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Childhood family stress modifies the association between perinatal stressful life events and depressive symptoms

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Abstract

Background: The childhood family environment can influence long-term well-being in part by modifying how individuals' respond to and cope with stress across the lifespan. Theoretical models propose that childhood stress will either exacerbate (*stress sensitization*) or attenuate (*steeling effect*) the effects of adult stress on mental health. This study tests whether childhood family stress modifies the association between stressful life events and depressive symptoms in pregnancy and consecutive postpartum periods.

Methods: A sample of 127 women reported on depressive symptoms after one birth, during a subsequent pregnancy, and postpartum following that birth. Childhood family stress was assessed with the Risky Families Questionnaire. Stressful life events were measured at all three timepoints to capture the number of life events during both pregnancies and between pregnancies.

Results: Associations between stressful life events and depressive symptoms varied as a function of childhood family stress. At the between-persons level, more stressful life events were associated with greater depressive symptoms among women who reported infrequent exposure to childhood family stress in this sample, but not among women who reported more frequent exposure to childhood family stress.

Conclusions: Results provides novel evidence that moderate exposure to childhood family stress may attenuate the association between stressful life events and depressive symptoms in the perinatal period, consistent with a steeling effect. That is, some degree of childhood family stress may promote resilience to perinatal stress. Findings underscore the utility of examining the interaction of risk factors across the lifespan in predicting perinatal mental health.

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Keywords

stress; family stress; depressive symptoms; pregnancy; postpartum

Depressive symptoms among women during the perinatal period (pregnancy and postpartum) are prevalent and have adverse implications for long-term maternal health, child development, and family functioning (Gavin et al., 2005; Haight et al., 2019; Meaney, 2018). A life course perspective proposes that maternal mental health during and after pregnancy is influenced by the interaction between risk and protective factors across the lifespan (Bernstein & Merkatz, 2010; Lu & Halfon, 2003), highlighting the importance of childhood experiences in predicting perinatal well-being. Childhood is a developmental period characterized by rapid development of biological stress response systems and heightened sensitivity to the social environment (Gunnar & Hostinar, 2015; Gunnar & Quevedo, 2007) such that experiences in childhood can have enduring effects on responses to stress and risk for mental health problems across the lifespan (Del Giudice et al., 2011; Repetti et al., 2002; Stroud, 2020). For example, the strength of the association between stress and mental health symptoms in adulthood varies as a function of childhood stress (e.g., Seery et al., 2010; Shapero et al., 2015; Stroud et al., 2020). However, the role of childhood stress in modifying the association between stress and mental health symptoms in the perinatal period remains unclear despite strong evidence that childhood stress and stressful life events in the perinatal period increase risk for perinatal depressive symptoms (Ertel et al., 2011; Racine et al., 2021; Yim et al., 2015). The current study utilizes a life course approach to test if childhood family stress modifies the association of stressful life events with depressive symptoms in pregnancy and postpartum.

As one of the primary contexts in which children's development unfolds, the family environment serves a particularly important role in calibrating responses to stress and risk for mental health problems (del Giudice et al., 2012; Ellis et al., 2017; Repetti et al., 2002). Childhood family environments characterized by high aggression, high conflict, low support, or low warmth has been found to disrupt emotion processing, social competence, and the development of biological stress response systems (Repetti et al., 2002). Theory and evidence diverge, however, in whether such experiences in childhood increase vulnerability or resilience to mental health problems after later stress (Ellis & Boyce, 2008; Hostinar & Gunnar, 2013; Shakiba et al., 2017; Stroud, 2020). The *stress sensitization model* posits that childhood experiences of stress will increase sensitivity to later stress and increase susceptibility to mental health problems after later stress (Stroud, 2020). Studies consistent with sensitization effects of childhood stress have found that individuals who were exposed to family dysfunction, parental psychopathology, family violence, or abuse in childhood report greater depressive symptoms following recent stressors when compared to individuals who have not experienced childhood stress (see Stroud, 2020 for a review).

Contrasting theories developed from evolutionary psychology and resilience research propose that some early stress exposures, such as exposure to a family environment that is neither highly threatening nor highly supportive (i.e., moderate childhood stress), may increase resistance to later stressful experiences and decrease vulnerability to mental health

problems after later stress, referred to as a *steeling effect* (Ellis et al., 2020; del Giudice et al., 2011; Rutter, 2012; Seery, 2011). Consistent with steeling effects, studies have found that individuals who experienced some stressful life events in childhood were at lower risk for emotional distress after later stress exposure compared to individuals exposed to no early life stress or extreme early life stress (Seery et al., 2010; Shapero et al., 2015). Such evidence from non-pregnant samples indicates that childhood experiences serve a critical role in modifying the association between stress and mental health symptoms across the lifespan by either sensitizing or steeling individuals to adult stress. To date, the role of childhood family stress in modifying associations of stress with depressive symptoms in the perinatal period is unclear. Further investigations in this area are necessary given the implications of perinatal depressive symptoms for long-term child and family health (Meaney, 2018).

In addition to being critical windows whereby maternal stress exposure and depressive symptoms can influence long-term family health, pregnancy and postpartum also represent periods in which childhood experiences are more salient. For example, individuals often draw on memories of childhood, attachment representations, and experiences with caregivers to prepare for parenthood (Aparicio, 2017; Davis & Narayan, 2020; Narayan et al., 2020, 2021). Therefore, childhood family social environment may play a particularly important role in predicting well-being after stress in the perinatal period. Accumulating evidence in recent years has indicated that childhood adversity, such as family dysfunction, abuse, and neglect, predicts lower well-being in the perinatal period (for a review, see Racine et al., 2021). In contrast, other evidence indicates that the perinatal period may be a time of resilience for individuals with a history of childhood family stress in which individuals draw on stress-adapted coping skills and social support to break the cycle of adversity (Atzl et al., 2019a; Chamberlain et al., 2019; Ellis et al., 2020; Davis & Narayan, 2020). Although such evidence highlights the centrality of childhood experiences to perinatal well-being, it remains unclear the extent to which childhood experiences modify associations between perinatal stress and well-being.

