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RESEARCH ARTICLE

Prefrontal activation in preschool children is associated with maternal adversity and child temperament: A preliminary fNIRS study of inhibitory control

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Abstract

Exposure to adversity is a well-documented risk factor for cognitive, behavioral, and mental health problems. In fact, the consequences of adversity may be intergenerational. A growing body of research suggests that maternal exposures to adversity, including those prior to childbirth, are associated with offspring biobehavioral development. In a sample of 36 mothers and their preschool-age children (mean child age = 4.21 ± 0.92 years), we used functional near-infrared spectroscopy to replicate and extend this work to include brain activation during inhibitory control in young children. We found that measures of maternal exposure to adversity, including cumulative, childhood, and preconception exposures, were significantly and positively associated with activation in the right frontopolar prefrontal cortex (PFC) and in the left temporal and parietal clusters during inhibitory control. In addition, and consistent with previous findings, children's increased negative affect and decreased effortful control were associated with increased right PFC activation during inhibitory control. These findings provide preliminary evidence that maternal and dispositional risk factors are linked to alterations in PFC functioning during the preschool years. Children of mothers with a history of exposure to adversity, as well as children who are less temperamentally regulated, may require increased neural resources to meet the cognitive demands of inhibitory control.

KEYWORDS
adversity, executive function, functional near-infrared spectroscopy (fNIRS), inhibitory control, preschool, temperament

1 | INTRODUCTION

Adverse experiences, such as family dysfunction, poverty, neglect, and abuse, are powerful predictors of health and well-being across the lifespan (LeMoult et al., 2020; Nelson et al., 2020; Shonkoff et al., 2012). Many of these associations are due, in part, to the adverse effects of adversity on brain development and cognitive function (Gee, 2021; McLaughlin et al., 2019; Miller, Chahal, et al., 2022). The majority of this research has focused on links between children's exposure to adversity and their own neurodevelopmental outcomes (e.g., Chahal et al., 2022; Demir-Lira et al., 2016; Miller et al., 2020; Sheridan et al., 2017). For example, altered prefrontal cortex (PFC) development and difficulties in executive functioning are frequently documented outcomes in children exposed to adversity (Hanson et al., 2012; Pechtel & Pizzagalli, 2011; Suntheimer et al., 2022).

It is important to recognize, however, that the consequences of adversity may not be limited to those who are directly exposed, but may also extend across generations (Bowers & Yehuda, 2016). Indeed, recent work suggests that mothers' exposure to adversity prior to the birth of their child can alter their offsprings' development (Bouvette-Turcot et al., 2020; Glackin et al., 2021; Gray et al., 2017; Roubinov et al., 2021). Maternal history of adversity may influence child development by affecting mother–child interactions and the quality of their relationships (Roubinov et al., 2021). For example, parents who experienced maltreatment in childhood are at increased risk for maltreating their own children (Madigan et al., 2019). Furthermore, cumulative life adversity may increase risk for mental health problems and psychological stress during pregnancy (Foss et al., 2022), which itself may affect the intrauterine environment and fetal development in ways that alter postnatal biobehavioral development (Bowers & Yehuda, 2016; Entringer et al., 2015). Animal and human research suggest that prenatal stress influences epigenetic processes, affecting the programming and ultimately the functioning of biological systems that are involved in postnatal health and disease (Cao-Lei et al., 2020). Taken together, mothers' cumulative adversity exposure and adversity exposure during different life stages, such as childhood and pregnancy, could have long-lasting consequences for the biobehavioral development of their offspring. In support of this perspective, researchers have reported that maternal history of adversity exposure is associated with individual differences in children's parasympathetic nervous system functioning (Glackin et al., 2021), behavioral problems (van de Ven et al., 2020), temperament (Bouvette-Turcot et al., 2020), and morphology (Moog et al., 2018) and functional connectivity (Hendrix et al., 2021) in the brain. It is not yet clear, however, whether young children's PFC functioning (i.e., activation) is altered following maternal or child adversity. Further, we do not currently know if there are windows during which maternal and child exposure to adversity has particularly enduring effects on children's PFC functioning.

