results indicated that assessment of maternal depressive symptoms even before pregnancy though postpartum may be important for identification of at-risk offspring for early intervention. Trajectories of maternal depressive symptoms were not associated with negative affect and cognitive flexibility; it may be that other unmeasured factors influence these domains, such as maternal physical health and anxiety in pregnancy or caregiving in early childhood (Mahrer et al., 2020a, 2020b; Morgan et al., 2020). Previous research has also found that changes in maternal depressive symptoms from pregnancy through the early postnatal period are associated with more social, emotional, and behavioral problems across development (Bailey et al., 2021; Betts et al., 2014; Fransson et al., 2020; Guyon-Harris et al., 2016; Van Der Waerden et al., 2015). The current results replicate and extend these findings by including assessments of depressive symptoms prior to conception.

Preconception maternal mental health sets the stage for the prenatal environment. For example, maternal depressive symptoms prior to and during pregnancy affect the in-utero environment through alterations to zygote implantation, uterine priming, placental development, and maternal immune and neuroendocrine function (Conradt et al., 2018; Keenan et al., 2018). Fetal adaptation to the in-utero environment is an evolutionarily

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adaptive trait that allows species to adjust growth and development to promote survival in the postnatal environment (Sandman et al., 2012). According to the *match-mismatch model*, adaptive development occurs when fetal predictions of the postnatal environment are congruent with postnatal environmental demands (Gluckman et al., 2005). Thus, stability in maternal depressive symptoms across preconception, pregnancy, and postpartum may confer an advantage because early developmental adjustments to the in-utero environment will match the demands of the postnatal environment.

Offspring development in an adverse in-utero environment may confer an adaptive advantage in a stressful postnatal environment through accelerated development of the fetal stress responses systems and enhanced ability to respond to stress (del Giudice et al., 2011; Nederhof and Schmidt, 2012). Conversely, developmental adjustments to conditions in-utero may be less adaptive postnatally if symptoms change. Previous studies have found that congruence in maternal depressive symptoms in pregnancy and postpartum confer more favorable motor and mental development during infancy, even if symptoms are high, compared to symptoms that change from pregnancy to postpartum (Sandman et al., 2012). Future research could employ within-family study designs over two pregnancies to ensure it is changes in symptoms that are influencing offspring developmental outcomes rather than other, unmeasured factors as well as examine how changes in symptoms may relate to offspring neurodevelopment, particularly the development of corticolimbic circuitry implicated in self-regulation (Gee and Cohodes, 2021).

Levels of preconception depressive symptoms were associated with lower maternal ratings of effortful control at age 4 and levels of depressive symptoms postpartum were associated with poorer performance on an inhibitory control task at age 5. Therefore, changes in symptoms may be associated with self-regulatory domains in part through high symptoms prior to conception or postpartum. These findings should be interpreted with caution as these associations were not statistically significant when adjusting for multiple tests. To our knowledge, only one other study has tested prospective associations between maternal mental health prior to conception and offspring developmental outcomes (Spry et al., 2020). More prospective investigations in this area are necessary as are tests of plausible mechanisms. In the first months of life, caregivers serve an external regulatory function as offspring self-regulation skills develop (Gee and Cohodes, 2021). Postpartum depressive symptoms may alter the availability of caregivers to serve this role and the development of offspring self-regulatory skills (Field, 2010; Kingston and Tough, 2014). Based on these results, assessment of maternal depressive symptoms prior to conception through postpartum as well as measurement of changes across this period may be warranted.

This longitudinal study identified individual patterns of maternal depressive symptoms and elucidated how symptoms change from preconception through postpartum, thereby extending previous research on maternal perinatal depressive symptom trajectories to include measures of symptoms prior to conception (Baron et al., 2017; Santos et al., 2017). Consistent with a life course perspective, most women reported stable symptoms across these periods, providing further evidence that maternal well-being in the perinatal period does not occur de novo of preconception health (Bernstein and Merkatz, 2010; Kee et al., 2021). Intervention before a first pregnancy or between pregnancies may be more

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effective in preventing adverse maternal and infant outcomes than intervention beginning in pregnancy (Atrash et al., 2006; Lu and Halfon, 2003). Therefore, the preconception period may be an important window for detection and intervention. Future research should examine the factors that distinguish women with low stable symptoms across this period from other symptom patterns to inform the content of screening (Baron et al., 2017).

