DISSERTATION

# FAMILY STRESS, HAIR CORTISOL, AND ERROR-RELATED NEGATIVITY IN CHILDREN:

# POSITIVE PARENTING AS A PROTECTIVE FACTOR

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# ABSTRACT


## FAMILY STRESS, HAIR CORTISOL, AND ERROR-RELATED NEGATIVITY IN CHILDREN: POSITIVE PARENTING AS A PROTECTIVE FACTOR

Socioeconomic disadvantage has been significantly associated with executive function difficulties in children. However, the neural underpinnings of this relationship are not yet fully understood. Socioeconomic context may influence the error-related negativity (ERN), an event-related potential reflecting neural processes underlying error monitoring. Family stress may be a key proximal mechanism through which socioeconomic disadvantage influences error-related neural activity. The primary goal of this study was to test preregistered hypotheses about associations among family stressor exposure (material hardship, stressful life events, parental stress), hair cortisol concentration (HCC; a measure of cortisol output across months), and ERN amplitude in children. A secondary goal was to examine whether positive parenting moderated associations between family stressor exposure and either HCC or ERN in children, as sensitive parenting is often protective against the impacts of socioeconomic disadvantage. Participants were typically developing 5- to 13-year-olds (57% male, $N = 108$). Results indicated that lower family income-to-needs ratio and parental education were associated with increased exposure to family stressors. Higher HCC was significantly associated with smaller ERN amplitude in children. Family stressor exposure was not significantly associated with either ERN amplitude or HCC in children. Positive parenting did not significantly moderate associations between family stressor exposure and HCC or ERN. This study is the first to show that HCC may be associated

with error-related neural activity in children. Altered regulation of the hypothalamic-pituitary-adrenocortical (HPA) axis may lead to blunted neural processing of errors in childhood.

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# INTRODUCTION

Growing up in a lower socioeconomic status (SES) environment has been frequently associated with reduced cognitive control (also termed executive function; EF) in children and adolescents (Boelema et al., 2014; Brieant et al., 2021; Lambert et al., 2017; Lawson et al., 2018; Merz et al., 2019; Spielberg et al., 2015). Researchers have started to shed light on the neural mechanisms underlying these associations (Hackman & Farah, 2009; Kamgang et al., 2023; Merz et al., 2024; Noble et al., 2015; Pietto et al., 2017; Ursache et al., 2016), but there is much more yet to be understood. Electroencephalography (EEG) is an advantageous tool that has been leveraged to reveal these neural mechanisms (Olson et al., 2021). EEG methodology has the strength of high temporal resolution, which allows for precise measurement of neural activity over time across stages of cognitive processing. EEG also provides a sensitive metric reflecting neural activity underlying cognitive processing, which may reveal differences even when differences in behavioral performance are not detected (Kishiyama et al., 2009).

Error monitoring, an important component of cognitive control, refers to the ability to identify one's mistakes (Ridderinkhof et al., 2004). This ability to monitor outcomes of actions and adapt behavior is necessary for effective goal-directed behavior (Falkenstein et al., 2000; Ridderinkhof et al., 2004). The neural basis of error monitoring is commonly assessed using EEG-based event-related potentials (ERPs). The error-related negativity (ERN) is an ERP component that can be seen about 100 milliseconds after an individual makes a mistake and appears as a negative deflection in the waveform (Falkenstein et al., 2000). A stronger negative ERN amplitude usually suggests more efficient neural processing of error monitoring (Meyer et al., 2019) and may be associated with better executive functioning (Grammer et al., 2018; Larson

& Clayson, 2011). The ERN is thought to reflect neural activation in the anterior cingulate cortex (ACC) (Bush et al., 2000). The associations between socioeconomic factors and the ERN in children and adolescents are not well understood.