Early childhood PFC functioning is of interest for a number of reasons. The PFC plays a central role in executive functioning, including working memory (temporary storage and manipulation of information to complete tasks), cognitive flexibility (rapidly shifting attention between competing demands), and inhibitory control (inhibiting a dominant response while remembering and following a different rule) (Diamond, 2013; Moriguchi & Hiraki, 2013). These processes support behaviors such as turn-taking, delaying gratification, and following directions that are foundational for success and well-being during preschool and beyond (Moffitt et al., 2011; Schoemaker et al., 2013). Recently, researchers have used functional near-infrared spectroscopy (fNIRS), which measures cortical changes in oxygenation, to assess PFC functioning in young children. fNIRS is more robust to motion-related artifact than other neuroimaging methods (Aslin et al., 2015), making it particularly useful for assessing executive function-related brain activity in young children (Moriguchi & Hiraki, 2013).

A growing body of fNIRS studies suggests that temperamental dimensions of reduced effortful control and increased proneness to negative affect are related to increased PFC activation during the performance of executive functioning tasks (Fishburn et al., 2019; Li et al., 2017; Quiñones-Camacho et al., 2019). One interpretation of these findings is that less temperamentally regulated children require more neural resources to deal with the cognitive demands of performing executive functioning tasks. In this context, increased PFC oxygenation, as assessed by fNIRS, has been found to track with increased cognitive load (Fishburn et al., 2014; Mandrick et al., 2016). Thus, greater PFC oxygenation while engaging executive functions could be a neural compensatory mechanism in children with reduced effortful control and increased negative affect. In addition to the PFC, children have also been found to recruit temporal and parietal regions when engaging executive functions (Buss et al., 2014; Mehnert et al., 2013). It is not yet clear whether risk factors other than child temperament, such as child and maternal adversities, are associated with early executive function-related brain activity in the PFC and temporal and parietal regions.

In this study, we examined associations of maternal adversity, child adversity, and child temperament with children's neural response to an inhibitory control task that has been used in prior research (Fishburn et al., 2019). We expected to replicate findings reported by Fishburn et al. (2019) linking increased temperamental negative affect to increased PFC reactivity to inhibitory control. Further, we hypothesized that maternal and child adversity will be linked to patterns of neural activation that are associated with less effortful control and increased negative affect. Thus, we hypothesized that maternal and child exposure to adversity will be positively associated with PFC activation during inhibitory control. Lastly, we explored whether maternal adversity experienced during different life stages is associated differentially with children's neural activation, whether adversity and temperament are associated with neural activation in temporal and parietal brain regions, and whether maternal and child adversity differ in the relative strength of their associations with neural activation.

2 | METHODS

2.1 | Participants and procedure

Participants were drawn from a larger sample of pregnant women who were recruited originally by the March of Dimes Prematurity Research Center at Stanford University (Aghaeepour et al., 2017; Becker et al., 2021) for a study focused on using biopsychosocial information to predict birth outcomes. Women in the original study were ≥18 years of age and in their first trimester of pregnancy. The current study included women who agreed to be contacted for a follow-up assessment and their (now) preschool-age children. Inclusion criteria for the follow-up assessment were that women had given birth, that their children were 3–5 years of age, and that mothers were fluent in English. Mother–child dyads were excluded from the follow-up assessment if the child was born premature, if the mother or child had serious cognitive or physical challenges that might interfere with their ability to understand or complete procedures, or if the child had experienced traumatic brain injury. At the follow-up assessment, mothers completed a battery of questionnaires, including measures of exposure to adversity and child

temperament. In a separate testing room, children completed a battery of computerized executive function tasks, while their brain activity was monitored using fNIRS. Of the 48 mother–child dyads who participated in the follow-up assessment, 12 children were missing fNIRS data due to their refusal to wear the fNIRS cap, absence of useable data, or technical issues. Thus, the current analyses included 36 mothers and their preschool-age children (42% female; mean child age = 4.21 ± 0.92 years, range = 3.04–5.99; 58% White, 19% Multiracial, 14% Asian or Asian American, 3% Black or African American, 3% American Indian or Alaskan Native, 8% Hispanic/Latinx ethnicity). Families were predominantly middle- to upper-middle class in terms of socioeconomic status (SES) measured by parent education and family income (median parent education = graduate degree; median family income = $150K–$200K). This study was approved by the Institutional Review Board at Stanford University. All mothers were compensated for their time, and children received a small toy.