To our knowledge, only one study has evaluated the interactive effect of childhood and perinatal stress on psychological distress in the perinatal period. In a community sample of 200 pregnant women, there was a significant interactive effect of early life stress and prenatal intimate partner violence in predicting emotional distress in pregnancy (Tung et al., 2019). Specifically, there was a U-shaped association between harsh parenting and prenatal distress in the context of prenatal intimate partner violence such that moderate levels of harsh parenting in childhood were associated with lower emotional distress compared to low or high levels of harsh parenting among women who experienced intimate partner violence during pregnancy. Conversely, exposure to traumatic violence in childhood, regardless of the level, was associated with greater emotional distress in the context of intimate partner violence. These results suggest differences in steeling and sensitization effects in pregnancy depending on the type of childhood stress, with steeling effects of moderate harsh parenting but sensitization effects of traumatic violence. Additional work is necessary to elucidate whether childhood experiences confer susceptibility or resilience to depressive symptoms in pregnancy and extend examination to the postpartum period.

The Current Study

In a prospective cohort study conducted in a racially and ethnically diverse and mostly low-income sample, we evaluate the interactive effects of childhood family environment and stressful life events in predicting perinatal depressive symptoms across consecutive births. This study focuses centrally on the perinatal period as a key window for long-term family health, as well as a time in which childhood experiences may be especially salient to stress responses and well-being (Davis & Narayan, 2020; Meaney, 2018). Using multilevel modeling, we test whether childhood family environment modifies the association between stressful life events and depressive symptoms in mothers postpartum of one birth, during a subsequent pregnancy, and postpartum of that subsequent birth. Stress is conceptualized as stressors and stress responses, including acute events and chronic conditions that may be interrelated (Epel et al., 2018). Stressful life events encompass one possible form of stressor that can occur in the perinatal period that include events such as death of someone close, loss of a job, and relationship problems. Of note, stressful life events are common in pregnancy and postpartum, with up to 70% of pregnant individuals reporting experiencing at least one stressful life event, and are associated with perinatal depressive symptoms (e.g., Baron et al., 2017; Salm Ward et al., 2017). Family social environment in childhood was evaluated as the mean frequency of exposure to low warmth, low support, high conflict, and high aggression (hereafter, childhood family stress) (Repetti et al., 2002).

We hypothesized that more frequent exposure to family stress in childhood and more perinatal stressful life events would each be associated with greater perinatal depressive symptoms, consistent with prior research (Baron et al., 2017; Ertel et al., 2011; Racine et al., 2021; Yim et al., 2015). However, we predicted that these main effects would be qualified by an interaction between childhood family stress and stressful life events such that the effect of perinatal stress on depressive symptoms will depend on childhood family stress. Prior theory and evidence diverge as to whether childhood stress confers vulnerability or resilience to depressive symptoms after later stress and only one study to our knowledge has tested this in the perinatal period (Tung et al., 2019). Thus, we had no specific hypotheses regarding the manner in which childhood family stress would modify the association between stressful life events and depressive symptoms.

Methods

Journal Article Reporting Standards

We report how we determined our sample size, all data exclusions (if any), all manipulations, and all measures in the study. Study data, materials, and analysis code is available from the first author upon request. The study was not pre-registered.

Participants and Procedure

Participants in this study were enrolled in the multi-site Community Child Health Network (CCHN; conducted from 2007 to 2012) designed to gain insights into the reasons for disparities in maternal and child health that sampled only Hispanic/Latina, Black/African American and non-Hispanic White women. Participants were recruited following a birth

(index birth) in five study sites and completed visits postpartum of that birth and through a subsequent pregnancy and postpartum period. Participants who were enrolled at three eligible CCHN study sites were invited to enroll in a follow-up study of the subsequent child's development. Participants enrolled in the follow-up study completed additional home visits four and five years after the subsequent birth. Further details on CCHN and the follow up study are reported elsewhere (Mahrer et al., 2020; Morgan et al., 2020). The current sample includes all participants enrolled in the follow-up study ($n = 127$). An overview of study visits is presented in Figure 1. Trained interviewers conducted structured interviews during in-home visits. The study was approved by the Institutional Review Boards at each site. All participants provided written and informed consent and were compensated for study visits.

Measures

Sociodemographic and Medical Variables—Participants reported their age, education, household income, household size, racial/ethnic identity, relationship status, and number of previous live births at the time of enrollment in CCHN. Updates to income and relationship status were collected at each study visit. Data on interpregnancy interval was obtained from medical records. Per capita income was calculated as household income divided by household size, adjusted for cost of living in each study site.

Childhood family stress—Participants reported on childhood family stress with the 10-item Risky Families Questionnaire four years following the subsequent birth. This measure assesses childhood family environment and dysfunction between ages 5 and 15 years (Repetti et al., 2002; Taylor et al., 2004; e.g., “how often did a parent or other adult in the household swear at you, insult you, put you down, or act in a way that made you feel threatened?”). Participants reported the frequency of which each item occurred on a scale of 1 (*not at all*) to 5 (*very often*). An average of the items was calculated with higher scores indicating a greater mean frequency of exposure to a family social environment characterized by low warmth, low support, high conflict, and/or high aggression. Cronbach's alpha indicated good reliability in the current sample ($\alpha = .88$).