SES refers to a family's access to economic and social resources and the social privileges that derive from those resources (Duncan & Magnuson, 2012). SES is often measured using parental educational attainment and/or family income, which tend to be strongly correlated with each other. These measures are considered to represent distinct resources that might impact cognitive development in different ways (Duncan & Magnuson, 2012). SES is a distal environmental factor thought to exert its effects on children's development through multiple proximal factors, such as family stress (Bronfenbrenner & Morris, 1998; Noble et al., 2012). Indeed, decades of research has established associations between socioeconomic disadvantage and increased exposure to family stressors, such as material hardship, crowding/noise, family conflict, neighborhood violence, and household chaos and unpredictability (Conger et al., 2000; Evans & Kim, 2013; Lichtin et al., 2021). Increased family stress exposure may partially explain why socioeconomic disadvantage is associated with differences in error monitoring in children. Based on animal models, family stress can alter physiological stress response system functioning, in turn influencing the development and function of neural circuits responsible for error monitoring (Blair, 2010; McEwen, 2007; Merz et al., 2024). Yet the role of family stress in the mechanisms through which socioeconomic factors may impact ERN amplitudes is not well understood. Therefore, one primary goal of this study is to examine how family stress related to socioeconomic disadvantage may influence error monitoring as measured by the ERN in children.

## Socioeconomic Factors and the ERN in Children and Adolescents

The relationship between socioeconomic factors and the ERN amplitude in children is not well understood. The few studies that have examined these associations have yielded inconsistent results (Brooker, 2018; Conejero et al., 2016; Perera-W.A. et al., 2021). One study found that low SES was associated with reduced ERN amplitudes in toddlers (16 to 18 months of age) (Conejero et al., 2016). Another study found that only preschoolers with a higher SES environment and responsive parents showed typical ERN development. Specifically, the expected increase in ERN amplitude between ages 3 and 4 years was observed only for children from higher SES backgrounds whose mothers self-reported high sensitive caregiving (Brooker, 2018). However, this study found no direct association between SES and ERN amplitude. Similarly, another study of 5- to 8-year-old children found no association between family income and ERN (Meyer & Klein, 2018).

## Socioeconomic Factors and Family Stressors in Children and Adolescents

Stress is a multifaceted construct that can be measured in terms of stressors individuals are exposed to (adverse experiences that elicit a stress response) or the functioning of physiological stress response systems. Here, I use the terminology in which 'stressors' refer to adverse experiences, and stress response systems refer to physiological systems in place to respond to stressors or threats (Hostinar et al., 2014). Socioeconomic disadvantage is associated with increased exposure to a range of family stressors, including frequent stressful life events, material hardship, and increased parental stress levels (Conger et al., 2000; Evans & Kim, 2013). These are the stressors that are the focus of this study. Children from lower SES environments are more likely to be exposed to greater cumulative negative life events (e.g. unstable home life, family conflict) such as those that are threatening and can lead to hyper-vigilance (Brooks-Gunn

et al., 1997; Glasscock et al., 2013; Kristenson et al., 2004; Lantz et al., 2005; Vogel et al., 2021). These increases in life stressors can impact children's cognitive skills and self-regulation (Blair et al., 2011).

Material hardship includes expense hardships, food insecurity, housing hardship, and medical hardship (Rodems & Shaefer, 2020). Experiencing material hardship can lead to negative long term outcomes in children (Goux & Maurin, 2005; Lave et al., 1998; Schenck-Fontaine et al., 2020; Whitaker et al., 2006), including decreased executive functioning (DeJoseph et al., 2021).

Socioeconomic disadvantage also often leads to increases in parental stress (Lefmann et al., 2017). For instance, parental stress is estimated to be four times higher in low SES parents than high SES parents (Martins et al., 2023). Research has supported the family stress model, which suggests that economic hardship increases pressure on parents and increases risk for child behavioral problems through exposure to parental distress (Conger & Conger, 2008; Masarik & Conger, 2017; Strack et al., 2026).For example, increased parental stress has been associated with altered long term outcomes in children, including decreased executive functioning (Lean et al., 2023). Thus, research is needed to understand the association between family stressors associated with socioeconomic disadvantage and the neural processing of error monitoring in children.