2.2 | Maternal and child adversity

Mothers reported on their own and their child's lifetime exposure to adversity using the Assessment of Parent and Child Adversity (APCA) (King et al., 2021). Briefly, as reported in King et al. (2021), the APCA is a computerized questionnaire measure that assesses maternal and child exposures to 40 and 10 types of adversity, respectively. For endorsed types of adversity, mothers completed a series of follow-up questions regarding the timing, severity, and duration of the adversity, as well as children's indirect exposure to parental adversities. Item responses were used to quantify maternal cumulative adversity (the total number of adversities that the mother reported experiencing in her lifetime), maternal childhood adversity (the total number of adversities that the mother reported experiencing <18 years of age), maternal preconception adversity (the total number of adversities that the mother reported experiencing ≥18 years of age but before they were pregnant with the focal child), maternal prenatal adversity (the total number of adversities that the mother reported experiencing during her pregnancy with the focal child), and child cumulative adversity (the sum of the number of maternal adversities that the child witnessed and the number of adversities for which the child was the primary individual exposed).

2.3 | Child temperament

Mothers completed the short-version of the Child Behavior Questionnaire (CBQ-SF) to assess child temperament (Putnam & Rothbart, 2006). The CBQ-SF consists of 94 items about a child's behavioral and emotional tendencies. These items were rated on a scale ranging from 1 ("extremely untrue of your child") to 7 ("extremely true of your child"). We averaged ratings for items within each CBQ-SF subscale to yield scores on 15 dimensions of child temperament: activity level, anger/frustration, approach, attentional focusing, discomfort, falling reactivity/soothability, fear, high-intensity pleasure, impulsivity, inhibitory control, low intensity pleasure, perceptual sensitivity, sadness, shyness, and smiling and laughter. These dimensions have been shown to load onto latent factors of negative affectivity, effortful control, and surgency (Rothbart et al., 2001). We focused our analyses on negative affectivity (anger/frustration, discomfort, fear, sadness, and soothability reverse scored) and effortful control (attentional focusing, inhibitory control, low-intensity pleasure, and perceptual sensitivity), and computed scores for these higher order dimensions of temperament using guidelines outlined by Rothbart and colleagues (Putnam & Rothbart, 2006; Rothbart et al., 2001).

2.4 | Inhibitory control task

fNIRS data were collected while children performed a validated, computerized Go/No-Go task, administered on a touch screen monitor, to assess neural activation during inhibitory control (Fishburn et al., 2019). As described in Fishburn et al., participants were told a story about a group of children who were playing outside when it starts to rain. The children wanted to keep playing, so the participant was asked to help to make the rain go away and the sun to come back. The task included three "inhibition" blocks consisting of a series of cartoon illustrations of sunshine ("go" stimulus), each presented for 1 s with a 500 ms interstimulus interval. These stimulus images were interspersed with occasional cartoon illustrations of rainy clouds ("no-go" stimulus). Children were instructed to touch the screen when they saw a picture of sunshine, but to not touch the screen when they saw a picture of a rainy cloud. The task also included three "control" blocks that presented a series of images of umbrellas (1 s duration, 500 ms interstimulus interval). Children were informed that they would only see these stimuli during these blocks and were instructed to touch the screen every time they saw an umbrella. Inhibition and control blocks were presented intermittently, always starting with an inhibition block. Each block consisted of 20 trials. Within the inhibition blocks, go and no-go stimuli were presented for 60% and 40% of trials, respectively. Blocks were separated by rest periods of 12–18 s. Each child completed a practice version of the task before participating in the test version.

2.5 | fNIRS data acquisition, preprocessing, and task activation

We measured hemodynamic activity in children using the NIRScout system (NIRx, Germany). Children wore a cap fitted with 16 sources and 16 detectors placed bilaterally over the PFC and temporal and parietal regions according to the international 10–20 EEG placement system. This cap montage yielded 48 channels of fNIRS data acquisition (see Figure 1). The fNIRS data were collected at a sampling frequency of 7.8 Hz.