Stressful life events—Stressful life events were assessed with the Life Events Inventory at three study visits (Dominguez et al., 2005). Participants retrospectively reported whether or not they had experienced 24 possible life events over a given period at each visit (e.g., “you were threatened with physical harm”, “serious arguments several times with any one person”, “you or someone close had a serious injury, illness, or hospitalization”). These items were used to calculate indices that reflected: (1) the number of life events that occurred in the index pregnancy (reported one month after the index birth); (2) the number of life events that occurred in the interpregnancy interval between the index birth and the subsequent birth (reported 12 months after the index birth); and (3) the number of life events that occurred in the subsequent pregnancy (reported three months after the subsequent birth).

Depressive Symptoms—Two instruments were used to assess maternal depressive symptoms to be appropriate to the time periods administered. The Edinburgh Postpartum Depression Scale (EPDS) was used to assess depressive symptoms during postpartum

periods after the index and subsequent births (Cox et al., 1987). The EPDS consists of 10 items that assess severity of depressive symptoms over the past week on a scale of 0 to 3 and is validated for use in the first year postpartum in Spanish and English (Garcia-Esteve et al., 2003). The Center for Epidemiological Studies Depression Scale 9-item version (CES-D) was administered during the second and third trimesters of pregnancy (Santor & Coyne, 1997). The CES-D assesses frequency of symptoms in the past week on a scale of 1 (*none of the days*) to 4 (*most or all days*) and is validated for use in pregnancy (Marcus et al., 2003). CES-D scores in the second and third trimester of pregnancy were intercorrelated and scored as an average ($r = .53, p < .001$). Mean scores were used in analyses. Cronbach's alpha indicated acceptable reliability for the EPDS ($\alpha = .82-.83$) and CES-D ($\alpha = .75-.86$).

There is substantial overlap between the EPDS and CES-D as demonstrated in prior studies in perinatal samples (Tandon et al., 2012). In the current sample, the measures were also positively correlated (r 's = 0.28–0.50, see Table 2). Depressive symptoms were scaled as the proportion of maximum score (POMS) to account for differences in instruments across study visits. POMS scores represent the proportion of the total possible scale score between 0 and 1, with higher scores indicating greater symptoms. This method maintains the proportion of absolute differences between observed response scores and is the recommended scale transformation for longitudinal data (Moeller, 2015).

Data Analytic Plan

We conducted analyses in R Version 3.6.1. Prior to analysis, we examined the primary study variables for normality (skewness < 3; kurtosis < 7) and outliers (> 3 standard deviations from sample mean). We accounted for non-normality by log-transformation and outliers by winsorizing to three standard deviations from the mean. None of the primary study variables violated assumptions of normality. Data included one outlier on depressive symptoms in pregnancy, one outlier on depressive symptoms postpartum of the subsequent birth, three outliers on stressful life events in the index pregnancy, one outlier on stressful life events between pregnancies, and three outliers on stressful life events in the subsequent pregnancy. Results were robust to outliers and did not change with or without winsorization; we present models with outliers winsorized.

Primary Analysis—We tested study hypotheses using multilevel modeling to account for the nested structure of the longitudinal data and repeated assessment of stressful life events and depressive symptoms. Multilevel modeling accounts for repeated measurements within individuals and allows for the examination of both within- and between-person sources of variability in the outcome (Luke, 2019). Focal predictors and covariates were mean centered prior to analysis to ease interpretation of results (Aiken & West, 1991). Childhood family stress was grand mean centered (Level 2). Stressful life events were grand mean centered (Level 2) and centered within each individual (Level 1) to evaluate within- and between-person effects (Snijders & Bosker, 2011). Between-person effects test whether an individual who reports more stressful life events is predicted to report greater depressive symptoms than an individual who reports fewer stressful life events. Within-person effects test whether an individual is predicted to report greater depressive symptoms at study visits when they report more stressful life events than their average number of stressful life events. Models

testing interactive effects included two interactive terms to test whether (1) childhood family stress modified the between-person association of stressful life events (average level of stressful life events across participants) with depressive symptoms and (2) childhood family stress modified the within-person association of stressful life events (changes in stressful life events at a given time point relate to an individual's average level of stressful life events) with depressive symptoms.

Multilevel modeling was conducted with a series of linear mixed models using the lme4 and lmerTest packages in R (Bates et al., 2007; Kuznetsova et al., 2015). To confirm whether multilevel modeling was the most appropriate analysis for the current data, we first ran an unconditional (empty) model to evaluate systematic variability in depressive symptoms at the within- and between-persons level and evaluated the intraclass coefficient (ICC). ICC values reflect nonindependence of observations and the proportion of variation in the outcome explained by between-person variation (Musca et al., 2011), with higher values indicating greater between-person variability. We also tested an unconditional slope model that evaluated the linear effect of time on depressive symptoms, given that time could be an important contributor to depressive symptoms over the study period (study period length $M = 4.79$ years, $SD = 0.53$ years) (Bolger & Laurenceau, 2013).

To test primary research questions, we added predictors to the models to test the main and interactive effects of childhood family stress and stressful life events on perinatal depressive symptoms. First, we tested a random intercept model that tested the effect of childhood stress and stressful life events on depressive symptoms to evaluate main effects. We then tested a random intercept model with the within-level and cross-level interaction, which allows for the intercept, but not the slope, to vary across individuals. Finally, we tested a random slope model that included a within- and cross-level interaction. The random slope model extends the random intercept and allows for both the intercept and slope to vary across individuals. We tested both a random intercept and random slope model for theoretical and methodological reasons. Theoretically, it is not only plausible that stress could predict average depressive symptoms across this period but also that the effect of stress on depressive symptoms varies across individuals (Snijders & Bosker, 2011). For example, individuals who report more stressful life events may report higher levels of depressive symptoms on average, but there may be some individuals for whom the associations between stressful life events and depressive symptoms are stronger or weaker. The latter can be captured with random slopes. Methodologically, modeling random slopes is recommended in the context of cross-level interactions as failure to do so may disregard cluster-driven heteroskedasticity and autocorrelation, thereby violating model assumptions and introducing systematic bias in model estimates (see Heisig & Schaeffer, 2018). The best fitting model of the random intercept and random slope model was determined with the log-likelihood ratio test (Snijders & Bosker, 2011). We interpret coefficients from the best fitting model.