4.1. Strengths and limitations

The current study has notable strengths. A strength is the inclusion of longitudinal assessments over a relatively long period of a woman's reproductive years, beginning prior to conception through postpartum in Study 1 and continuing through early childhood in Study 2. There are few prospective studies that include assessments prior to conception and continue assessments through postpartum and into early childhood due to logistical challenges. In addition, the women are racially, and ethnically diverse and were recruited from rural and urban sites across the United States. All assessments took place in home interviews in a more naturalistic setting and using well-validated measures and standardized procedures. Offspring developmental outcomes were assessed based on maternal report and established assessments at two timepoints approximately a year apart which reduces shared method bias. Finally, the current analytic method expands from traditional growth curve modeling to capture individual heterogeneity in symptoms from preconception to postpartum.

Several limitations warrant mention. There were small sizes of some groups in the trajectories results because most women reported low-stable symptoms; however, trajectories that include more than 5% of the sample are viable (Jung and Wickrama, 2008) and the size of trajectories were consistent with prior research. Although the full CCHN cohort and follow-up study cohort were both racially and ethnically, diverse and mostly low income, there may have been bias in the women who opted to participate in the longitudinal follow up study. In addition, there was variability in the between the most proximal preconception visit and pregnancy and it may be that depressive symptoms more proximal to pregnancy may have stronger effects on maternal perinatal mental health and fetal development. Nonetheless, weeks between the preconception visit and the subsequent pregnancy were not significantly associated with maternal depressive symptoms in pregnancy and postpartum or child developmental outcomes. Each measure of depressive symptoms was selected to be most appropriate for the timing of the study visit when administered but different ratings scales necessitated standardization to compare across measures. Finally, due to study design, maternal depressive symptoms in the first trimester of pregnancy as well as between postpartum and early childhood were not assessed. These may be other important periods for offspring development to consider in future studies.

4.2. Conclusions

Results of this prospective longitudinal study indicate that a woman's mental health prior to conception influences her mental health in pregnancy and postpartum, supporting the value of a life course approach to maternal mental health. To our knowledge, the current study is the first test of the *cumulative stress* and *match-mismatch models* of early development from preconception to postpartum. Significant changes in maternal depressive symptoms

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across this period are associated with less favorable developmental outcomes which provides evidence for the *match-mismatch model*. Assessment of maternal depressive symptoms prior to conception and changes in symptoms over time may allow for early identification and intervention for women and children.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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References

Andruff H, Carraro N, Thompson A, Gaudreau P, Louvet B, 2009. Latent class growth modelling: a tutorial. Tutor.Quant.MethodsPsychol 5 (1), 11–24.

Atrash HK, Johnson K, Adams M, Cordero JF, Howse J, 2006. Preconception care for improving perinatal outcomes: the time to act. Matern. Child Health J 10 (1), 3–11. 10.1007/s10995-006-0100-4.

Bailey NA, Irwin JL, Davis EP, Sandman CA, Glynn LM, 2021. Patterns of maternal distress from pregnancy through childhood predict psychopathology during early adolescence. Child Psychiatry Hum. Dev 1–11 10.1007/s10578-021-01259-7.

Baron E, Bass J, Murray SM, Schneider M, Lund C, 2017. A systematic review of growth curve mixture modelling literature investigating trajectories of perinatal depressive symptoms and associated risk factors. J. Affect. Disord 223, 194–208. 10.1016/j.jad.2017.07.046. [PubMed: 28763638]

Beck DM, Schaefer C, Pang K, Carlson SM, 2011. Executive function in preschool children: test-retest reliability. J. Cogn. Dev 12 (2), 169–193. 10.1080/15248372.2011.563485. [PubMed: 21643523]

Bernstein PS, Merkatz IR, 2010. A life course perspective on women's reproductive health and the outcome of pregnancy. J. Women's Health 19 (3), 555–560. 10.1089/jwh.2009.1397.

Betts KS, Williams GM, Najman JM, Alati R, 2014. Maternal depressive, anxious, and stress symptoms during pregnancy predict internalizing problems in adolescence. DepressionAnxiety 31 (1), 9–18. 10.1002/da.22210.