The raw fNIRS data were preprocessed using the Homer2 package in MATLAB (https://www.nitrc.org/projects/homer2) and customized MATLAB-based scripts. We implemented a data processing stream

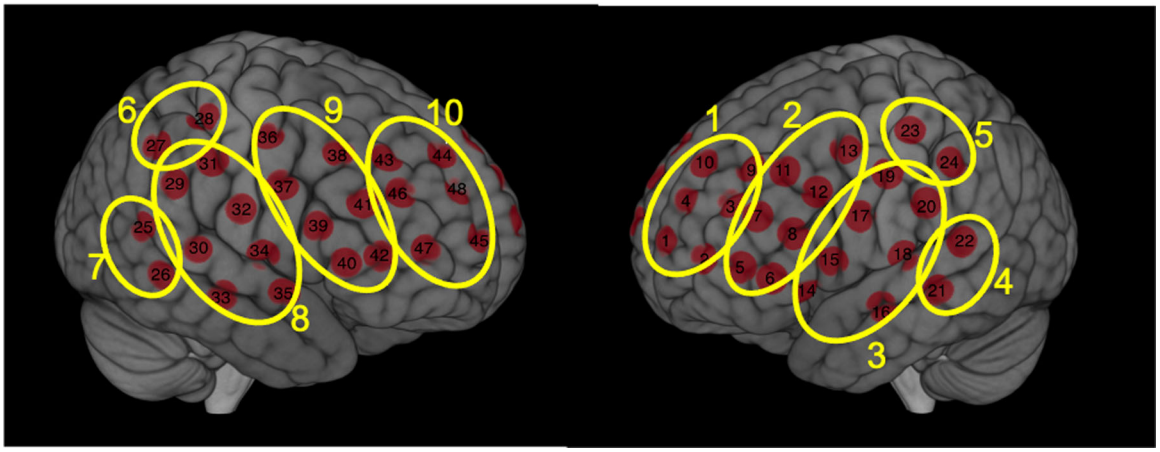

FIGURE 1 Functional near-infrared spectroscopy (fNIRS) channels and localization clusters. *Note*: fNIRS channels ($n = 48$) and localization clusters ($n = 10$). Red circles indicate channel locations visualized as the midpoint between each source and detector pair. Yellow circles are clusters based on source and include the following regions: 1 = left frontopolar prefrontal cortex (PFC); 2 = left lateral PFC; 3 = left superior temporal gyrus/sulcus and inferior parietal lobule; 4 = left posterior superior/middle temporal gyrus/sulcus; 5 = left superior parietal lobule; 6 = right superior parietal lobule; 7 = right posterior superior/middle temporal gyrus/sulcus; 8 = right superior temporal gyrus/sulcus; 9 = right lateral PFC; 10 = right frontopolar PFC

that incorporated recommendations outlined by Di Lorenzo et al. (2019). Specifically, raw fNIRS data were converted to optical density. Motion artifacts were first identified in the optical density data using the hmrMotionArtifactByChannel function in Homer2. The amplitude and standard deviation thresholds for identifying artifacts were set at 0.4 and 15, respectively (Di Lorenzo et al., 2019). Motion artifacts were corrected with spline-interpolation (Scholkmann et al., 2010) using the hmrMotionCorrectSpline function in Homer2. The interpolation parameter p was set to .99 (Cooper et al., 2012; Scholkmann et al., 2010). Remaining motion artifacts were identified and corrected with wavelet analysis and filtering (Molavi & Dumont, 2012) using the hmrMotionCorrectWavelet function in Homer2. The wavelet IQR parameter was set to 1.5 (Jahani et al., 2018). The motion-corrected optical density data were band-pass filtered with cutoff frequencies of 0.01 and 0.5 to remove physiological noise (Cui et al., 2011). The motion-corrected, filtered optical density data were converted to oxygenated and deoxygenated hemoglobin concentrations using the modified Beer–Lambert Law, with a differential pathlength factor that was determined by child age and wavelength (Scholkmann & Wolf, 2013).

In addition to these preprocessing steps, we used three methods to evaluate whether channels would be included in subsequent analyses. First, all raw data were visually inspected in time series plots for excessive noise (Miller et al., 2019). Second, all motion-corrected data were visually inspected using wavelet transform plots; channels that did not show a clear heartbeat frequency band were identified as noisy and were excluded from analysis (Liu et al., 2015). Lastly, channels showing positive correlations between oxygenated and deoxygenated concentration changes above 0.50 were identified as noisy (Cui et al., 2010) and were also excluded.

