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Regular Article

Associations of prenatal stress with 5-year-old children's executive function in a low socioeconomic status population

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Abstract

Prenatal stress has a significant, but small, negative effect on children's executive function (EF) in middle and high socioeconomic status (SES) households. Importantly, rates and severity of prenatal stress are higher and protective factors are reduced in lower SES households, suggesting prenatal stress may be particularly detrimental for children's EF in this population. This study examined whether prenatal stress was linked to 5-year-old's EF in a predominantly low SES sample and child sex moderated this association, as males may be more vulnerable to adverse prenatal experiences. Participants were 132 mother-child dyads drawn from a prospective prenatal cohort. Mothers reported on their depression symptoms, trait anxiety, perceived stress, everyday discrimination, and sleep quality at enrollment and once each trimester, to form a composite prenatal stress measure. Children's EF was assessed at age 5 years using the parent-report Behavior Rating Inventory of Executive Function - Preschool (BRIEF-P) Global Executive Composite subscale and neuropsychological tasks completed by the children. Mixed models revealed higher prenatal stress was associated with lower BRIEF-P scores, indicating better EF, for females only. Higher prenatal stress was associated with lower performance on neuropsychological EF measures for both males and females. Results add to the limited evidence about prenatal stress effects on children's EF in low SES households.

Keywords: early childhood; executive function; pregnancy; prenatal stress; sex differences; socioeconomic status

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Introduction

Depression symptoms, anxiety symptoms, and perceived stress during pregnancy – collectively referred to as prenatal stress – are some of the most common medical complications affecting pregnant individuals (Babineau et al., 2022). Approximately 1 in 7 pregnant individuals experience depression during pregnancy, 1 in 4 experience anxiety, and 1 in 3 experience mild to moderate stress (ACOG, 2018; Field, 2017). Critically, prenatal stress is twice as common and more severe in pregnant individuals from lower SES households compared to middle and high SES households (Goyal et al., 2010).

Prenatal stress doubles children's risk for developing mental health problems, including internalizing and externalizing behavior problems (Monk et al., 2019). One of the ways prenatal stress may increase children's risk for psychopathology is via deficits in executive function (EF). Executive function (EF) is the set of higher-order cognitive abilities needed for carrying out goal-directed behavior in cognitively demanding situations (Diamond, 2013). By age 5 years, EF difficulties are a trans-diagnostic risk factor for psychosocial adjustment, psychopathology, and developmental difficulties across the lifespan (Zelazo, 2020). In addition, males and females are theorized to be differentially impacted by prenatal exposures, with males more likely to be adversely impacted by prenatal stress (Sandman et al., 2013).

Most previous research has examined the impact of prenatal stress on children's EF in middle and high SES households (Power et al., 2021). A better understanding of the association between prenatal stress and children's EF in lower SES households is essential for understanding the generalizability of findings from middle and high SES households and for identifying the specific needs of this higher risk population. The goals of the present study are to examine associations between prenatal stress and children's EF at age 5 years, and child sex as a moderator of these associations, in primarily low SES families.

Executive function in early childhood

EF is typically conceptualized as including inhibitory control (the ability to stop an automatic or prepotent response), cognitive

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flexibility (the ability to modify thoughts and behaviors in response to changing circumstances), working memory (the ability to hold in mind and manipulate information), and higher-level cognitive processes like planning (the ability to identify a goal, and plan and execute the steps required to achieve the goal; Diamond, 2013). In early childhood, the cognitive abilities comprising EF may be best accounted for by a single factor, rather than more complex, multi-factor models supported in older age groups (Willoughby et al., 2010, 2012).

Early childhood is a period of substantial quantitative and qualitative EF development. EF emerges in infancy and individual differences in EF are moderately stable by age 2 years (Carlson et al., 2004; Diamond, 2013). Between ages 3 and 5 years accuracy and reaction time on EF tasks improve most rapidly and children transition from perseverating on cognitive flexibility tasks to being able to appropriately shift sets (Blakey et al., 2016; Wiebe et al., 2012).

Prenatal stress exposure, socioeconomic status, and children's executive function

The Developmental Origins of Health and Disease (Gillman, 2005) and Fetal Programming (Barker, 2004) hypotheses argue that the fetal environment has a life-long impact on offspring's health and development because gestation is a critical period for brain and stress physiology development. Prenatal stress is associated with alterations in the development of the neural systems underlying EF, including the prefrontal cortex (Sandman et al., 2015), hypothalamic-pituitary-adrenal axis (Glover et al., 2010), and neurotransmitter systems like the dopaminergic system (Pastor et al., 2017), suggesting that prenatal stress is likely to impact children's EF. Consistent with this suggestion, a growing number of pregnancy cohort studies have found a negative effect of prenatal stress exposure on children's inhibitory control, cognitive flexibility, and working memory across childhood and adolescence (Babineau et al., 2022; Buss et al., 2011; Pearson et al., 2016), with a recent meta-analysis reporting a statistically significant but small (Cohen's $d = .14$) adverse effect of prenatal stress on children's EF between the ages of 5 months and 15 years (Power et al., 2021).