**Stress Physiology and the HPA Axis**

Allostasis is the activation of neural, neuroendocrine and neuroendocrine-immune mechanisms activated to respond to stressors. Dealing with a high level of stress puts strain on these systems, and the extent to which they are overtaxed or can no longer perform normally is

termed allostatic load (McEwen, 2007). Allostatic load over a long period of time can interfere with physical and mental health. At the physiological and neuroendocrine level, the hypothalamic-pituitary-adrenal (HPA) axis is one of the main stress response systems. Stressor or threat exposure activates the HPA axis. Specifically, stressors are detected and interpreted by frontolimbic circuitry (e.g., amygdala, medial prefrontal cortex [mPFC], ACC), which signals to activate the HPA axis stress response (Pruessner et al., 2010). Secretion of corticotropin-releasing hormone from the hypothalamus then prompts the pituitary gland to release adrenocorticotropic hormone, which in turn prompts the adrenal glands to release glucocorticoids (e.g., cortisol in humans). An increase in cortisol then triggers a negative feedback loop involving neural structures including the hypothalamus and hippocampus, which leads to reduced circulation of cortisol (Sapolsky et al., 2000). Acute HPA axis responses are important and necessary for an adaptive response to a stressor or threat. However, an overactive or underactive HPA axis can lead to nonoptimal adaptation and development. Stress exposure and HPA axis overactivation exert especially pronounced effects on the structure and function of ACC and mPFC circuitry responsible for error monitoring; these brain regions have abundant glucocorticoid receptors (Davis et al., 2002; Francis et al., 1999).

Although cortisol can be measured in saliva, serum, and urine, the measurement of cortisol in hair (hair cortisol concentration or HCC) reflects the accumulation of cortisol over months, which more closely reflects *chronic* stress compared to other methods (Meyer & Novak, 2012). HCC has proven to be a valid and reliable index of extended cortisol secretion (Grass et al., 2015; Lee et al., 2015; Short et al., 2016; Stalder et al., 2012).

**Socioeconomic Factors, Family Stressors, and HCC in Children and Adolescents**

Socioeconomic disadvantage has frequently been associated with elevated HCC in children and adolescents (Anand et al., 2020; Cantave et al., 2022, 2023; Gray et al., 2018; Kao et al., 2019; Merz et al., 2024; Tarullo et al., 2020; Vliegenthart et al., 2016). However, results have been inconsistent. Some studies have found that low parental education, but not low family income, is related to higher HCC (Merz et al., 2019; Tarullo et al., 2020; Ursache et al., 2017; Vaghri et al., 2013) while others have found the opposite pattern (Rippe et al., 2016; Simmons et al., 2019). Some studies find that both low family income and low parental education are related to higher HCC (White et al., 2017). Others have found lower HCC in children from low SES environments (Ertekin et al., 2021), while others have found no association between socioeconomic factors and HCC in children (Bryson et al., 2021; Malanchini et al., 2021; Wagner et al., 2019). Together, these studies imply that relying solely on distal SES measures may provide an incomplete picture of these associations. Research is needed to shed light on the role of proximal environmental factors such as family stressor exposure.

Greater exposure to stressors has been associated with higher HCC in children (Babarro et al., 2023; Bates et al., 2017; Bhopal et al., 2019; Li et al., 2023; Ouellet-Morin et al., 2021; Shan et al., 2024; Simmons et al., 2016). For example, food insecurity and overall economic hardship have been linked with higher HCC in children (Ling et al., 2019; Santaularia et al., 2023; Tarullo et al., 2020). Children living in apartments and non-ownership housing (where there is likely to be greater noise and crowding and less stability) tend to have higher HCC (Bryson et al., 2019; Karlén et al., 2013). However, some studies have found no association between family stressor exposure and HCC in children (Michels et al., 2017; Milam et al., 2014; Shapero et al., 2019). These studies found no association between child self-reported negative

life experiences (Michels et al., 2017) or stressful life experiences (Milam et al., 2014; Shapero et al., 2019) and HCC in children. Some studies have found a positive association between parental stress and children's HCC (Babarro et al., 2023; Bates et al., 2017; Bhopal et al., 2019; Bilodeau-Houle et al., 2025; Kang et al., 2024; Li et al., 2023; Ouellet-Morin et al., 2021; Palmer et al., 2013; Shan et al., 2024; Simmons et al., 2016), while others have found no association (Isaac et al., 2023; Koenig et al., 2018; Liu et al., 2016; Olstad et al., 2016; Romero-Gonzalez et al., 2018). Together, these findings suggest that exposure to family stressors may impact HCC in children, but more research is needed to gain a complete picture of the associations among socioeconomic context, exposure to family stressors, and HCC in children.