Bridgett DJ, Oddi KB, Laake LM, Murdock KW, Bachmann MN, 2013. Integrating and differentiating aspects of self-regulation: effortful control, executive functioning, and links to negative affectivity. Emotion 13 (1), 47. 10.1037/a0029536. [PubMed: 22906086]

Chan JC, Nugent BM, Bale TL, 2018. Parental advisory: maternal and paternal stress can impact offspring neurodevelopment. Biol. Psychiatry 83 (10), 886–894. 10.1016/j.biopsych.2017.10.005. [PubMed: 29198470]

Class QA, Abel KM, Khashan AS, Rickert ME, Dalman C, Larsson H, Hultman CM, Långström N, Lichtenstein P, D'Onofrio BM, 2014. Offspring psychopathology following preconception, prenatal and postnatal maternal bereavement stress. Psychol. Med 44 (1), 71–84. 10.1017/S0033291713000780. [PubMed: 23591021]

Conradt E, Adkins DE, Crowell SE, Raby KL, Diamond LM, Ellis B, 2018. Incorporating epigenetic mechanisms to advance fetal programming theories. Dev. Psychopathol 30 (3), 807–824. 10.1017/S0954579418000469. [PubMed: 30068415]

Cox JL, Holden JM, Sagovsky R, 1987. Edinburgh postnatal depression scale (EPDS). Br. J. Psychiatry 150 (6), 782–786. [PubMed: 3651732]

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Davis EP, Sandman CA, 2012. Prenatal psychobiological predictors of anxiety risk in preadolescent children. Psychoneuroendocrinology 37 (8), 1224–1233. 10.1016/J.PSYNEUEN.2011.12.016. [PubMed: 22265195]

Davis EP, Korja R, Karlsson L, Glynn LM, Sandman CA, Vegetabile B, Baram TZ, 2019. Across continents and demographics, unpredictable maternal signals are associated with children's cognitive function. EBioMedicine 46, 256–263. [PubMed: 31362905]

del Giudice M, Ellis BJ, Shirtcliff EA, 2011. The adaptive calibration model of stress responsivity. Neurosci. Biobehav. Rev 35 (7), 1562–1592. 10.1016/j.neubiorev.2010.11.007. [PubMed: 21145350]

Enders CK, 2010. Applied Missing Data Analysis. Guilford Press.

Erdfelder E, Faul F, Buchner A, Lang AG, 2009. Statistical power analyses using G*Power 3.1: tests for correlation and regression analyses. Behav. Res. Methods 41 (4), 1149–1160. 10.3758/BRM.41.4.1149. [PubMed: 19897823]

Ferguson CJ, 2009. An effect size primer: a guide for clinicians and researchers. Prof. Psychol. Res. Pract 40 (5), 532–538. 10.1037/a0015808.

Field T, 2010. Postpartum depression effects on early interactions, parenting, and safety practices: a review. Infant Behav. Dev 33 (1), 1–6. 10.1016/j.infbeh.2009.10.005. [PubMed: 19962196]

Fransson E, Sörensen F, Kunovac Kallak T, Ramklint M, Eckerdal P, Heimgärtner M, Krägeloh Mann I, Skalkidou A, 2020. Maternal perinatal depressive symptoms trajectories and impact on toddler behavior – the importance of symptom duration and maternal bonding. J. Affect. Disord 273, 542–551. 10.1016/j.jad.2020.04.003. [PubMed: 32560952]

Garcia-Esteve L, Ascaso C, Ojuel J, Navarro P, 2003. Validation of the Edinburgh Postnatal Depression Scale (EPDS) in Spanish mothers. J. Affect. Disord 75 (1), 71–76. 10.1016/S0165-0327(02)00020-4. [PubMed: 12781353]

Gavin NI, Gaynes BN, Lohr KN, Meltzer-Brody S, Gartlehner G, Swinson T, 2005. Perinatal depression: a systematic review of prevalence and incidence. Obstet. Gynecol 106 (5), 1071–1083. 10.1097/01.AOG.0000183597.31630.db. [PubMed: 16260528]

Gee DG, Cohodes EM, 2021. Influences of caregiving on development: a sensitive period for biological embedding of predictability and safety cues. Curr. Dir. Psychol. Sci 1–8 10.1177/09637214211015673.

Gluckman PD, Hanson MA, Spencer HG, 2005. Predictive adaptive responses and human evolution. TrendsEcol.Evol 20 (10), 527–533. 10.1016/j.tree.2005.08.001.

Goodman SH, Gotlib IH, 1999. Risk for psychopathology in the children of depressed mothers: a developmental model for understanding mechanisms of transmission. Psychol. Rev 106 (3), 458–490. 10.1037/0033-295x.106.3.458. [PubMed: 10467895]

Guardino CM, Schetter CD, Saxbe DE, Adam EK, Ramey SL, Shalowitz MU, 2016. Diurnal salivary cortisol patterns prior to pregnancy predict infant birth weight. Health Psychol. 35 (6), 625–633. 10.1037/hea0000313. [PubMed: 26844584]

Guyon-Harris K, Huth-Bocks A, Lauterbach D, Janisse H, 2016. Trajectories of maternal depressive symptoms across the birth of a child: associations with toddler emotional development. Arch.Women's Ment.Health 19 (1), 153–165. 10.1007/s00737-015-0546-8. [PubMed: 26184834]

Jung T, Wickrama KAS, 2008. An introduction to latent class growth analysis and growth mixture modeling. Soc. Personal. Psychol. Compass 2 (1), 302–317. 10.1111/j.1751-9004.2007.00054.x.