Research examining the association between prenatal stress and children's EF has been primarily conducted with middle and high SES samples, characterized by pregnant individuals with postsecondary educations and household incomes greater than $50,000 (Power et al., 2021). Research on middle and high SES families is likely to be inadequate in determining the effect of prenatal stress on children's EF. This is because stress during pregnancy is both more common and more severe among pregnant individuals from lower SES households – defined here as both lower maternal education and household income – than those from middle and high SES households (Goyal et al., 2010). In part, this reflects differences in exposure to social determinants of health. Individuals from low SES households are more likely to experience major stressors like job and food insecurity, poor work conditions, issues with housing quality and neighborhood safety, discrimination, and reduced access to affordable and high-quality health services (Maggi et al., 2010). Pregnant individuals from low SES households are also likely to have fewer protective factors that buffer the adverse effects of stressors on them and their children, such as the presence of a partner in the home, access to social support, and social capital (Nagy et al., 2020). Prenatal stress is likely to have a greater adverse impact on children's EF in lower SES households given the higher risk nature of this population.

Few studies have examined the impact of prenatal stress on children's EF in early childhood (≤5 years) specifically and their findings conflict. Studies that report a negative association have primarily assessed EF using parent-report measures, such as the *Behavior Rating Inventory of Executive Function - Preschool* (BRIEF-P; El Marroun et al., 2017; Plamondon et al., 2015). In contrast, studies that used neuropsychological tasks to assess EF, such as the *Attentional Network Task*, have generally not reported a statistically significant negative association between prenatal stress and children's EF (Babineau et al., 2022; Nolvi et al., 2018). Importantly, parents with mental health problems tend to overreport their children's negative behaviors, and this may inflate estimates of the association between parental stress and parent-report child outcomes (Ringoot et al., 2015). In addition, correlations between EF questionnaires and neuropsychological tasks tend to be low (Vrantsidis, Wuest, et al., 2022). The low correlations are attributable to questionnaires and neuropsychological tasks assessing different aspects of EF. The mixed findings regarding the effect of prenatal stress on children's EF in early childhood might reflect measurement differences rather than true differences in EF.

The association between prenatal stress and children's EF may reflect a passive gene × environment correlation rather than a direct effect of prenatal stress on children's EF. A passive gene × environment correlation occurs when parents that are genetically related to the child provide an environment that is correlated with the genotype of the child (Scarr & McCartney, 1983). Maternal EF is related to physiological regulation of stress (e.g., cortisol levels in response to a stressor) and child EF (Bridgett et al., 2015). In support of a passive gene × environment correlation, studies that have examined the impact of maternal IQ, a construct that overlaps with EF, on associations between prenatal and early postnatal maternal stress and children's EF found that controlling for IQ attenuated or eliminated the association between stress and child outcomes (Faleschini et al., 2019; Pearson et al., 2016). The present study included a measure of maternal EF to help minimize the effect of a passive gene × environment correlation on the association between prenatal stress and children's EF.

Sex differences in prenatal stress effects on children's executive function

Limited but growing evidence suggests that adverse prenatal and early postnatal experiences, such as prenatal substance exposure or less responsive parental behavior, may be particularly detrimental for EF in males compared to females (Vrantsidis, Wakschlag, et al., 2022; Wiebe et al., 2015). Higher prenatal stress has been linked to lower inhibitory control in 4- to 8-year-old males but not females (Babineau et al., 2022). Similarly, higher prenatal stress is associated with lower working memory at age 4 years but only for males who experienced less maternal sensitivity at age 4 years (Plamondon et al., 2015). Finally, for 6-year-old males, but not females, cortisol reactivity mediates the association between prenatal stress and EF (Neuenschwander et al., 2018). However, results are not always consistent as higher prenatal stress is also linked to lower inhibitory control for 6- to 9-year-old females but not males (Buss et al., 2011). The reasons for increased male vulnerability are unclear. Increased vulnerability may reflect sex differences in in-utero androgen and testosterone exposure (Del Giudice et al., 2018). Higher androgen and testosterone exposure are associated with increased sensitivity to environmental stimuli. Increased male vulnerability may also reflect differences in fetal

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development. Compared to female fetuses, male fetuses undergo more physical growth, which makes them less able to adapt to prenatal insults and more vulnerable to developmental and cognitive difficulties (Sandman et al., 2013). Because males may be more vulnerable to the adverse effects of prenatal stress, this study also examined child sex as a moderator of the effect of prenatal stress on children's EF.

The present study

The current report used data from a pregnancy cohort to achieve two aims. First, in a predominantly low SES cohort, we examined whether a composite index of maternal prenatal stress (depression symptoms, trait anxiety, and perceived stress) across pregnancy was related to children's EF at age 5 years as assessed using both parent-report measures and neuropsychological tasks, while controlling for key confounders like maternal EF. Consistent with previous research (El Marroun et al., 2017; Power et al., 2021), we hypothesized that higher prenatal stress would be associated with lower child EF. Second, we examined whether child sex moderated the effect of prenatal stress on child EF. Consistent with a male vulnerability model (Sandman et al., 2013), we hypothesized that higher prenatal stress would be associated with lower EF for males but not females.