**Cortisol Levels and the ERN in Children and Adolescents**

The developmental period spanning across middle childhood into early adolescence is a time of HPA axis reorganization (Doom & Gunnar, 2013; M. R. Gunnar & Vazquez, 2015). Heightened reactivity to stressors and increased neural sensitivity to the effects of cortisol have been observed during this time (Dahl & Gunnar, 2009; M. Gunnar & Quevedo, 2007; M. R. Gunnar et al., 2009; M. R. Gunnar & Donzella, 2002; Lupien et al., 2009). Altered HPA axis function during this developmental period could have pronounced effects on the neural development of error monitoring (Blair et al., 2005; Lipska et al., 2002). Thus, there is a need to investigate the associations between HPA axis functioning and ERN amplitudes during middle childhood through early adolescence.

Some MRI research has found associations between cortisol levels and brain structure and function, including structure and function of the ACC (Merz et al., 2024; Thomason et al., 2011), which is found to be the source of the ERN (Carter et al., 1998). Yet, only a few studies have examined associations between cortisol levels and the ERN, and these have all been studies

of acute stress using salivary cortisol methods in adults. Specifically, multiple studies have found that a smaller ERN is associated with increased salivary cortisol levels in adults (Compton et al., 2013; Hu et al., 2019; Tops & Boksem, 2011). These studies also varied considerably in the timing of cortisol collection relative to the stressor and the ERN task, complicating direct comparisons of findings across studies. One study found that individuals who had high cortisol responses to a stressful task also had a significantly smaller ERN after a stressful task (Hu et al., 2019). Another study found that smaller ERNs predicted more cortisol increase during a stressful task (Compton et al., 2013). In another study, higher basal salivary cortisol before a flanker task was associated with a smaller ERN amplitude during the task (Tops & Boksem, 2011). Taken together, these studies suggest that acute salivary cortisol may be related to ERN amplitudes in adults. However, to my knowledge, no study has examined the association between HCC and the ERN in children.

In sum, although socioeconomic disadvantage has been consistently associated with lower levels of cognitive control at the behavioral level (Lawson et al., 2018; Merz et al., 2019), the proximal and neural mechanisms underlying these associations are not well understood. Theoretical and empirical evidence suggests that family stressor exposure and differences in neural processing underlying error monitoring may link socioeconomic disadvantage to disruptions in cognitive control in children. Socioeconomic disadvantage has long been associated with increased exposure to family stressors (Conger et al., 2000; Evans & Kim, 2013). Animal models have yielded strong evidence that stressor exposure alters HPA axis regulation, especially early in life, and influences the function of neural substrates, such as the ACC, associated with error monitoring (Shonkoff et al., 2009; Weinberg et al., 2016). Yet, there are no studies examining the associations between chronic cortisol output and the ERN in children.

Therefore, in this study, I examined the associations among socioeconomic factors, family stress, HCC, and the ERN in children. I also examined the possible mediating role of HCC in the association between SES-related family stress and the ERN in children.

**Positive Parenting as a Protective Factor**

Resilience refers to exhibiting positive outcomes (adapting to environmental demands) despite having been exposed to significant adversity (Masten & Reed, 2002). Many children exposed to socioeconomic disadvantage "beat the odds" and demonstrate resilience. Positive parenting has been proposed as a key protective factor explaining resilience in children (Merz et al., 2024; Newland, 2014). Positive parenting characterized by high levels of warmth and responsiveness has been found to bolster the development of cognitive control in children (Fay-Stammbach et al., 2014; Lugo-Gil & Tamis-LeMonda, 2008; Sroufe, 2000). Positive parenting is also a key form of social support found to promote adaptive HPA axis regulation in children (M. Gunnar & Quevedo, 2007; Hostinar et al., 2014). Therefore, it is important to consider the potentially protective role of positive parenting when examining the effects of socioeconomic disadvantage and family stress on HCC and ERN in children.

Multiple studies have found that positive parenting protects children against the negative impacts of poverty on physical (Brody et al., 2016) and mental health (Grant et al., 2000; Kim-Cohen et al., 2004; Labella et al., 2019; Loukas & Prelow, 2004; Smith et al., 2022) as well as cognitive development (Lee et al., 2019; Liu & Lachman, 2019). At the neural level, positive parenting has been found to protect against the effects of socioeconomic disadvantage on neural function associated with emotion regulation and cognitive control (Brody et al., 2019; Hyde et al., 2020; Whittle et al., 2017). For example, Brody and colleagues (2019) found reduced functional connectivity in the central-executive and emotion-regulation networks in individuals

who grew up in poverty with low parental support but not in those with high parental support. The role of positive parenting as a protective factor in regard to error monitoring as measured by the ERN is not understood.