Kee MZL, Ponmudi S, Phua DY, Rifkin-Graboi A, Chong YS, Tan KH, Chan JKY, Broekman BFP, Chen H, Meaney MJ, 2021. Preconception origins of perinatal maternal mental health. Arch.Women's Ment.Health 10.1007/s00737-020-01096-y.

Keenan K, Hipwell AE, Class QA, Mbayiwa K, 2018. Extending the developmental origins of disease model: impact of preconception stress exposure on offspring neurodevelopment. Dev. Psychobiol 60 (7), 753–764. 10.1002/dev.21773. [PubMed: 30144041]

Kingston D, Tough S, 2014. Prenatal and postnatal maternal mental health and school-age child development: a systematic review. Matern. Child Health J 18 (7), 1728–1741. 10.1007/s10995-013-1418-3. [PubMed: 24352625]

Kingston D, Kehler H, Austin MP, Mughal MK, Wajid A, Vermeyden L, Benzies K, Brown S, Stuart S, Giallo R, 2018. Trajectories of maternal depressive symptoms during pregnancy and the first 12

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months postpartum and child externalizing and internalizing behavior at three years. PLoS ONE 13 (4). 10.1371/journal.pone.0195365.

Lu MC, Halfon N, 2003. Racial and ethnic disparities in birth outcomes: a life-course perspective. Matern. Child Health J 7 (1), 13–30. 10.1023/A:1022537516969. [PubMed: 12710797]

Mahrer NE, Guardino CM, Hobel C, Dunkel Schetter C, 2020. Maternal stress before conception is associated with shorter gestation. Ann. Behav. Med 1–11. 10.1093/abm/kaaa047. [PubMed: 31185074]

Mahrer NE, Ramos IF, Guardino C, Davis EP, Ramey SL, Shalowitz M, Dunkel Schetter C, 2020. Pregnancy anxiety in expectant mothers predicts offspring negative affect: the moderating role of acculturation. Early Hum. Dev 141, 104932 10.1016/j.earlhumdev.2019.104932. [PubMed: 31775096]

Marcus SM, Flynn HA, Blow FC, Barry KL, 2003. Depressive symptoms among pregnant women screened in obstetrics settings. J. Women's Health 12 (4), 373–380. 10.1089/154099903765448880.

Meaney MJ, 2018. Perinatal maternal depressive symptoms as an issue for population health. Am. J. Psychiatr 175(11), 1084–1093. 10.1176/appi.ajp.2018.17091031. [PubMed: 30068258]

Meltzer-Brody S, 2011. New insights into perinatal depression: pathogenesis and treatment during pregnancy and postpartum. Dialogues Clin. Neurosci 13 (1), 89–100. 10.31887/dcns.2011.13.1/smbrody. [PubMed: 21485749]

Morgan JE, Lee SS, Mahrer NE, Guardino CM, Davis EP, Shalowitz MU, Ramey SL, Dunkel Schetter C, 2020. Prenatal maternal C-reactive protein prospectively predicts child executive functioning at ages 4–6 years. Dev. Psychobiol 00, 1–13. 10.1002/dev.21982.

Muthén LK, Muthén BO, 1998-2017. Mplus User's Guide, Eighth Edition. Muthén & Muthén, Los Angeles, CA.

Nederhof E, Schmidt MV, 2012. Mismatch or cumulative stress: toward an integrated hypothesis of programming effects. Physiol. Behav 106 (5), 691–700. 10.1016/j.physbeh.2011.12.008. [PubMed: 22210393]

Nigg JT, 2006. Temperament and developmental psychopathology. J.Child Psychol. PsychiatryAllied Discip 47 (3–4), 395–422. 10.1111/j.1469-7610.2006.01612.x.

Park M, Brain U, Grunau RE, Diamond A, Oberlander TF, 2018. Maternal depression trajectories from pregnancy to 3 years postpartum are associated with children's behavior and executive functions at 3 and 6 years. Arch.Women's Ment. Health 21 (3), 353–363. 10.1007/s00737-017-0803-0. [PubMed: 29340801]

Putnam SP, Rothbart MK, 2006. Development of short and very short forms of the children's behavior questionnaire. J. Pers. Assess 87 (1), 103–113.