We conducted simple effects analyses to probe significant interactions at the mean and one standard deviation above and below the mean of the moderator (Aiken & West, 1991). Additionally, we explored regions of significance using Johnson-Neyman analyses to determine the values of the moderator (childhood family stress) where the relation between

stressful life events and depressive symptoms moved from non-significance to significance (Preacher et al., 2006). Significant main effects were interpreted if interactions were not statistically significant ($\alpha = .05$).

Covariates.: Models adjusted for covariates based on potential confounding influences. In primary analysis models, time since start of the study was included as a covariate, as is recommended in multilevel models (Bolger & Laurenceau, 2013). Number of previous live births at time of enrollment (primiparity; no previous live births vs. one or more previous live births) and interpregnancy interval were included as covariates given associations with perinatal depressive symptoms in prior studies. Sociodemographic characteristics of age, education, per capita household income, racial/ethnic identity, and relationship status were evaluated as covariates and included if significantly associated with depressive symptoms at any study visit ($\alpha = .05$). Participants who were single reported significantly higher depressive symptoms than participants who were not single at each study visit. Higher per capita household income was associated with lower depressive symptoms in the subsequent pregnancy ($r = -0.21, p = .04$). Thus, all models adjusted for relationship status (dummy coded; married or not married but cohabiting with a partner [partnered] vs. neither married nor cohabiting with a partner [not partnered]), income (mean centered), primiparity (dummy coded; no previous live births vs. one or more previous live births), and interpregnancy interval (mean centered).

Results

Sample description

A complete sample description is presented in Table 1. Mean participant age at enrollment was 26.6 years ($SD = 6.1$ years). Participants identified as Hispanic/Latina (48.8%), non-Hispanic White (31.5%), and Black/African American (21.3%). The modal level of educational attainment was high school, GED, or technical degree and the mean per capita household income was \$15,307 ($SD = \$18,384$). A majority of participants were either married (46.5%) or not married but cohabiting with their partner (30.7%) one month postpartum of the index birth. The index birth was the first birth for half of the participants (51.8%).

Descriptive statistics

Table 2 presents descriptive statistics and bivariate associations of primary study variables. On average, participants reported mean frequency of exposure (averaged across all items) to family stress in childhood as occurring “rarely” ($M = 1.92, SD = 0.86$). Across all items, most of the sample (84%) reported mean frequency of exposure to childhood family stress as “never” or “rarely” whereas 12% reported mean frequency of exposure to childhood family stress as occurring “sometimes” and 4% reported exposure to childhood family stress as occurring “often” or “very often”. Mean levels of exposure to childhood family stress were mostly low with some variation and comparable to levels in other community samples (Felitti et al., 1998; Lehman et al., 2009). Additional information on the frequency of childhood family stress is presented in the Supplemental Materials. Number of stressful life events were similar during the index pregnancy ($M = 2.95, SD = 2.87, range = 0-15$) and

subsequent pregnancy ($M = 2.54$, $SD = 2.80$, range = 0–12), but slightly higher during the interpregnancy interval ($M = 3.73$, $SD = 3.21$, range = 0–22). Across study visits, between 76.8% to 83.2% of the sample experienced at least one stressful life event, indicating slightly higher rates in the current sample compared to other community samples (e.g., Salm-Ward et al., 2017).

In bivariate associations, more frequent childhood family stress was associated with higher depressive symptoms in the subsequent pregnancy and postpartum of the subsequent birth. More frequent childhood family stress was also positively associated with reporting more stressful life events in pregnancy but was not associated with stressful life events in the index pregnancy or between pregnancies. More stressful life events were associated with greater depressive symptoms at each timepoint (r 's = 0.12–0.38).

Primary analysis

We used multilevel modeling to evaluate the interactive effect of childhood family stress and perinatal stressful life events on depressive symptoms in pregnancy and postpartum. In the unconditional (empty) model, the estimated mean of depressive symptoms across the three timepoints was 0.19 and the ICC indicated that a substantial proportion (19%) of the variation in depressive symptoms was due to variability between-persons. Thus, multilevel modeling is the most appropriate analytic method to account for the nested longitudinal data structure and avoid inflation of the Type I error rate (Musca et al., 2011). In the unconditional slope model evaluating the effect of time on depressive symptoms, we found that time significantly predicted depressive symptoms such that time positively predicted depressive symptoms. Complete coefficients from each model are presented in Table 3.

First, we tested the main effects of childhood family stress and stressful life events on depressive symptoms in the perinatal period. Childhood family stress was not significantly associated with depressive symptoms, but more stressful life events were significantly associated with greater depressive symptoms. When including interactive effects in the model, we found that the random slope model significantly improved model fit from the random intercept model based on the log-likelihood test; therefore, we interpret coefficients from the random slope model. In the random slope model that included main and interactive effects of childhood stress and stressful life events, more frequent exposure to family stress in childhood and more stressful life events were independently associated with greater depressive symptoms in the perinatal period. However, these effects were qualified by a significant between-person interactive effect of childhood family stress and average levels of stressful life events (Level 2) on depressive symptoms such that the association between stressful life events and depressive symptoms varied across individuals depending on childhood family stress at the between-person level.