Parenting quality has also been found to moderate the association between SES and HPA axis function, as measured by salivary cortisol (Blair et al., 2008; Blair et al., 2011; Blair et al., 2011; Johnson et al., 2018; Zalewski et al., 2012). However, to my knowledge, only one study has examined the associations between positive parenting and HCC in children from under-resourced backgrounds. In this study, positive parenting was associated with reduced HCC in children from disadvantaged backgrounds (Simmons et al., 2019). Thus, little is known regarding the protective effects of positive parenting on HCC. In sum, the moderating effects of positive parenting on associations of socioeconomic factors and family stressors with HCC and ERN amplitudes in children are not well understood.

**Current Study**

The goals of this study were to examine (1) the associations among socioeconomic factors, family stressor exposure, HCC, and ERN amplitudes in children and (2) positive parenting as a moderator of associations between family stressor exposure and HCC and ERN in children. Participants were typically-developing 5- to 13-year-olds from socioeconomically diverse families. Family income-to-needs ratio and parental education were examined separately as they may have distinct effects on children's environments and development (Duncan & Magnuson, 2012). Family stressor exposure was operationalized as a composite of stressful life events, material hardship, and parental perceived stress (Merz et al., 2019), with supplemental analyses focused on these stressors individually (Malanchini et al., 2021). EEG activity was recorded while children complete a speeded flanker task (Lin et al., 2020), and the ERN was

computed based on these data. The aims and hypotheses of this study were preregistered (Strack & Merz, 2024a, 2024b)

For my first study goal, I hypothesized that lower family income-to-needs ratio and parental education would be associated with greater family stressor exposure (see Figure 1). In addition, I expected that more family stressor exposure would be associated with a smaller ERN amplitude in children. I also hypothesized that HCC would mediate the association between family stressor exposure and ERN amplitude in children. Specifically, I expected that family stressor exposure would be associated with higher HCC, which in turn would be associated with reduced ERN amplitude in children (see Figure A1).

For my second study goal, I hypothesized that the associations of family stressor exposure with HCC and ERN would be moderated by positive parenting. I expected that children with more exposure to family stressors, but who experienced greater positive parenting, would exhibit HCC levels and ERN amplitudes comparable to those of children with low family stressor exposure (see Figure A2).

In this study, I aim to clarify how stress-related biological processes may influence children's capacity to learn from mistakes and adapt their behavior. Identifying these pathways is essential for moving beyond descriptive associations toward actionable targets for early intervention. Moreover, examining positive parenting as a potential protective factor helps inform resilience-based frameworks, highlighting opportunities to buffer stress-related risk and promote adaptive development in children growing up in disadvantaged environments.

# METHODS

## Participants

### *Recruitment*

Participants were recruited by posting flyers in the local community; conducting outreach with schools, childcare centers, and local organizations; contacting summer camps in northern Colorado neighborhoods; and posting flyers on community-centered websites. Screening was conducted over the phone to determine eligibility to participate in the study. Families were eligible based on multiple factors. They had to have a typically-developing child between 5 and 13 years of age who spoke English as a primary language. To attain a socioeconomically representative sample, families were enrolled at each of the following parental education levels: high school diploma or fewer years of education; some college; four-year college degree; and more than a four-year college degree. Parents were asked on the phone about their educational attainment and were considered for enrollment if it fell into a category for which the participation quota has not been reached (Merz et al., 2019; Merz et al., 2020). Families were ineligible to participate in the study if the child had ever been diagnosed with a neurodevelopmental disorder (e.g., autism spectrum disorder) or neurological disorder (e.g., epilepsy); was the product of a multiple birth (e.g., twin, triplet); or was born premature (36 gestational weeks or less). Families were also ineligible if the parent was not fluent in English or Spanish or was younger than 18 years of age.

*Sample characteristics*

The total sample size for this study was 108 children (57% male, 75% white, non-Hispanic/Latine). On average, parental education was 16.44 years (SD = 2.82), and family income-to-needs ratio was 4.18 (SD = 2.48). Income-to-needs ratios above 1 indicate income above the poverty line. Family income ranged from $500 to $300,000, and parental education ranged from 11 to 22 years (see Table 1 for descriptive statistics on sample characteristics).