R Core Team, 2021. R: A language and environment for statistical computing. In: R Foundation for Statistical Computing, Vienna, Austria. URL. https://www.R-project.org/.

Ramey SL, Schafer P, DeClerque JL, Lanzi RG, Hobel C, Shalowitz M, Chinchilli V, Raju TNK, 2015. The preconception stress and resiliency pathways model: a multi-level framework on maternal, paternal, and child health disparities derived by community-based participatory research. Matern. Child Health J 19 (4), 707–719. 10.1007/s10995-014-1581-1. [PubMed: 25070734]

Rogers A, Obst S, Teague SJ, Rossen L, Spry EA, MacDonald JA, Sunderland M, Olsson CA, Youssef G, Hutchinson D, 2020. Association between maternal perinatal depression and anxiety and child and adolescent development: a meta-analysis. JAMA Pediatr. 10.1001/jamapediatrics.2020.2910.

Rothbart MK, Ahadi SA, Evans DE, 2000. Temperament and personality: origins and outcomes. J. Pers. Soc. Psychol 78 (1), 122–135. 10.1037//0022-3514.78.1.122. [PubMed: 10653510]

Rothbart MK, Sheese BE, Posner MI, 2007. Executive attention and effortful control: linking temperament, brain networks, and genes. Child Dev. Perspect 1 (1), 2–7. 10.1111/j.1750-8606.2007.00002.x.

Sandman CA, Davis EP, Buss C, Glynn LM, 2011. Prenatal programming of human neurological function. Int. J. Pept 10.1155/2011/837596.

Sandman CA, Davis EP, Glynn LM, 2012. Prescient human fetuses thrive. Psychol. Sci 23 (1), 93–100. 10.1177/0956797611422073. [PubMed: 22173740]

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Santor DA, Coyne JC, 1997. Shortening the CES-D to improve its ability to detect cases of depression. Psychol. Assess 9 (3), 233–243. 10.1037/1040-3590.9.3.233.

Santos H, Tan X, Salomon R, 2017. Heterogeneity in perinatal depression: how far have we come? A systematic review. Arch.Women's Ment.Health 20 (1), 11–23. 10.1007/s00737-016-0691-8. [PubMed: 27796597]

Shonkoff JP, Garner AS, Siegel BS, Dobbins MI, Earls MF, McGuinn L, Committee on Early Childhood, Adoption, and Dependent Care, 2012. The lifelong effects of early childhood adversity and toxic stress. Pediatrics 129 (1), e232–e246. [PubMed: 22201156]

Spry E, Moreno-Betancur M, Becker D, Romaniuk H, Carlin JB, Molyneaux E, Howard LM, Ryan J, Letcher P, McIntosh J, Macdonald JA, Greenwood CJ, Thomson KC, McAnally H, Hancox R, Hutchinson DM, Youssef GJ, Olsson CA, Patton GC, 2020. Maternal mental health and infant emotional reactivity: a 20-year two-cohort study of preconception and perinatal exposures. Psychol. Med 50 (5), 827–837. 10.1017/S0033291719000709. [PubMed: 30968786]

Tandon SD, Cluxton-Keller F, Leis J, Le HN, Perry DF, 2012. A comparison of three screening tools to identify perinatal depression among low-income African American women. J. Affect. Disord 136 (1–2), 155–162. [PubMed: 21864914]

Tirumalaraju V, Suchting R, Evans J, Goetzl L, Refuerzo J, Neumann A, Anand D, Ravikumar R, Green CE, Cowen PJ, Selvaraj S, 2020. Risk of depression in the adolescent and adult offspring of mothers with perinatal depression: a systematic review and meta-analysis. JAMA Netw. Open 3 (6), e208783. 10.1001/jamanetworkopen.2020.8783. [PubMed: 32602910]

Van Der Waerden J, Galéra C, Larroque B, Saurel-Cubizolles MJ, Sutter-Dallay AL, Melchior M, 2015. Maternal depression trajectories and children's behavior at age 5 years. J. Pediatr 166 (6), 1440–1448.e1. 10.1016/j.jpeds.2015.03.002. [PubMed: 25866387]

Weintraub S, Dikmen SS, Heaton RK, Tulsky DS, Zelazo PD, Bauer PJ, Gershon RC, 2013. Cognition assessment using the NIH Toolbox. Neurology 80 (11 Supplement 3), S54–S64. [PubMed: 23479546]

Zelazo PD, 2020. Executive function and psychopathology: a neurodevelopmental perspective. Annu. Rev. Clin. Psychol 16, 431–454. 10.1146/annurev-clinpsy-072319-024242. [PubMed: 32075434]

Zhou Q, Chen SH, Main A, 2012. Commonalities and differences in the research on children's effortful control and executive function: a call for an integrated model of self-regulation. Child Dev. Perspect 6 (2), 112–121. 10.1111/j.1750-8606.2011.00176.x.