Methods

Study design and participants

Mother-child dyads ($N = 132$) included in the present analyses were drawn from the Lifestyle and Early Achievement in Families (LEAF) study, a primarily low SES, Black cohort prospectively recruited during pregnancy to study the effects of lifestyle exposures on child development (Klebanoff et al., 2020). Sample demographic information is presented in Table 1.

Individuals receiving prenatal care from clinics at a university affiliated medical center in the Midwest, United States were recruited during pregnancy to participate in a general-purpose perinatal research repository ($N = 497$). To be eligible for the repository, pregnant individuals needed to be between ages 16 to 50 years, able to communicate in English, and intend to deliver at the medical center. Thirty nine percent ($n = 194$) of mothers enrolled during their first trimester, 55% ($n = 273$) during their second, and 6% ($n = 30$) during their third.

When children were between the ages of 3.5 and 7 years, families who consented to be contacted to participate in future research ($n = 360$) were invited to participate in up to two LEAF follow-up visits depending on their child's age. Follow-up visits were at child ages 3.5 years, 5 years, and 7 years. Recruitment for the age 5-year follow-up was not attempted for five families (not age eligible: $n = 3$; no attempt to recruit: $n = 2$). Of the 355 families contacted to participate in the follow-up, 105 families did not complete the follow-up visit. Reasons for not completing the visit include being unable to locate the family or schedule them for a visit ($n = 70$), families refused to participate ($n = 29$), or Child Protective Services had custody of the child ($n = 6$). Sixty-nine percent ($n = 250$) of families with age-eligible children participated in the age 5-year follow-up. Of the 250 families, 128 were excluded because of missing data on the age 5-year child EF measures, prenatal stress measures, or covariates. Reasons for missing data on the child EF measures included the child had difficulty completing the behavioral tasks and the family cut the age 5-year visit short. Missing data on the prenatal stress measures were due to individuals registering late for prenatal care, not attending all indicated prenatal care visits, or no opportunity during the visit to approach individuals to complete the questionnaires. Missing data on covariates were primarily due to the biological mother not being able to complete the age 5-year maternal EF tasks. Mothers provided informed consent when they enrolled into the general-purpose perinatal research repository and again when their child came in for their first LEAF study visit.

Attrition analyses comparing (1) families who consented to participate in future research ($n = 360$) to families who participated in the age 5-year follow-up ($n = 250$), and (2) families

Table 1. Sample demographic information

Construct	M (SD)/N (%)
Child sex (% male)	63 (48%)
Child age (years)	5.45 (.29)
Child race and ethnicity (maternal report):	
Non-Hispanic Black	84 (64%)
Non-Hispanic White	18 (14%)
Hispanic	5 (4%)
Other	3 (2%)
More than one race or ethnicity	22 (17%)
Maternal self-reported race and ethnicity:	
Non-Hispanic Black	86 (65%)
Non-Hispanic White	33 (25%)
Hispanic	2 (2%)
Other	4 (3%)
More than one race or ethnicity	7 (5%)
Marital status:	
Never married or cohabitating	80 (60%)
Married or cohabitating	39 (30%)
Separated or divorced	13 (10%)
Household income:	
< $10,000 per year	62 (47%)
$10,000–$19,999 per year	30 (23%)
$20,000–$29,999 per year	5 (4%)
$30,000–$39,999 per year	4 (3%)
≥ $40,000 per year	2 (2%)
Missing	29 (22%)
Maternal education:	
Less than high school graduate	29 (22%)
High school graduate or GED	46 (35%)
Some college	44 (33%)
College graduate	13 (10%)
Substance use during pregnancy:	91 (69%)
Tobacco	54 (41%)
Marijuana	42 (32%)
Alcohol	34 (26%)
Any other substance	25 (19%)

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included in the final sample ($n = 132$) to families who completed the age 5-year follow-up were conducted. Families who participated in the 5-year follow-up did not significantly differ from families who did not in terms of demographic characteristics or prenatal stress. Families included in the final sample had higher prenatal stress (t (193) = 2.21, $p = .03$) and lower SES ($b = -.31$, $p = .02$) than families excluded due to missing data. Families included and excluded from the final sample did not significantly differ in terms of maternal self-reported race or ethnicity or child sex.

Procedures

At enrollment into the perinatal research repository, mothers completed a demographic questionnaire. Additionally, at enrollment and once each trimester during pregnancy, mothers completed the same five self-report questionnaires assessing depression symptoms, trait anxiety, and perceived stress. Mothers completed all questionnaires during prenatal care visits. At the age 5-year follow-up, mother-child dyads visited a clinical research laboratory at a pediatric hospital. In separate rooms, children completed a battery of neurocognitive tasks and mothers completed a battery of neurocognitive tasks, and background, demographic, and child EF questionnaires. Visits lasted for approximately two to three hours. A complete list of test batteries at each timepoint can be found in Klebanoff et al. (2020). Study procedures were approved by the hospital's Institutional Review Board.