Simple slopes analyses (Figure 2) revealed that more stressful life events were associated with greater depressive symptoms among individuals who experienced low (Intercept = 0.18, $B = 0.028$, $SE = 0.006$, $p < .001$) or mean (Intercept = 0.20, $B = 0.018$, $SE = 0.005$, $p < .001$) frequency of exposure to childhood family stress in the current sample. Stressful life events were not significantly associated with depressive symptoms among individuals who reported high frequency of exposure to childhood family stress in the current sample (Intercept =

0.22, $B = 0.008$, $SE = 0.006$, $p = .19$). Simple slopes in the current sample corresponded to mean frequency of exposure to childhood stress as never (low), rarely (sample mean), and sometimes (high), respectively. Johnson-Neyman region of significance analyses (Figure 3) indicated that the association between average stressful life events and depressive symptoms was positive and significant for individuals who reported mean frequency of exposure to childhood family stress at a mean centered value less than 0.66 (observed range = -0.93 to 2.67), which corresponded to a mean frequency of less often than “sometimes”.

Within-person changes in stressful life events were not associated with depressive symptoms nor did association between within-person changes in stressful life events and depressive symptoms vary depending on childhood family stress. That is, depressive symptoms did not vary across study visits as a function of whether stressful life events deviated from an individual’s average levels of stressful life events and frequency of childhood family stress did not modify this association.

Discussion

A life course perspective underscores the importance of the cumulative and interactive effects of risk and resilience factors across the lifespan on maternal mental health during and after pregnancy (Bernstein & Merkatz, 2010; Lu & Halfon, 2003), yet the interactive effects of childhood and perinatal stress on perinatal depressive symptoms are understudied. To our knowledge, this is the first study test childhood family environment as a moderator of the association between stressful life events and depressive symptoms in pregnancy and across consecutive postpartum periods. In the current study, mean frequency of exposure to family stress was low on average but there was considerably variability in the sample. Rates in this sample were also similar to rates of childhood family stress in other community samples (e.g., Felitti et al., 1998; Lehman et al., 2009). Within this context, more frequent exposure to a childhood family environment characterized by low warmth and support and high conflict and dysfunction was associated with greater depressive symptoms in pregnancy and postpartum. More stressful life events in the perinatal period were also independently associated with greater depressive symptoms. However, the association between stressful life events and depressive symptoms was conditional on childhood family stress. More stressful life events were associated with greater depressive symptoms among individuals who reported a never or rarely experiencing childhood family stress. However, stressful life events were not associated with depressive symptoms among individuals who reported experiencing childhood family stress sometimes or more often. These results suggest that moderate levels of family stress in childhood may promote resilience to depressive symptoms in the context of perinatal stress.

The present findings add to small but growing evidence that childhood family experiences play an important role in modifying responses to stress and subsequent risk for depressive symptoms in the perinatal period. This pattern of findings may be consistent with a steeling effect of childhood stress (Rutter, 2012), which refers to evidence that early life stress may serve to increase resistance to later stress thereby reducing vulnerability to depressive symptoms (Seery, 2011; Seery et al., 2010; Shapero et al., 2019). Consistent with the present findings, evidence in non-pregnant samples suggests that the childhood family stress

serves an especially important role in calibrating responses to stress across development compared to other forms of stress in childhood (del Giudice et al., 2012; Ellis et al., 2017). In the context of the perinatal period, another recent study reported a steeling effect of moderate levels of harsh parenting in childhood to emotional distress in pregnancy among individuals experiencing prenatal intimate partner violence (Tung et al., 2019). Together with the current results, these findings indicate moderate levels of childhood family stress (e.g., harsh parenting, low parental warmth and support) in particular may contribute to a steeling effect in the context of perinatal stress.

There are several pathways in which moderate childhood family stress could protect against depressive symptoms after stressful life events in the perinatal period. Moderate family stress may promote the development of stress-adapted coping abilities, social skills, and cognitive resources that are advantageous in the face of later stressors (i.e., “hidden talents”; Ellis et al., 2020). The perinatal period has been conceptualized as a potential period of resilience for individuals with a history of early life stress (Davis & Narayan, 2020). Therefore, the perinatal period may be a time in which individuals who experienced childhood stress actively draw on coping skills and social support to break the cycle of adversity (Atzl et al., 2019a; Chamberlain et al., 2019). Moreover, the transition to parenthood may be a time of positive posttraumatic growth and positive social role change for individuals with a history of childhood stress (Aparicio, 2017; Fava et al., 2016). These factors may allow individuals to cope with stressful events in the perinatal period and mitigate risk for depressive symptoms after stressful life events.

This is not to say that more frequent exposure to family stress in childhood is beneficial in all circumstances in pregnancy and postpartum. For example, in the current sample higher family stress in childhood was associated with more stressful life events in pregnancy and postpartum. In this sense, childhood stress may indirectly increase vulnerability to depressive symptoms by contributing to higher levels of stressful life events, potentially through stress generation pathways (Davis & Narayan, 2020; Liu et al., 2013). Furthermore, growing evidence indicates a significant, positive association of childhood family stress with depressive symptoms in pregnancy and postpartum (for a review, see Racine et al., 2021). Consistent with such evidence, childhood stress was positively associated with perinatal depressive symptoms in this sample. Moreover, individuals who reported greater frequency of exposure to childhood family stress had greater depressive symptoms at low levels of perinatal stress as compared to individuals who reported less frequent exposure to childhood family stress. Therefore, this pattern of results suggests that moderate frequency of exposure to family stress may promote resilience to depressive symptoms when faced with later stressful events. Rather than adopting a deficit-focused lens to childhood stress and lifespan mental health, future research should aim to identify the contexts in which early experiences of stress may be resilience-promoting and the pathways through which this might occur.