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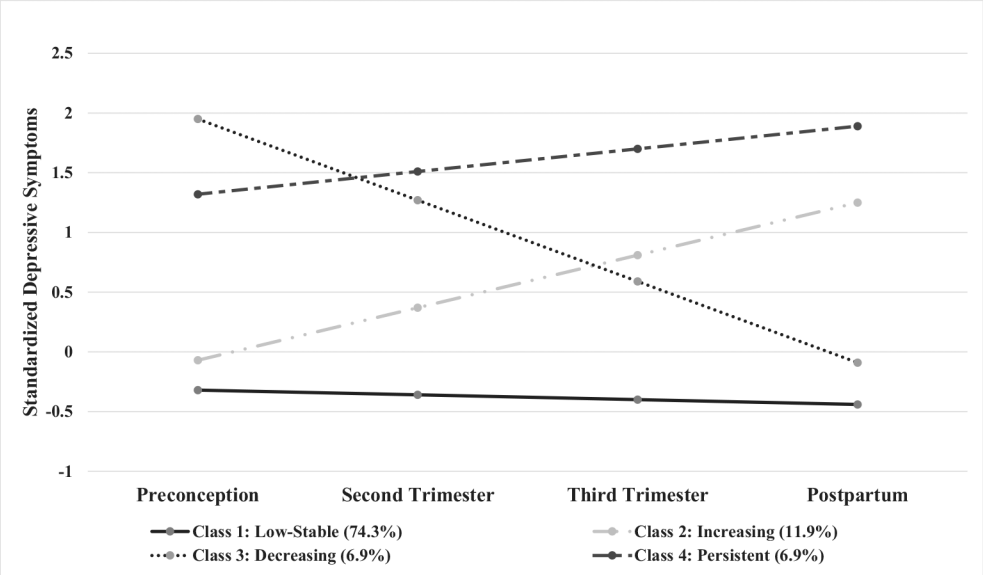

Fig. 1.

Estimated mean latent class trajectories for depressive symptoms (n = 362).

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Table 1

Sample characteristics and differences by trajectory group.

	CCHN cohort (*n* = 362)	Child follow-up cohort (*n* = 125)	Low-stable (*n* = 269)	Increasing (*n* = 43)	Decreasing (*n* = 25)	Persistent (*n* = 25)	*p*-Value or F statistic[a]
Age at enrollment	25.2 (5.7)	26.8 (6.1)	25.4 (5.1)	25.4 (5.4)	24.1 (4.7)	24.2 (4.1)	0.86
Relationship status							
Single	30.0 (108)	24.4 (30)	28.5 (76)	32.6 (14)	48.0 (12)	24.0 (6)	.21
Cohabiting, not married	31.7 (114)	29.3 (36)	29.6 (79)	27.9 (12)	32.0 (8)	60.0 (15)	.02*
Married	38.3 (138)	46.3 (57)	41.9 (112)	39.5 (17)	20.0 (5)	16.0 (4)	.01*
Race/ethnicity							
African American/Black	44.8 (162)	21.1 (26)	43.1 (116)	37.2 (16)	56.0 (14)	64.0 (16)	.10
Hispanic/Latina	29.3 (106)	48.8 (60)	21.2 (57)	32.6 (14)	16.0 (4)	0.04 (1)	.98
Non-Hispanic White	26.0 (94)	30.1 (37)	29.0 (78)	30.2 (13)	28.8 (7)	32.0 (8)	.02*
Income	11,619 (13,951)	15,327 (18,601)	12,394 (19,949)	11,867 (13,685)	7370 (11,285)	7121 (10,470)	1.11
Education (years)	12.6 (2.9)	12.7 (3.6)	12.8 (2.8)	12.4 (2.9)	11.5 (2.2)	11.9 (2.0)	2.62^
Depressive symptoms							
Preconception	4.6 (4.6)	4.7 (4.4)	3.2 (3.1)	4.8 (2.1)	14.3 (2.8)	10.6 (2.8)	70.37***
2nd trimester	18.1 (6.4)	17.0 (5.4)	16.2 (4.0)	18.7 (5.4)	27.0 (6.4)	28.2 (4.0)	60.95***
3rd trimester	17.8 (5.6)	16.8 (5.3)	16.1 (3.6)	21.4 (4.5)	20.5 (5.0)	27.0 (4.2)	63.50***
Postpartum	4.0 (4.5)	4.1 (4.5)	2.0 (2.1)	10.0 (2.9)	3.6 (3.0)	11.9 (3.2)	198.20***