Our analyses focused on oxygenated hemoglobin concentrations. Task activation was quantified using a general linear model, which estimated beta values for inhibition and control blocks within each channel. Inhibitory control-related activation was defined as the contrast, or difference, between the inhibition and control betas. Task activations in 48 channels were grouped into 10 clusters based on source localization. Clusters of channels are presented in Figure 1.

2.6 | Statistical analyses

We computed Pearson correlations to examine whether child age, sex, maternal adversity, child adversity, and child temperament were associated with children's neural activation during inhibitory control (inhibition minus control block betas). We conducted follow-up regression analyses to determine whether adversity and child temperament were associated with neural activation after covarying these variables. Lastly, we compared associations of maternal and child adversity with neural activation; specifically, we tested whether relevant zero-order correlation coefficients differed significantly from each other (Diedenhofen & Musch, 2015). We used pairwise deletion of missing data in order to maximize our sample size for each analysis.

3 | RESULTS

Table 1 presents descriptive statistics for maternal adversity, child adversity, and child temperament measures. Distributions of these variables are presented in Figure S1.

Child age and sex were not associated significantly with either neural activation (all $ps > .058$) or temperament (all $ps > .184$). Mothers who reported that their children were exposed to more adversity reported higher levels of maternal cumulative adversity ($r = 0.47$, $p = .004$, $n = 36$) and adversity during their own childhood ($r = 0.43$, $p = .008$, $n = 36$). The positive association between child adversity

TABLE 1 Descriptive statistics for maternal adversity, child adversity, and child temperament variables

	Mean or % (SD)	Range
Maternal cumulative adversity	9.53 (4.76)	1–24
Maternal childhood adversity	3.64 (3.02)	0–13
Maternal preconception adversity	2.69 (1.98)	0–7
Maternal prenatal adversity	1.03 (1.67)	0–8
Child adversity	1.64 (1.62)	0–6
Child negative affect	3.99 (0.62)	2.56–5.22
Child effortful control	5.33 (0.63)	3.67–6.55

and maternal prenatal adversity did not reach statistical significance (r = 0.29, p = .087, n = 36). Older children performed better than younger children during the inhibition blocks (r = 0.56, p = .001, n = 31), whereas other study variables were not significantly related to performance (all ps > .078). Pearson correlations among all study variables are available at https://osf.io/654hj/.

3.1 | Associations of adversity and temperament with neural activation

Activation in the right frontopolar PFC (localization cluster 10) was positively associated with negative affectivity (r = 0.45, p = .015, n = 29) and negatively associated with effortful control (r = −0.53, p = .003, n = 29). Activation in the right frontopolar PFC was also positively associated with maternal exposure to adversity, including cumulative adversity (r = 0.42, p = .024, n = 29), adversity during childhood (r = 0.49, p = .003, n = 29), and adversity during preconception adulthood (r = 0.38, p = .043, n = 29). Figure 2 shows both the statistically significant and the non-significant associations of right frontopolar PFC with measures of maternal adversity, child adversity, and child temperament. Maternal cumulative adversity and preconception adversity (both r = 0.37, p = .040, n = 31) were also positively associated with activation in the left superior temporal gyrus/sulcus and inferior parietal lobule (localization cluster 3). Preconception adversity was also associated with increased activation in the left superior parietal lobule (localization cluster 5; r = 0.44, p = .012, n = 31). Maternal adversity during pregnancy and child exposure to adversity were not associated significantly with neural activation (all ps > .162), nor were child temperament variables associated significantly with activation in regions outside of the right frontopolar PFC (all ps > .186).

Given that exposure to adversity and SES are correlated, and that prior studies have linked SES to executive function and prefrontal development (Moriguchi & Shinohara, 2019; Noble et al., 2005; Olson et al., 2021), we conducted sensitivity analyses in which we included SES as a covariate. Maternal education and family income were positively correlated (r = 0.38, p = .024); therefore, we standardized and averaged these variables to form a single measure of SES. Maternal cumulative adversity and exposure to adversity during childhood were modestly associated with lower SES, although these correlations were not statistically significant (r = −0.29, p = .082, and r = −0.32, p = .054, respectively). SES was not significantly associated with activation in brain regions that were linked with adversity measures (i.e., localization clusters 10 and 3; both p > .269). The significant associations of maternal cumulative adversity and exposure to adversity during childhood with right frontopolar PFC activation were still present when covarying SES (β = .39, p = .0497, and β = .49, p = .016). In contrast, the association between maternal preconception adversity and prefrontal activation was no longer statistically significant after covarying SES (β = .35, p = .071).