Resilience is a dynamic concept involving the interaction between multiple systems (Masten, 2018; Masten & Barnes, 2018) and we cannot conclude whether childhood family stress modifies the association between perinatal stress and other health outcomes. While moderate exposure to family stress in childhood may protect against depressive symptoms in the context of perinatal stress, there may be a cost to physiological systems. *Skin deep*

resilience refers to the observation that some individuals who appear to be psychosocially resilient (e.g., more favorable mental health) in the face of stress demonstrate high allostatic load and accelerated epigenetic aging (Brody et al., 2016). For example, childhood stress is associated with dampened cortisol reactivity in pregnancy and postpartum (Epstein et al., 2021). Dampened cortisol reactivity may protect against depressive symptoms in the context of perinatal stress but can hold adverse implications for health behaviors and physical health outcomes (Obradovi , 2012; Saxbe et al., 2012). Future assessment of psychological and physiological functioning in perinatal samples is necessary.

Strengths and Limitations

The current study has notable strengths including the use of validated measures of stress and depressive symptoms administered during structured, in-home interviews. Moreover, study visits occurred across consecutive births, thus extending prior work evaluating the interactive effects of childhood and perinatal stress in predicting perinatal well-being in pregnancy only. The sample included representation from three study sites and was racially and ethnically diverse. Furthermore, measures of perinatal stress were based on report of objective occurrence of life events rather than subjective evaluation of their impact thereby reducing the risk of mood bias in reporting. Reports of depressive symptoms and early life adversity were collected at separate study timepoints and therefore retrospective report of early life stress may be less strongly influenced by mood. Employing multilevel modeling as the analytic method of the current study accounted for repeated measurements within individuals and prevented inflation of the Type I error rate (Musca, 2011).

There are also several limitations that warrant mention. Due to low to moderate frequency of exposure to family stress in most of our sample, the current study was limited in its ability to test the association between stressful life events and depressive symptoms among individuals with higher frequencies of exposure to family stress. Furthermore, we only tested the moderating role of childhood family stress which may not capture other forms of early life stress. The type, timing, and chronicity of childhood stress may also have differential associations with perinatal mental health and serve different roles in modifying the associations between perinatal stress and mental health (Tung et al., 2019). Although rates of childhood family stress were similar to other community samples, future tests in perinatal samples with a wider range of exposure to childhood stress are necessary to replicate these findings. The use of different measures of depressive symptoms is another possible limitation. Although both are validated and show strong correspondence, there may be differences in the degree of symptom severity necessary to score a certain proportion of the maximum score across measures. Additionally, reports of early life stress were limited to retrospective report. This method is validated and commonly used in empirical studies of early life stress, but there is some evidence of disagreement between retrospective and prospective results (Baldwin et al., 2019). All measures in the current sample were self-report, thus increasing risk of shared method bias and precluding causal inference.

Conclusions

The current study used a life course perspective to examine the interactive effects of childhood family social environment and stressful life events in predicting depressive

symptoms during pregnancy and postpartum. We found that there was a positive association between stressful life events and depressive symptoms in the perinatal period among individuals with a low frequency of exposure to family stress in childhood, but not among individuals with more frequent exposure to childhood family stress. This study provides novel evidence that childhood experiences modifying risk for mental health problems in pregnancy and postpartum and highlight the utility of considering the interaction between risk factors across the lifespan in predicting perinatal mental health.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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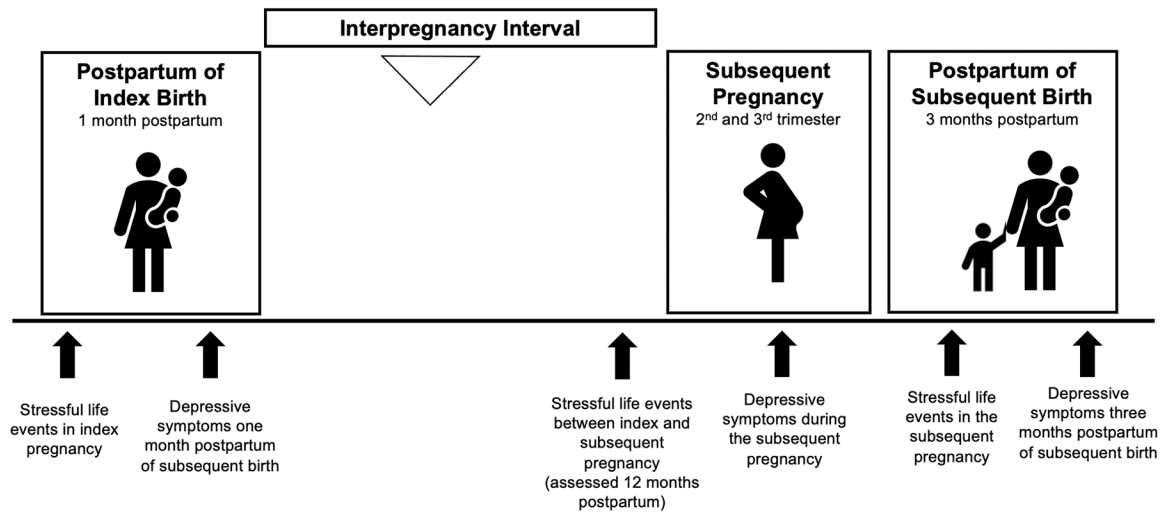


Figure 1.
Study Overview

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Interactive Effect of Childhood Family Stress and Stressful Life Events
on Perinatal Depressive Symptoms