Measures

Prenatal stress

Mothers completed five self-report questionnaires assessing depression symptoms (*Center for Epidemiological Studies Depression Scale*), trait anxiety (*State-Trait Anxiety Inventory*), and perceived stress (*Perceived Stress Scale-10*; *Everyday Discrimination Scale*; and *Pittsburgh Sleep Quality Index*).

The *Center for Epidemiological Studies Depression Scale* (CESD; Radloff, 1977) consists of 20 items assessing how often over the past week the rater has experienced symptoms associated with depression. Items, such as "I felt lonely", were rated on a 4-point scale ranging from 0 ("Less than 1 day") to 3 ("5–7 days"). Scores on each item were summed and divided by the number of items answered to create a measure of depression at each trimester. Higher scores indicated more depression symptoms. The measure had excellent internal consistency across trimesters ($\alpha s = .87–.92$) and is a valid and reliable measure of maternal depression in diverse populations (Radloff, 1977).

The *State-Trait Anxiety Inventory* (STAI; Speilberger, 1983) consisted of 40 items assessing state and trait anxiety. Participants completed the 20 items assessing trait anxiety. Statements such as "I feel nervous and restless" were rated on a 4-point Likert scale ranging from 1 ("Almost Never") to 4 ("Almost Always"). Scores on each item were summed and divided by the number of items answered to create an anxiety measure. Higher scores indicated higher trait anxiety. At each trimester, the measure had excellent internal consistency ($\alpha s = .88–.92$). The STAI has established validity and reliability among pregnant individuals (Gunning et al., 2010).

The *Perceived Stress Scale-10* (PSS; Cohen et al., 1983) consisted of 10 items assessing the perception of stress. Questions, such as "In the last month, how often have you felt stressed?", were rated on a 5-point scale ranging from 0 ("Never") to 4 ("Very often"). To create a measure of perceived stress, scores on each item were summed and divided by the number of questions answered. Higher scores indicated more perceived stress. The measure had excellent internal consistency at each trimester ($\alpha s = .88–.93$) and is a validated, reliable measure of perceived stress (Cohen et al., 1994).

The *Everyday Discrimination Scale* (EDS; Williams et al., 1997) consists of 9 items assessing the frequency of routine, subtle experiences of discrimination in everyday situations. Items, such as "You were treated with less courtesy than other people?", are rated on a 6-point scale ranging from 1 ("Almost every day") to 6 ("Never"). Scores on each question were summed and divided by the number of questions answered to create a measure of perceived discrimination at each trimester. Higher scores indicated more perceived discrimination. The measures had excellent internal consistency ($\alpha s = .90–.93$). EDS scores are correlated with measures of psychological distress (Krieger et al., 2005).

The *Pittsburgh Sleep Quality Index* (PSQI; Buysse et al., 1998) consists of 19 items assessing sleep quality and disturbances over a 1-month period. The 19 items are combined into seven clinically derived component scores, including subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. Each component score yields an ordinal score ranging from 0 (least dysfunction) to 3 (greatest dysfunction). Component scores were summed to create a total global score which was used as the measure of sleep quality. Higher scores indicated worse sleep quality. The sleep quality measure at each trimester had good internal consistency ($\alpha s = .71–.98$). PSQI scores are correlated with perceived stress (Kashani et al., 2012).

Scores on the CESD (F (2, 224) = 2.51, $p = .08$), STAI (F (2, 193) = .08, $p = .93$), PSS (F (2, 223) = .69, $p = .50$), EDS (F (2, 192) = 2.06, $p = .13$), and PSQI (F (2, 217) = .20, $p = .82$) did not differ significantly across trimesters. Therefore, scores on each questionnaire were averaged across trimesters. To create a composite score for the prenatal stress measures, we conducted a principal components analysis (PCA) using an oblique rotation (oblimin). The PCA extracted one factor for the five average scores ($\lambda = 3.34$) that accounted for 67% of variance. Individual factor loadings ranged from .59 to .93. Based on these results, the five average scores were converted to z-scores and averaged to create a composite score capturing prenatal stress across pregnancy.

Executive function

Children's EF was assessed at the age 5-year follow-up using both parent-report measures and neuropsychological tasks. Mothers completed the BRIEF-P (Gioia et al., 2003), a 63-item parent-report measure of children's EF in everyday contexts. Mothers rated how often during the past 6 months a variety of behaviors (e.g., their child overreacts to small problems) have been a problem on a 3-point scale ranging from 1 ("Never") to 3 ("Often"). Raw scores for the Global Executive Composite (GEC) were converted to age-corrected t-scores and used as the measure of EF. This measure had excellent internal consistency ($\alpha = .97$). Higher scores indicated lower EF.