*Sample sizes*

One hundred and six out of the current total of 108 children in the study have EEG data. Two children declined to wear the EEG cap. ERN data from 95 children were included in analyses as 11 children had too many errors on the flanker task to be included (> 38%). Ninety-seven children had hair long enough to take hair samples from. Of those children, 11 were excluded from analyses for having HCC values outside 1.5 × interquartile range (IQR), leaving 86 with HCC data usable in analyses. The parenting quality questionnaire was added to the study after a number of families had already participated (as detailed below), and 85 total parents completed it. Out of those 85, 51 parents completed the parenting quality questionnaire online (out of 74 who were emailed the questionnaire) and 34 completed the questionnaire during their in-person lab visit. There was relatively little missing data; missing data were handled using listwise deletion, such that participants were included in each analysis only if they had complete data for the variables involved in that specific model. Approximately 93 children were included in family stress–ERN analyses, 84 in family stress–HCC analyses, 75 in ERN–HCC analyses, 75 in ERN–family stress–parenting moderation models, and 66 in HCC–family stress–parenting moderation models.

## Procedures

Families deemed eligible who were interested in participating were scheduled for a 3-hour visit to our shared lab space on the CSU campus. During the visit, parents/guardians first provided written informed consent, and children ages 5-6 years provided verbal assent while children ages 7-13 years provided their written assent to participate in the study. The child was then accompanied into an electrically shielded, sound-attenuating booth and fitted with an EEG cap. Once the child had been fully fitted and the impedances had been reduced 20 or better, the child then underwent artifact training. They were told to perform a myriad of behaviors such as blinking, swallowing, and clenching their jaw, then they were shown the artifacts created by these behaviors. They were asked to then sit quietly and monitor their brainwaves, once they were able to see the decreased activity related to their stillness, they were told that they can help keep the brainwaves "quiet" for the researchers by staying as still and quiet as possible during the tasks. Impedances were monitored and an offset of 20 or better was maintained throughout the data collection process.

The child then completed a set of tasks, including the speeded flanker task, while EEG activity was being recorded. Following this EEG session and a break, the child was involved in other aspects of the study including hair sample collection. While the child was involved in these activities, the parent completed a set of questionnaires including surveys asking about the family's sociodemographic background and the child's past experiences and emotional and behavioral functioning. Parents and children received compensation for their participation. The parenting quality questionnaire was added to the study after a number of families had already participated. Parents/guardians who had already participated in the study were emailed and asked

to complete the parenting quality questionnaire online. These procedures were approved by the Colorado State University Institutional Review Board.

## Measures

### *Socioeconomic factors*

Parents reported on household income and the number of adults and children in the household, and family income-to-needs ratio was computed by dividing the household income by the federal poverty threshold for the family size. Income-to-needs ratios above 1 indicate income above the poverty line. To measure parental education, parents reported their exact number of years of education completed, and educational attainment was averaged across the child's parents (Dearing et al., 2001). As expected, family income-to-needs ratio and parental education were significantly correlated ($r = .67$, $p < .001$).

### *Stressful life events*

Parents completed the Life Experiences Survey (LES) (Sarason et al., 1978), a 47-item questionnaire focused on life events in the past year and the impact they had on the family. Each item is rated on a 7-point Likert scale ranging from extremely negative impact (-3) to extremely positive impact (+3). Item scores for events rated as having a negative impact were added together and then converted to a positive number for analyses. Larger numbers indicate more negative life experiences. The LES is considered reliable with a strong test-retest reliability (Sarason et al., 1978).

*Material hardship*

Parents also completed a material hardship questionnaire (Pilkauskas et al., 2012). Parents respond with yes (1) or no (0) to 11 questions regarding lack of resources due to lack of finances in the past year. Example items include "Did you not pay the full amount of rent or mortgage payments?", "Did you not pay the full amount of the gas or electricity bill?", and "Did you borrow money from friends or family to pay bills?" (Gershoff et al., 2007). Higher scores indicate more material hardship (Merz et al., 2019).