3.2 | Follow-up regression analyses covarying maternal adversity and child temperament

We conducted follow-up regression analyses to test whether the zero-order correlations between maternal exposures to adversity and right frontopolar PFC activation remained significant after covarying for child temperament (see Table 2). The positive associations of maternal exposure to cumulative adversity and to childhood adversity with right frontopolar PFC activation were still present in models controlling for child temperament. The negative association of child effortful control with right frontopolar PFC activation was also still significant in the regression models. In contrast, associations with maternal exposure to preconception adversity and with child negative affect were no longer statistically significant.

3.3 | Comparing maternal and child adversity associations with right frontopolar PFC activation

As described above, child adversity was positively (but weakly and non-significantly) associated with right frontopolar PFC activation. Conversely, the positive associations between most maternal adversity measures and right frontopolar PFC were moderate in strength and significant. We conducted analyses to statistically compare these Pearson correlations (Pearson & Filon, 1898; Zou, 2007). The correlation of right frontopolar PFC activation with maternal cumulative adversity did not differ significantly from the correlation with child adversity (r difference = 0.25, 95% CI [−0.11, 0.59], Pearson and Filon's z = 1.44, p = .149). Although maternal adversity experienced during childhood was the measure of adversity that was correlated most strongly with right frontopolar PFC activation, this association was also not statistically different from that between right frontopolar PFC activation and child adversity (r difference = 0.32, 95% CI [−0.05, 0.67], Pearson and Filon's z = 1.80, p = .071).