Children completed an iPad version of the NIH Toolbox Early Childhood Cognition Battery (Weintraub et al., 2010), a standardized measure of cognitive ability that includes tasks assessing inhibitory control (*Flanker*), cognitive flexibility (*Dimension Change Card Sort*), and working memory (*List Sorting Working Memory*). Age-corrected standard scores were used as the dependent measures. Lower scores indicated lower EF.

To assess planning, children completed the *Tower of Hanoi* (Bull et al., 2004). Children completed three practice problems and up to six test problems, each with a maximum of two trials. Test

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problems increased in difficulty. The first problem required a minimum of two moves to solve and the sixth problem required a minimum of seven moves. If the child broke a rule or did not solve the problem in 20 moves, the trial was scored as a failure. The task ended when children failed two practice problems and the first test problem or two consecutive test problems. Each problem received a score based on the minimum number of moves required to solve it (e.g., problem one received a point value of two and problem six received a point value of seven). If the child solved the problem on the first trial, they received full points. If they solved the problem on the second trial, they received half the number of points. The total number of points per problem were summed to create a planning score ranging from 0 to 27 (Murnan et al., 2021). Interrater reliability was excellent ($\kappa s \geq .99$; $M \geq 99\%$). Lower scores indicated lower EF.

To create a composite score for the neuropsychological tasks, we conducted a PCA using an oblique rotation (oblimin). *List Sorting Working Memory* was excluded from the PCA because the Early Childhood Cognition Battery excludes the task from composite scores (Hook & Giella, 2023). The PCA extracted one factor for the three remaining tasks ($\lambda = 1.72$) that accounted for 57% of variance. Individual factor loadings ranged from .61 to .85. Based on the results of the PCA, a composite score for *Flanker*, *Dimension Change Card Sort*, and *Tower of Hanoi* was created by averaging the z-scores for each task. Lower scores indicated lower EF.

Covariates

Household socioeconomic status, maternal self-reported race and ethnicity, EF, substance use during pregnancy, and marital status were adjusted for in the analyses because they were identified a priori as possible confounders (Murnan et al., 2021; Vrantsidis et al., 2020, 2023). These variables temporally preceded both prenatal stress and child EF and previous research found associations with both constructs (Rothman & Greenland, 1998). Maternal depression and anxiety symptoms at the age 5-year follow-up were also controlled for because of their concurrent association with children's EF (Vrantsidis et al., 2023). At enrollment into the general research repository, mothers reported on their highest educational degree completed and household income. The average z-score of these two measures was used as the measure of household SES. Maternal self-reported race and ethnicity at the age 5-year follow-up were used if available. If these data were missing, maternal self-reported race and ethnicity at enrollment into the general research repository were used. Race and ethnicity were coded using a set of dummy codes with non-Hispanic Black serving as the reference. At the age 5-year follow-up, mothers completed the adult Cognitive Battery of the NIH Toolbox (Gershon et al., 2013). The fluid cognition composite score was used as the measure of maternal EF (Murnan et al., 2021). Pregnant individuals were deemed to have used substances during pregnancy if they used one or more of marijuana, alcohol, tobacco, or 16 other drugs (e.g., cocaine or methamphetamine) as indicated via maternal self-report at enrollment, noted use on an obstetric record abstraction, or a positive urine test at enrollment or any trimester for marijuana or one of the 16 other drugs screened for (Klebanoff et al., 2020). Substance use was dummy coded (no substance use = 0; use of one or more substances = 1). Mothers reported on their marital status at enrollment into the general research repository. Marital status was dummy coded (married or cohabitating = 0; not married or not cohabitating = 1). At the age 5-year follow-up, mother's completed the Adult Self Report (Achenbach & Rescorla, 2003), a questionnaire assessing adaptive functioning and problems. *T* scores on the anxious/depressed subscale were used as the measure of maternal depresison and anxiety.

Analytic strategy

Because 13 (10%) children included in the final sample were siblings, mixed models were used to examine the effect of prenatal stress on children's EF. Family relationship was included as a random effect to account for the dependence among sibling participants. Analyses were run separately for the neuropsychological composite and BRIEF-P GEC. Predictors in each model were prenatal stress, child sex (dummy coded as males = 0, females = 1), and the prenatal stress × child sex interaction term. If the interaction term was not significant ($p > .05$), it was trimmed from the model. Significant interactions were probed using simple slopes analyses. Models were run twice: once without adjustment for covariates and once with adjustment for covariates. Effect sizes were estimated using Nakagawa and Schielzeth's (2013) r^2.

Results

Descriptive statistics

Descriptive statistics are presented in Table 2. The cohort had lower educational attainment than the US population and the majority of families had incomes less than two times the federal poverty threshold for a 1-parent, 1-child household (< $30,000) (U.S. Census Bureau, 2011, 2014). Depression symptoms, trait anxiety, perceived stress, and sleep quality scores were lower in the present sample compared to primarily middle and high SES cohorts (Babineau et al., 2022; Hackman et al., 2015; Huizink et al., 2017; Sedov et al., 2018). Everyday discrimination scores were comparable to those of middle SES Hispanic and non-Hispanic Black cohorts (Fazeli Dehkordy et al., 2016).