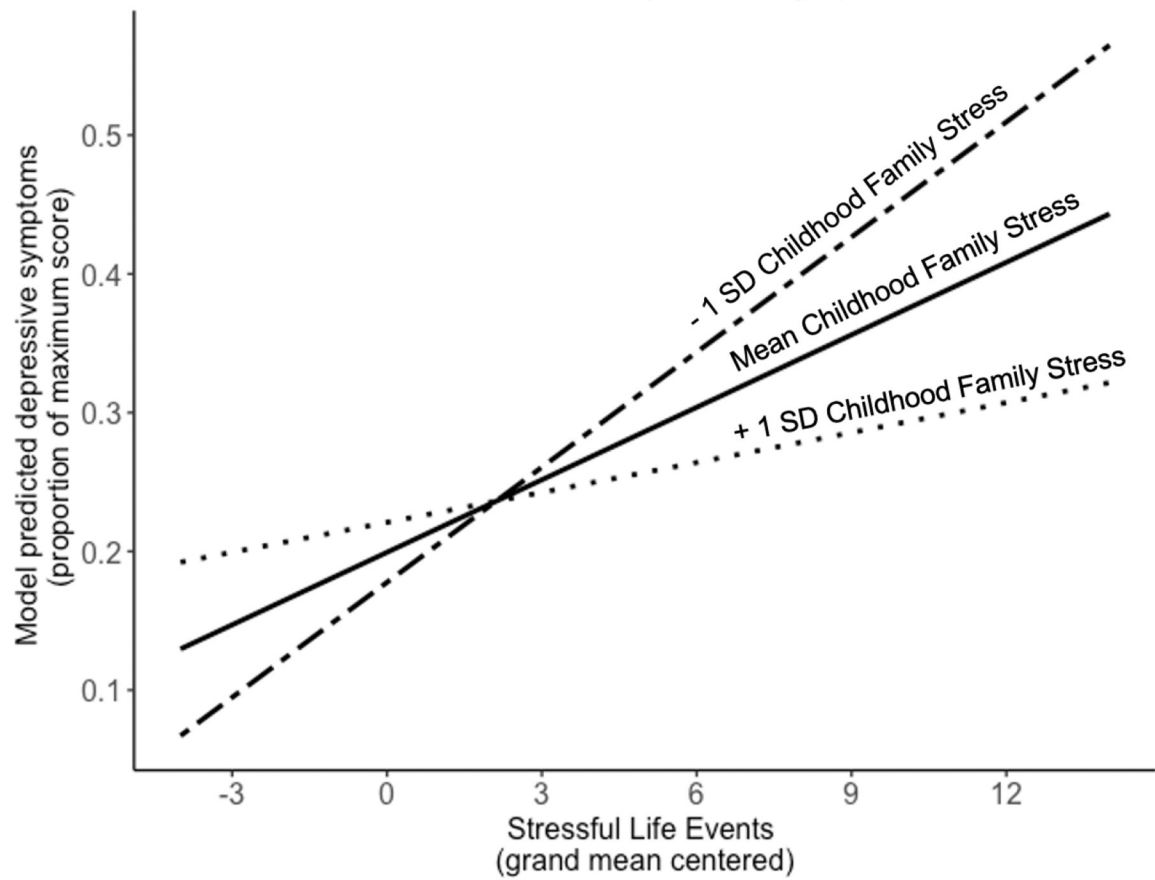


Figure 2. Simple slopes analysis of the association between stressful life events and depressive symptoms by childhood family stress

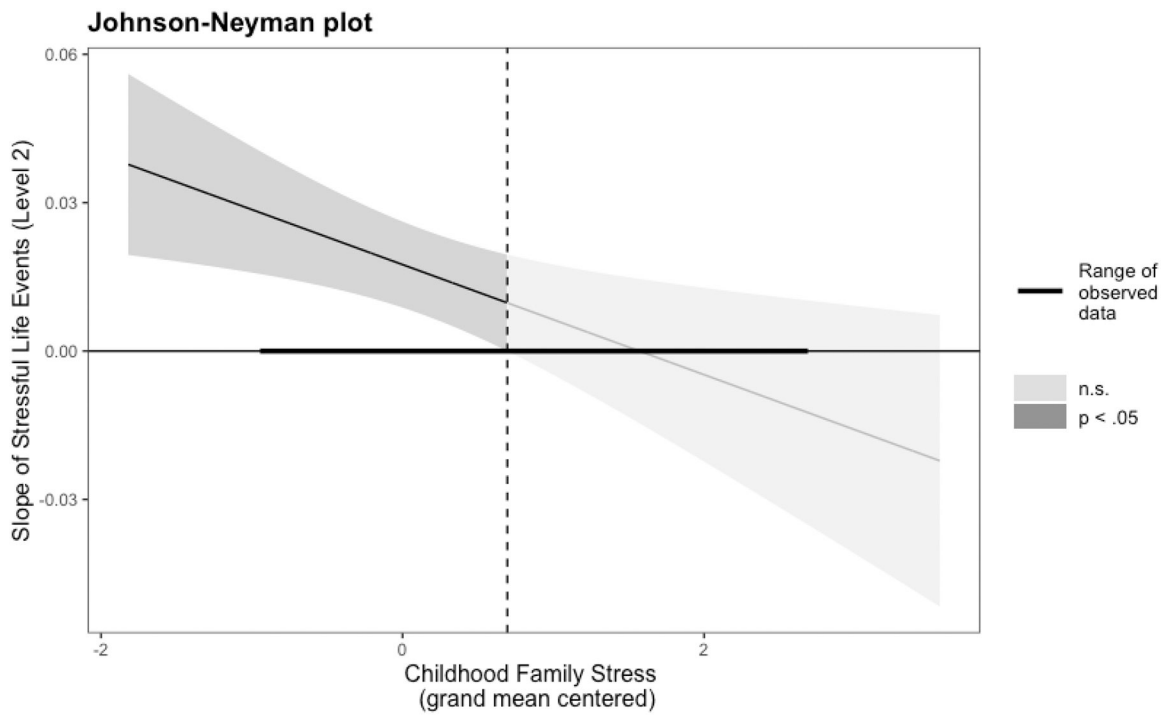


Figure 3.
Johnson-Neyman region of significance analyses

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Table 1.Sample Description ($n = 127$)

	M (SD) or % (n)
Age at enrollment (years)	26.6 (6.1)
Per capita household income (\$)	15,307 (18,384)
Interpregnancy interval (months)	17.6 (13.0)
Education level	
Less than high school	22.8 (29)
High school/GED/Technical	40.9 (52)
Some college	10.2 (13)
College or higher degree	22.8 (29)
Other	3.1 (4)
Relationship status	
Married	46.5 (59)
Cohabiting, not married	30.7 (38)
Single	23.6 (30)
Race/Ethnicity	
Hispanic/Latina	48.8 (62)
Non-Hispanic White	31.5 (40)
Black or African American	21.3 (27)
Parity at index birth	
First birth	51.8 (65)
Not first birth	49.6 (63)
Study Site	
Chicago	77.2 (98)
Washington, D.C.	14.2 (18)
Eastern North Carolina	8.7 (11)

Note. Per capita income adjusted for cost of living at each study site. Some percentages do not add to 100 due to missing data.