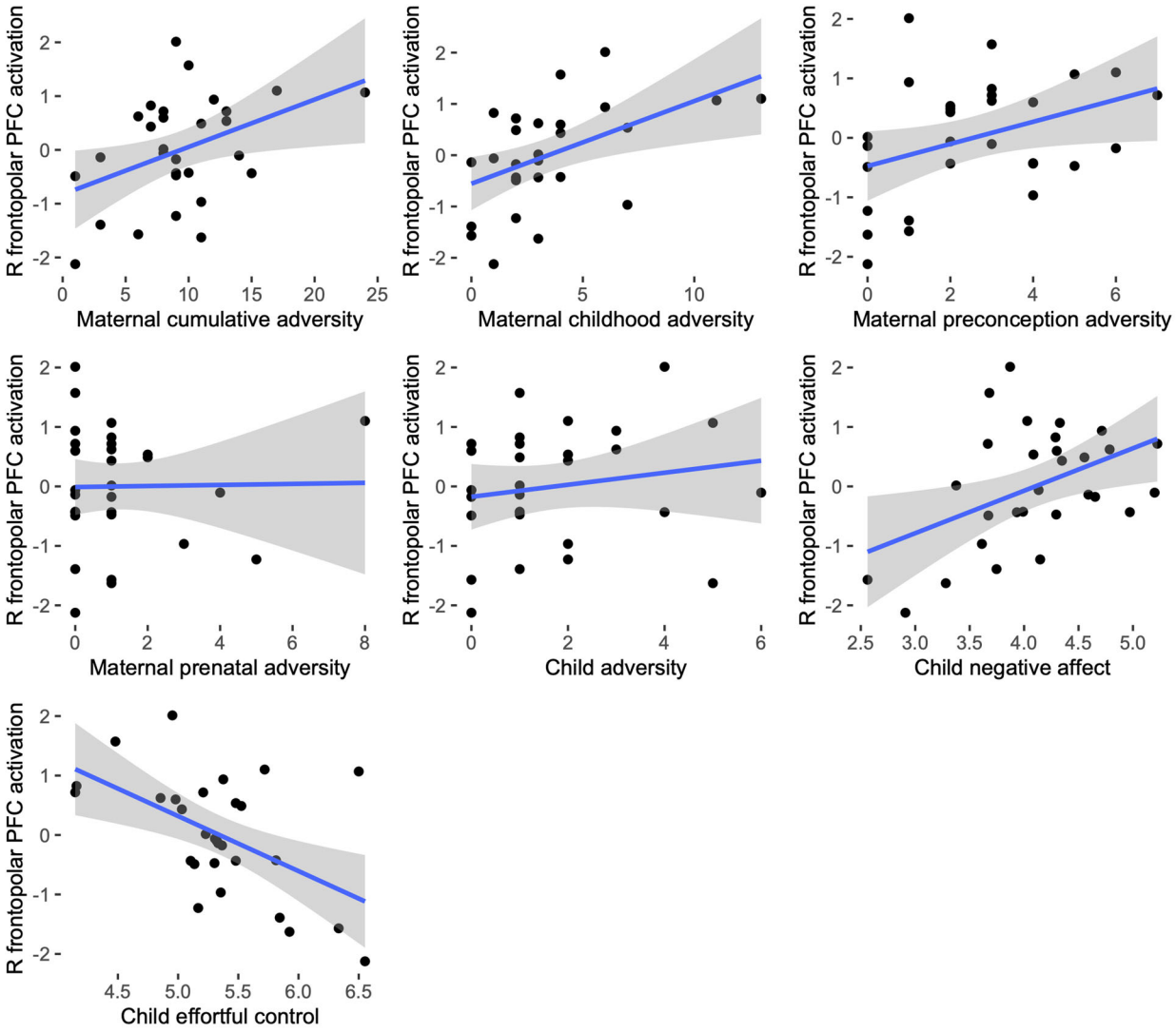

FIGURE 2 Associations of right frontopolar PFC activation with maternal adversity, child adversity, and child temperament. Abbreviations: R, right; PFC, prefrontal cortex

TABLE 2 Regression models for associations maternal adversity with right frontopoloar prefrontal cortex (PFC) activation controlling for child negative affect and effortful control

	Cumulative adversity as predictor				Childhood adversity as predictor				Preconception adversity as predictor			
	β	SE	95% CI	*p*	*β*	SE	95% CI	*p*	*β*	SE	95% CI	*p*
Child negative affect	.06	0.17	−0.30, 0.42	.729	.13	0.14	−0.15, 0.41	.335	.18	0.19	−0.22, 0.57	.368
Child effortful control	−.54	0.16	−0.88, −0.20	.003	−.57	0.14	−0.86, −0.29	<.001	−.40	0.18	−0.77, −0.03	.034
Maternal adversity	.45	0.15	0.13, 0.77	.008	.58	0.12	0.33, 0.84	<.001	.21	0.18	−0.16, 0.57	.255

4 | DISCUSSION

Children's exposure to adversity is a well-documented risk factor for impaired cognitive functioning, behavioral difficulties, and physical and mental health problems (McLaughlin et al., 2019; Nelson et al., 2020; Shonkoff et al., 2012). Growing evidence also suggests that mothers' exposure to adversity, including before the birth of their child, has long-term consequences for offspring biobehavioral development (Bowers & Yehuda, 2016; Roubinov et al., 2021). In this context, children's PFC functioning may be an important outcome; the PFC is central to executive functions that support positive development and well-being, and is sensitive to the effects of adversity (Pechtel & Pizzagalli, 2011).

In a sample of preschool-age children, we used fNIRS to investigate whether maternal and child adversities were associated with PFC reactivity to an inhibitory control task. We found that measures of maternal adversity, but not of child adversity, were significantly positively associated with right frontopolar PFC activation during the inhibitory control task. These findings provide preliminary evidence that maternal adversity is linked to children requiring greater prefrontal recruitment to meet the cognitive demands of executive function. Consistent with prior studies (Fishburn et al., 2019; Li et al., 2017; Quiñones-Camacho et al., 2019), we also found that children's reduced effortful control and increased negative affect were associated with greater PFC activation. Overall, our results replicate and extend findings of prior fNIRS studies of individual differences in children's PFC activation during executive function.