The prenatal stress composite was significantly but modestly correlated with the neuropsychological EF composite ($r = -.21$, $p = .02$). The correlations between the BRIEF-P GEC and prenatal stress composite ($r = -.08$, $p = .39$) and neuropsychological EF composite ($r = .08$, $p = .37$) were not statistically significant. Males had lower neuropsychological EF composite scores ($M = -.23$, SD = .80) than females ($M = .17$, SD = .73; t (128) = −2.96, $p = .004$). Males and females did not significantly differ on the BRIEF-P GEC (t(130) = −1.56, $p = .12$) or prenatal stress exposure (t (130) = .06, $p = .96$). Neuropsychological EF (F (4, 125) = 1.99, $p = .10$), BRIEF-P GEC (F (4, 127) = .44, $p = .78$), and prenatal stress (F (4, 127) = .97, $p = .43$) did not significantly differ by maternal self-reported race or ethnicity. Neuropsychological EF ($b = .02$, $p = .80$), BRIEF-P GEC ($b = .36$, $p = .79$), and prenatal stress ($b = -.13$, $p = .12$) were not significantly related to household SES.

Mixed model results

Results of the mixed models, with and without adjustment for covariates, are presented in Table 3. In the model unadjusted for covariates, the prenatal stress × child sex interaction term ($b = -.03$, $SE = .13$, $p = .85$) was not significant so it was trimmed from the model. The association between prenatal stress and neuropsychological EF was statistically significant. Higher prenatal stress was associated with lower neuropsychological EF ($r^2 = .11$). When analyses were rerun adjusting for covariates, the interaction

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Table 2. Descriptive statistics for the measures of prenatal stress and maternal and child executive function

Construct	*N*	*M*	SD	Range
CESD first trimester	39	0.86	0.49	0.15–2.11
CESD second trimester	86	0.85	0.61	0.00–2.63
CESD third trimester	102	0.69	0.49	0.00–2.00
CESD pregnancy average	129	0.78	0.49	0.00–2.00
STAI first trimester	39	1.87	0.47	1.00–2.90
STAI second trimester	81	1.91	0.54	1.00–3.18
STAI third trimester	75	1.88	0.56	1.00–3.47
STAI pregnancy average	128	1.92	0.52	1.00–3.47
PSS first trimester	38	1.59	0.66	0.20–3.10
PSS second trimester	81	1.72	0.82	0.00–3.40
PSS third trimester	77	1.60	0.79	0.00–3.90
PSS pregnancy average	128	1.67	0.72	0.20–3.90
EDS first trimester	38	1.75	0.93	1.00–4.00
EDS second trimester	88	2.01	1.13	1.00–5.00
EDS third trimester	100	1.70	0.92	1.00–5.44
EDS pregnancy average	129	1.89	1.04	1.00–5.44
PSQI first trimester	38	1.08	0.46	0.29–2.14
PSQI second trimester	85	1.15	0.54	0.17–2.57
PSQI third trimester	97	1.14	0.51	0.143–2.29
PSQI pregnancy average	129	1.12	0.49	0.143–2.29
Prenatal stress average	132	−0.01	0.82	−1.47–2.45
Maternal depression and anxiety (age 5-year follow-up)	132	54.11	6.60	50–88
Flanker	126	95.71	14.79	50–123
Dimensional change card sort	125	91.70	13.29	60–120
List sorting working memory	73	80.77	11.10	58–109
Tower of Hanoi	127	11.90	9.18	0.00–27
Neuropsychological executive function composite	130	−0.02	0.78	−2.19–1.55
BRIEF-P Global Executive Composite	132	77.30	12.62	43–103
Maternal executive function (fluid cognition composite)	132	87.99	15.57	56–134

term was trimmed from the model because it was not significant ($b = -.05$, $SE = .13$, $p = .71$). Higher prenatal stress continued to be significantly associated with lower neuropsychological EF ($r^2 = .23$).

For the BRIEF-P GEC, the interaction between prenatal stress and child sex on BRIEF-P GEC was statistically significant ($r^2 = .08$) and therefore retained in the unadjusted model. The interaction was probed using simple slopes and results are presented in Figure 1. The effect of prenatal stress on BRIEF-P GEC was statistically significant for females ($b = -4.43$, $SE = 1.74$, $p = .01$), such that higher prenatal stress was associated with lower BRIEF-P GEC, indicating better EF. The association between prenatal stress and the BRIEF-P GEC was not statistically significant for males ($b = 2.60$, $SE = 1.89$, $p = .17$). Analyses were rerun with covariates and results were unchanged ($r^2 = .09$).

Discussion

This study examined whether prenatal stress was related to children's EF in early childhood and whether the effect of prenatal stress differed for males and females in families from predominantly low SES households. To test these questions, we adopted a multi-method approach to assessing children's EF, using both parent-report measures and child-completed neuropsychological tasks. We hypothesized that higher prenatal stress would be associated with lower child EF and that males would be more vulnerable to the adverse effect of prenatal stress. Results were partially consistent with our hypotheses. Higher prenatal stress was associated with lower EF as reflected by poorer performance on neuropsychological tasks. Child sex did not moderate this association. Unexpectedly, higher prenatal stress was associated with lower BRIEF-P GEC, indicating better EF, but only for females. The association between prenatal stress and the BRIEF-P GEC was not statistically significant for males.