Table 2.

Descriptive statistics and bivariate correlations of primary study variables

Variable	<i>M</i>	<i>SD</i>	1	2	3	4	5	6
1. Childhood family stress	1.92	0.86						
2. Stressful life events: Index pregnancy	2.95	2.87	.17 [-.02, .34]					
3. Stressful life events: Interpregnancy interval	3.73	3.21	.13 [-.06, .31]	.48** [.32, .61]				
4. Stressful life events: Subsequent pregnancy	2.54	2.80	.27* [.06, .46]	.43** [.24, .59]	.58** [.42, .71]			
5. Depressive symptoms postpartum index birth (EPDS)	5.05	4.00	.20 [-.00, .38]	.12 [-.08, .31]	.09 [-.11, .28]	.31** [.09, .50]		
6. Depressive symptoms subsequent pregnancy (CES-D)	17.07	4.90	.20* [.00, .38]	.24* [.04, .42]	.38** [.19, .54]	.50** [.32, .64]	.28** [.07, .46]	
7. Depressive symptoms postpartum subsequent birth (EPDS)	4.04	4.27	.21* [.01, .39]	.12 [-.08, .31]	.17 [-.03, .35]	.19 [-.03, .39]	.31** [.11, .49]	.50** [.32, .64]

Note. *M* and *SD* are used to represent mean and standard deviation, respectively. Values in square brackets indicate the 95% confidence interval for each correlation.

* $p < .05$.

** $p < .01$.

Table 3

Mixed Linear Model Results

	Unconditional Model, Intercept Only			Unconditional Model, Linear Effect of Time			Random Intercept Model with Main Effects Only			Random Intercept Model with Main and Interactive Effects			Random Slope Model with Main and Interactive Effects		
	B	SE	95% CI	B	SE	95% CI	B	SE	95% CI	B	SE	95% CI	B	SE	95% CI
Fixed Components															
Intercept	0.190***	0.011	0.169, 0.212	0.173***	0.014	0.145, 0.200	0.198	0.028	0.145, 0.251	0.204***	0.027	0.151, 0.256	0.205***	0.027	0.153, 0.256
Level 1															
Stressful life events							-0.002	0.007	-0.016, 0.011	-0.007	0.007	-0.020, 0.007	-0.006	-0.008	-0.022, 0.009
Stressful life events x childhood family stress										-0.003	0.008	-0.018, 0.012	0.002	0.009	-0.015, 0.021
Time							0.003**	0.009	0.008, 0.044	0.032**	0.010	0.012, 0.052	0.032**	0.010	0.013, 0.050
Level 2															
Stressful life events							0.017***	0.005	0.012, 0.052	0.018***	0.005	0.009, 0.027	0.018***	0.005	0.009, 0.027
Childhood family stress							0.014	0.014	-0.012, 0.040	0.026^	0.014	-0.001, 0.053	0.026^	0.014	-0.001, 0.005
Stressful life events x childhood family stress										-0.011*	0.005	-0.020, -0.003	-0.011*	0.005	-0.020, -0.002
- 1 SD										0.028***	0.007	0.016, 0.041	0.028***	0.006	0.015, 0.041
Mean										0.019***	0.005	0.009, 0.028	0.018***	0.005	0.009, 0.027
+ 1 SD										0.009	0.006	-0.003, 0.020	0.008	0.006	-0.003, 0.020
Interpregnancy interval							-0.002^	0.001	-0.004, 0.000	-0.002^	0.001	-0.004, 0.000	-0.002^	0.001	-0.004, -0.000
Relationship status							-0.013	0.027	-0.065, 0.039	-0.018	0.027	-0.069, 0.033	-0.022	0.027	-0.070, -0.031
Primiparity							-0.031	0.023	-0.076, 0.014	-0.025	0.023	-0.070, 0.019	-0.025	0.023	-0.070, 0.019

Per capita income									
Variance Components	Variance	SD	Variance	SD	Variance	SD	Variance	SD	Variance
Intercept	0.006	0.077	0.006	0.080	0.005	0.070	0.005	0.070	0.007
Residual	0.023	0.153	0.024	0.154	0.023	0.152	0.022	0.150	0.018
Stressful life events									0.001
ICC		0.19		0.19		0.18		0.18	
Deviance		-250.53		-252.54		-219.42		-230.73	
Log-likelihood ratio test									

^a $p < .10$;
 * $p < .05$;
 ** $p < .01$;
 *** $p < .001$

^a Log-likelihood ratio test of whether the random slope model significantly improved model fit from the random intercept model.

Note. B = unstandardized coefficient; SE = standard error; CI = confidence interval; ICC = intraclass coefficient. Simple slopes were probed if interaction term was significant. For visual clarity, covariates are not included in the table. Models adjusted for time since study start, primiparity, income, relationship status, and interpregnancy interval. All continuous predictors and covariates were mean centered for ease of interpretation. Stressful life events were centered within cluster (Level 1) and grand mean centered (Level 2).