The present study also extends previous research linking maternal life adversity with child biobehavioral outcomes to children's PFC functioning. We found that maternal life adversity, particularly during childhood, was associated with PFC functioning independent of child temperament. Interestingly, the right frontopolar PFC (localization cluster 10) was the region that was linked most consistently with both adversity and child temperament measures. The frontopolar PFC has been found to play a role in multiple executive function processes (Wager et al., 2004), and may be particularly important for monitoring and switching between goals (Mansouri et al., 2017). In the context of our inhibitory control task, one interpretation of our findings is that withholding a dominant response while remembering and following a different rule is particularly taxing for children of mothers with a history of adversity and in children with less effortful control and more negative affect. We also found that maternal cumulative adversity and preconception adversity were associated with increased activation in left temporal and parietal regions. Some researchers have suggested that these regions are involved in maintaining task relevant information during inhibitory control (Durston et al., 2002). Taken together, these preliminary findings raise the possibility that maternal adversity experienced during different periods of life is related to children's functioning in distinct brain regions; maternal adversity during childhood may map onto offspring PFC development, whereas maternal adversity during adulthood (preconception) may have stronger effects on children's temporal and parietal functioning. Clearly, this regional specificity interpretation is speculative and requires further investigation.

The specific pathways by which maternal cumulative, childhood, and preconception adversity are related to children's inhibitory control-related PFC activity are still unclear. Several pathways have been proposed to explain how maternal adversity prior to birth could influence offspring biobehavioral developmental, some of which include prenatal adversity effects via fetal programming (Bowers & Yehuda, 2016; Entringer et al., 2015; Roubinov et al., 2021). Although we did not find a relation between prenatal adversity and child neural activation, prior research has found that mothers' adverse childhood experiences are associated with psychosocial stress during pregnancy (Roubinov et al., 2021). Thus, maternal childhood and preconception adversity effects may be mediated in part by prenatal processes. Alternatively, exposure to adversity may undermine caregiving behaviors that have been found to support children's executive function and neurodevelopment (Kahle et al., 2017; Swingler et al., 2018).

Contrary to our hypotheses, the positive association between child adversity and PFC activation was not statistically significant. There are several potential explanations for this finding. We were likely underpowered to observe a significant effect and may have required greater variability or a wider range of child adversity scores. Regarding this latter point, there is evidence that mothers tend to underreport their children's exposure to adversity (Fisher et al., 2011). To the extent that underreporting child adversity leads to a restricted range of scores, the correlation with neural activation would be reduced (Bland & Altman, 2011). It is also important to note that the observed effects of child adversity and maternal adversity were not statistically different from each other in our study. Thus, taken together, the lack of a statistically significant relation in our study may not be strong evidence against a possible link between child adversity and preschool age brain functioning.

Limitations of this study include the sample size and sample composition, limited variability for some adversity measures, and retrospective reporting of adversity. First, as we noted above, we may have needed a larger sample to detect statistically significant relations between some of the adversity measures and neural activation. Greater statistical power may also be required to adequately compare the associations of maternal adversity and child adversity with neural activation. As a related point, our sample was, on average, socioeconomically advantaged and not representative of the U.S. population. Second, some of our adversity measures elicited a wider range and greater variability of scores than others (see Supporting Information). Scores on maternal prenatal adversity had the least variability, which may have contributed to this measure showing the weakest association with right frontopolar PFC activation. It should also be noted, given the preliminary nature of this study, we did not correct for multiple comparisons in our analyses. Studies with larger, more representative samples that capture more variability in adversity exposure are necessary to replicate our preliminary findings using more conservative statistical thresholds. Finally, we used retrospective report of adversity. Although this is one of the most frequently used methods for measuring life adversity (Glackin et al., 2021; Miller, Dennis, et al., 2022; van de Ven et al., 2020), retrospective and prospective reports have been shown to have low agreement and may be associated with health outcomes via different pathways (Baldwin et al., 2019).

These preliminary findings support the perspective that adversity has intergenerational effects on biobehavioral development. The current study provides novel evidence that these effects extend to PFC functioning related to inhibitory control in early childhood. Our findings also replicate recent work suggesting that less effortful control and more negative affect are associated with increased PFC activation during executive function (Fishburn et al., 2019; Li et al., 2017; Quiñones-Camacho et al., 2019). Taken together, the current study suggests that maternal and dispositional risk factors are linked to PFC functioning during the preschool years. Children of mothers with a history of exposure to adversity, as well as children with

temperamental characteristics such as reduced effortful control skills, may require increased metabolic resources in the PFC to meet the cognitive demands of inhibitory control. Future work should replicate our findings, examine more explicitly whether there are windows of increased sensitivity to the effects of maternal and child adversity exposure on early PFC functioning, and elucidate the mechanisms and developmental implications of these associations.

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CONFLICTS OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

Data will be made available upon reasonable request.

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