Higher prenatal stress was moderately associated with lower EF as reflected by neuropsychological task performance in low SES families. This result extends findings from research on middle and high SES households that reported a small negative effect of prenatal stress on children's EF (Power et al., 2021). The larger adverse effect of prenatal stress may reflect lower SES families' higher exposure to stressors and reduced access to protective factors that can buffer the adverse effect of prenatal stress. For example, maternal responsiveness, social support, and cognitive stimulation tend to be higher in high SES households and buffer the negative effect of prenatal stress on children's attention and externalizing problems (Nolvi et al., 2023; Vrantsidis et al., 2020). Attention and externalizing problems are associated with EF deficits (Zelazo, 2020), suggesting that these environmental factors are likely to ameliorate the adverse impact of prenatal stress on children's EF as well. In early childhood, environmental influences on children's EF development are particularly robust (Diamond, 2002). Thus, among higher risk families, early childhood interventions to improve children's EF may be beneficial.

The association between prenatal stress and task-based EF also highlights the importance of considering the role of the fetal environment in individual differences in EF and has potential implications for understanding pathways from prenatal stress to the development of psychopathology. The present findings are consistent with the Developmental Origins of Health and Disease (Gillman, 2005) and Fetal Programming (Barker, 2004) hypotheses, which argue that the in-utero environment has a long-term impact on children's health and development. Importantly, this study was not designed to examine mechanisms linking prenatal stress to children's EF. Prenatal stress is likely to impact children's EF through multiple pathways, including epigenetics; inflammatory processes; alterations in stress physiology; neurotransmitter system development; and environmental differences, such as changes in parental behavior (Monk et al., 2019; Vrantsidis et al., 2020). Furthermore, these pathways also link prenatal stress to adverse mental and physical health outcomes across the lifespan (Monk et al., 2019). Therefore, EF deficits in early childhood may be a potential endophenotype for health and well-being across the lifespan in the context of prenatal adversity.

Child sex moderated the effect of prenatal stress on the BRIEF-P GEC. Higher prenatal stress was significantly associated with lower BRIEF-P GEC scores for females, indicating better EF, but not for males. At least two studies have reported accelerated neurocognitive development in females exposed to prenatal stress

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Table 3. Associations of prenatal stress and covariates with children's executive function

	Neuropsychological executive function				BRIEF-P global executive composite			
	Unadjusted model		Adjusted model		Unadjusted model		Adjusted model	
Predictor	b (SE)	*p*	b (SE)	*p*	b (SE)	*p*	b (SE)	*p*
Intercept	−.18 (.09)	**.045**	−1.59 (.65)	**.02**	75.79 (1.53)	**<.001**	74.14 (11.41)	**<.001**
Prenatal stress	−.26 (.08)	**.001**	−.27 (.08)	**.001**	2.60 (1.89)	.17	2.16 (2.05)	.30
Child sex	.32 (.11)	**.01**	.31 (.11)	**.01**	3.30 (2.06)	.12	3.05 (2.08)	.14
Prenatal stress × child sex					−7.03 (2.52)	**.01**	−6.39 (2.58)	**.01**
Non-Hispanic White			.08 (.16)	.64			1.18 (2.82)	.68
Hispanic			.03 (.31)	.94			−.99 (6.18)	.87
Other			.75 (.52)	.15			11.72 (9.01)	.20
More than one race or ethnicity			−.16 (.28)	.58			2.09 (4.87)	.67
Household SES			−.09 (.08)	.29			.59 (1.42)	.68
Maternal executive function			.02 (.00)	**<.001**			.02 (.07)	.86
Substance use during pregnancy			.06 (.14)	.70			1.92 (2.61)	.46
Marital status			.02 (.14)	.80			−.1.01 (2.54)	.69
Maternal depression and anxiety (5 years)			−.00 (.01)	.96			−.02 (.18)	.92
r^2	11%		23%		8%		9%	

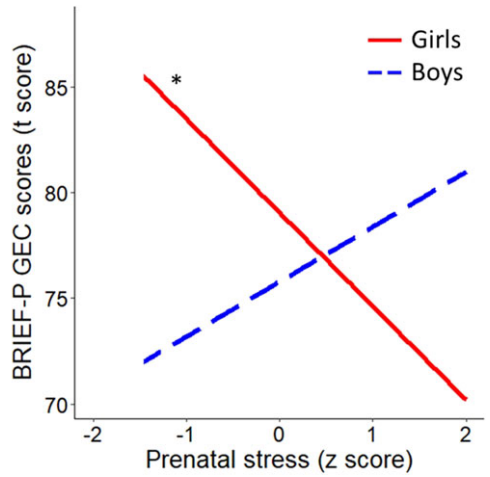

Figure 1. Association between prenatal stress and Behavior Rating Inventory of Executive Function - Preschool Global Executive Composite scores by child sex. *$p < .05$.