Note. One-way ANOVAs and Fisher's Exact Test used to examine differences by trajectory for continuous and categorical variables, respectively. EPDS assessed depressive symptoms preconception and postpartum. CES-D assessed depressive symptoms in the second and third trimester.

^ $p < .10$.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

[a] *p*-Value or F statistic compares differences in sociodemographic characteristics and depressive symptoms in the CCHN cohort by trajectory group.

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Table 2

Model fit information for latent class growth curve trajectory analysis in CCHN cohort (n = 362).

Number of classes	BIC	BPLRT χ^2	BPLRT *p*-Value	Entropy	Predicted proportion of sample in each class, *n* (%)				
					Class 1	Class 2	Class 3	Class 4	Class 5
1	2785.25	–	–	–	362 (100%)				
2	2632.32	170.61	<.001	0.82	298 (82.3%)	64 (17.7%)			
3	2610.14	39.85	<.001	0.81	287 (79.3%)	55 (15.2%)	20 (5.5%)		
4[a]	2610.04	17.78	<.001	0.77	269 (74.3%)	43 (11.9%)	25 (6.9%)	25 (6.9%)	
5	2609.39	8.05	.13	0.80	264 (72.9%)	43 (11.9%)	28 (7.7%)	26 (7.2%)	1 (0.3%)

Note. BIC = Bayesian Information Criteria. BPLRT = Bootstrapped parametric likelihood ratio test.

[a]Four class model was selected as best fitting model. BIC was lower than the three class model. The four class model was a significant improvement from the three class model and the five class model did not significantly improve in model fit from the four class model. Entropy for the four class model was acceptable.

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Table 3

Means, standard deviations, and bivariate associations between primary study variables in child follow-up cohort.

Variable	*M*	*SD*	1	2	3	4	5	6	7	8	9
1. Preconception depressive symptoms[a]	4.67	4.13									
2. 2nd trimester depressive symptoms[b]	16.95	5.09	.50**								
3. 3rd trimester depressive symptoms[b]	16.77	5.00	.37**	.34*							
4. Postpartum depressive symptoms[a]	4.05	4.16	.50**	.31*	.47**						
5. Maternal depressive symptoms at age 4[b]	14.01	5.10	.43***	.41***	.30**	.32***					
6. Maternal depressive symptoms at age 5[b]	14.82	5.48	.50***	.49***	.43***	.41***	.66***				
7. Effortful control at age 4	5.20	0.74	−.14	−.13	.13	−.08	−.04	−.13^			
8. Negative affect at age 4	4.16	0.80	.05	.21^	.17	.17^	.15^	.21*	.04		
9. Inhibitory control at age 5	51.49	10.50	−.06	.03	−.09	−.22*	.03	.06	.21*	−.03	
10. Cognitive flexibility at age 5	50.28	11.28	−.04	.15	.12	−.07	.09	.06	.19^	−.03	.36***

Note. *M* and *SD* are used to represent mean and standard deviation, respectively.

^ $p < .10$.

* $p < .05$.

** $p < .01$.

*** $p < .001$. Preliminary correlations were not conducted using full information maximum likelihood to handle missing data.

[a] Measured with EPDS

[b] Measured with CES-D 9-item version.

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Table 4

Associations between potential covariates and offspring developmental outcomes.

	Temperament at 4 years		Executive function at 5 years[a]	
	Negative affect	**Effortful control**	**Inhibitory control**	**Cognitive flexibility**
	Test statistic or M (SD)			
Child sex	0.55	5.49***	–	–
Child age	0.19	−0.14	–	–
Birth order	−0.04	−0.17	−0.12	−0.06
Maternal education (years)	−0.10	0.12	–	–
Maternal relationship status	4.61*	0.28	0.26	0.22
Single	4.36 (0.94)	5.20 (0.87)	51.44 (9.51)	48.84 (9.29)
In a relationship	4.29 (0.69)	5.29 (0.81)	50.14 (8.57)	51.00 (9.41)
Married	4.01 (0.76)[b]	5.16 (0.67)	51.91 (10.18)	50.23 (11.68)
Maternal race/ethnicity	1.63	6.07*	–	–
African American/Black	4.41 (0.86)	5.42 (0.82)		
Non-Hispanic White	4.06 (0.92)	5.31 (0.73)		
Hispanic/Latina	4.12 (0.67)	5.03 (0.70)[c]		
Income	−0.23*	0.10	−0.02	0.14
Birth weight	0.10	−0.13	0.08	0.08
Gestational age	0.18^	−0.07	0.19^	0.16
Weeks between preconception visit and pregnancy	−0.04	−0.06	−0.07	−0.07

Note. Pearson's correlation coefficient, T-statistic, or F-statistic presented for continuous, dichotomous, or categorical variables, respectively. Maternal relationship status and income based on maternal report at early childhood visit.

^ $p < .10$.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

[a] Executive function domain scores fully adjusted for child sex, child age, child race/ethnicity, and maternal education.

[b] Married women rated children significantly lower in negative affect compared to single women.

[c] Hispanic/Latina women rated children significantly lower in effortful control compared to African American/Black women.

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Table 5

Multivariate linear regression results of depressive symptom trajectories predicting offspring developmental outcomes.

	Child effortful control at age 4			Child negative affect at age 4			Child inhibitory control at age 5			Child cognitive flexibility at age 5		
	Model 1			Model 2			Model 3			Model 4		
	β	B (95% CI)	*p*	β	B (95% CI)	*p*	β	B (95% CI)	*p*	β	B (95% CI)	*p*
Unadjusted												
Low-Stable	Reference			Reference			Reference			Reference		
Increasing	−0.12	−0.29 (—0.70, 0.13)	0.17	0.08	0.19 (−0.25, 0.64)	0.40	−0.33	−10.02 (−16.34, −3.70)	0.002**	−0.15	−5.01 (−11.86, 4.90)	0.15
Decreasing	−0.17	−0.53 (−1.06, 0.01)	0.05^	0.02	0.06 (−0.52, 0.63)	0.85	−0.15	−5.75 (−12.85, 1.36)	0.11	−0.07	−3.13 (−11.17, 4.90)	0.44
Persistent	−0.00	−0.01 (−0.57, 0.56)	0.98	0.08	0.27 (−0.34, 0.88)	0.38	−0.05	−2.03 (−9.14, 5.07)	0.58	0.09	3.87 (−4.17, 11.90)	0.35
		R^2	0.04		R^2	0.01		R^2	0.12		R^2	0.04
Adjusted												
Low-Stable	Reference			Reference			Reference			Reference		
Increasing	−0.10	−0.25 (−0.61, 0.11)	0.18	0.09	0.23 (−0.21, 0.67)	0.30	−0.35	−10.74 (−17.04, −4.43)	0.001**	−0.17	−5.59 (−12.54, 1.36)	0.12
Decreasing	−0.24	−0.72 (−1.20, −0.24)	0.003**	−0.03	−0.11 (−0.69, 0.47)	0.71	−0.21	−8.10 (−15.70, −0.50)	0.04*	−0.11	−4.62 (−13.29, 4.05)	0.30
Persistent	−0.03	−0.09 (−0.59, 0.41)	0.72	−0.00	−0.02 (−0.62, 0.59)	0.96	−0.12	−4.99 (−12.94, 2.95)	0.22	0.04	1.99 (−7.06, 11.04)	0.67
		R^2	0.33		R^2	0.10		R^2	0.14		R^2	0.04

Note.

^ $p < .10$.

* $p < .05$.

** $p < .01$.

*** $p < .001$. β = standardized coefficient. B = unstandardized coefficient. CI = confidence interval. Unadjusted models are presented for comparison to adjusted models. Adjusted models were conducted in four multiple regression models with trajectory membership dummy coded and entered simultaneously into separate models for each developmental outcome. A post-hoc Bonferroni multiple testing correction was applied to primary adjusted models (adjusted *p*-value = .013).

[a] Adjusted for child sex, per capita income, maternal relationship status (dummy coded; reference group = married), maternal race/ethnicity (dummy coded; reference group = Hispanic/Latina), maternal depressive symptoms when the child was age 4.

[b] Adjusted for maternal depressive symptoms in early childhood (standardized composite of depressive symptoms when child was 4 and 5 years old). Inhibitory control and cognitive flexibility scores based on T-scores adjusting for child sex, age, race/ethnicity, and maternal education.