relative to males in infancy (Glynn & Sandman, 2012; Sandman et al., 2012). A positive association between prenatal stress exposure and EF may suggest that for females, prenatal stress is associated with accelerated development in the behavioral domains of EF assessed by the BRIEF-P, such as emotional control. Whether the positive association between prenatal stress and females' EF at age 5 years persists over time and is beneficial for their development long-term remains to be seen. Females are at increased risk for internalizing problems compared to males when exposed to stress prenatally (Sandman et al., 2013). Stress-induced early maturation of the brain regions involved in EF and emotion regulation, such as amygdala and prefrontal cortex, are related to internalizing problems in adolescence (van Tieghem & Tottenham, 2018). Thus, the positive association between prenatal stress and EF for females may be maladaptive long-term. Importantly, it is not possible to rule out the role of reporter bias in the BRIEF-P GEC findings, particularly as child sex did not moderate the effect of prenatal stress on the neuropsychological EF composite. For example, during the preschool period, parents expect females to have better self-regulation abilities and to be able to use more complex self-regulation strategies than males (Davis, 1995). Further research replicating these findings, examining sex differences in the impact of prenatal stress on the BRIEF-P GEC at different developmental stages, and linking these findings to psychopathology and psychosocial adjustment long-term are necessary to aid in the interpretation of the present results.

Contrary to our hypothesis, child sex did not moderate the effect of prenatal stress on neuropsychological EF. This result was surprising because previous research found increased male vulnerability to the impact of prenatal stress on child EF, as indexed by neuropsychological measures (Babineau et al., 2022; Plamondon et al., 2015; van den Bergh et al., 2006). Differing results across studies may suggest that males are more vulnerable to the negative effect of prenatal stress on EF in the context of more extreme risk or that sex differences depend on the timing of prenatal stress exposure. Babineau et al. (2022) found that males only had lower EF scores than females when mothers had clinically significant levels of depression during pregnancy. Similarly, Plamondon et al. (2015) reported increased male vulnerability but only in the presence of compounded risk factors. Sex differences may have emerged in the present study if the sample had been stratified by clinically relevant cutoffs on the prenatal stress measures or by the number of risk factors for lower EF in the home environment. In addition, in a study of adolescents, maternal anxiety between 12 and 22 weeks' gestational age was most strongly associated with lower sustained attention and impulsivity in males compared to females (van den Bergh et al., 2006). Between 8- and 24-weeks' gestational age, neuron proliferation, migration, and differentiation occur in brain regions connected to the prefrontal cortex, including the amygdala and anterior cingulate cortex

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(Nowakowski & Hayes, 2002). The limited number of mothers with prenatal stress data at all three trimesters meant we were not able to explore timing effects.

Key strengths of this study include a robust approach to the measurement of prenatal stress and controlling for maternal EF in the analyses. This study assessed five components of subjective stress in a cohort of predominantly low SES, Black families. Most previous prenatal stress research has focused on depression, anxiety, and perceived stress (i.e., the PSS). A more comprehensive assessment of subjective stress, including subjective stress experiences that are more likely to affect Asian, Indigenous, Hispanic, and Black women, such as everyday experiences of discrimination (Gong et al., 2017), is necessary to advance our understanding of how prenatal stress is related to children's neurocognitive outcomes and increase the generalizability of research findings. In addition, including maternal EF as a covariate in the analyses reduced the effect of a passive gene × environment correlation between prenatal stress and children's EF on outcomes.

This study also had several limitations. First, the sample size was relatively small. This limited our ability to run separate analyses for males and females and stratified by maternal self-reported race and ethnicity. Second, prenatal stressors can be categorized as objective, subjective, and physiological. Objective, subjective, and physiological stressors are differentially related to child outcomes, with physiological stress measures having the largest impact on children (King et al., 2012). While this study was rigorous in its approach to the measurement of subjective stress, more work examining the effects of maternal objective and physiological stress during pregnancy on children's EF is necessary for a more nuanced understanding of the impact of prenatal stress on children's EF.

The aim of this study was to better understand the impact of prenatal stress on children's EF in early childhood in low SES families. For both females and males, higher prenatal stress was associated with lower EF as reflected by neuropsychological task performance. For females only, higher prenatal stress was associated with lower BRIEF-P GEC scores, indicating better EF. Robust evidence links higher prenatal stress to psychopathology in children (Monk et al., 2019). EF deficits in early childhood are theorized to be a transdiagnostic risk factor for mental health problems across the lifespan (Zelazo, 2020). Findings from the present study suggest EF difficulties may be one pathway linking prenatal stress to psychopathology. Identifying the mechanisms driving the association between prenatal stress and child EF and examining pathways to psychopathology via EF in the context of high prenatal stress are likely to aid in the development of interventions aimed at improving EF and decreasing children's risk for mental health problems in the context of early adversity.

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