*Parental perceived stress*

Parents completed the Perceived Stress Scale (PSS), a 14-item self-report questionnaire focused on their level of stress (Cohen, 1988). Items include "How often have you found that you could not cope with all the things that you had to do?". Items are rated on a 5-point Likert scale ranging from never (0) to very often (4). Higher scores indicate more perceived stress. The Cronbach's alpha coefficient for the PSS has been reported as .78 ( Cohen, 1988). Studies have consistently reported that the PSS has good internal validity (Barbosa-Leiker et al., 2013; Golden-Kreutz et al., 2004) and adequate convergence with other measures (Mitchell et al., 2008; Roberti et al., 2006; S. M. Wu & Amtmann, 2013).

A family stressor exposure composite was created using the PSS, LES, and material hardship scores. These indicators were significantly correlated with one another: PSS and LES, $r = .48$, $p < .001$; PSS and material hardship, $r = .42$, $p < .001$; and LES and material hardship, $r = .43$, $p < .001$. Each variable was then standardized using z-scores. The standardized values were averaged to form a family stressor composite score, with higher values reflecting greater family stressor exposure (Mazziotta & Pareto, 2013). The main analyses were run using this composite, with

supplemental analyses focused on each of the stressors individually (Malanchini et al., 2021). When running analyses on individual stressors, negative life experiences and material hardship scores were log transformed to adjust for skew.

*Positive parenting*

Parents completed the Multidimensional Assessment of Parenting Scale (MAPS), a 34-item self-report questionnaire used to measure both the positive and negative dimensions of parenting (Parent & Forehand, 2017). Each item is rated using a 5-point scale ranging from *never* (1) to *always* (5). The MAPS yields a positive parenting total score, which is the sum of the scores on the proactive parenting (e.g., "I give reasons for my requests"), positive reinforcement (e.g., "If my child does their chores, I will recognize their behavior"), warmth (e.g., "I express affection"), and supportiveness (e.g., "I encourage my child to talk about their troubles") subscales. Higher scores indicate more positive parenting. The positive parenting score was used in analyses. The MAPS has been reliably used for parents of children ages 3 – 17 years (Parent & Forehand, 2017).

**Electrophysiological Recording**

EEG is recorded with 64 electrodes from the Active-Two BioSemi system (BioSemi, Inc., Amsterdam, the Netherlands) according to the 10-20 system. Two electrodes, the common mode sense (CMS) and the driven right leg (DRL), generated the reference voltage for the online EEG data collection. Signals collected from the left and right earlobes are collected to be averaged and used for the offline reference. Two other electrodes are placed above and below the participant's left eye to track vertical eye movements. An additional two electrodes are placed on the left and right temple to track horizontal eye movements. The data is sampled at 2048 Hz.

*Speeded arrow flanker task*

ERN is elicited using the speeded arrow flanker task (Eriksen & Eriksen, 1974; Lin et al., 2020) presented by E-prime software, version 3 (Psychology Software Tools, Pittsburgh, PA). Five horizontally aligned arrows are presented, with the center arrow being the target. Participants press a button with their right index finger if the middle arrow is facing right or press a button with their left index finger if the middle arrow is facing left. Participants completed a brief practice block of 12 trials before data collection began. There are 240 trials presented in two blocks, for a total of 480 trials. These two blocks of speeded arrow flanker trials are broken up with another ERP paradigm in between. Of these trials, 160 are congruent and 320 are incongruent (see Figure A3). On each trial, the stimulus is presented rapidly, with a duration of 250 ms and an initial interstimulus interval (ISI) of 1400 ms. The E-prime program reevaluated the error rate every 30 trials, allowing for the adjustment of the ISI by ±100 ms if the error rate was either greater than 30% or fewer than 10%, such that if the participant made too many errors, the presentation of stimuli slowed down with a longer ISI and likewise sped up if they made too few errors with a shorter ISI (Lin et al., 2020). The ISI parameters are set to not decrease below 800 ms, which allows sufficient time for the brain to process the stimulus before the subsequent stimulus is presented. For each trial, behavioral parameters of error rate and response time (RT) both on correct and incorrect trials are calculated.

*ERN waveform and component analysis*

The raw EEG data was preprocessed using Brain Vision Analyzer 2.0 software. Preprocessing includes referencing the data to the averaged bilateral earlobe sites and filtering with a 0.1-30 Hz bandpass filter. Data is then segmented into response-locked segments. These segments are 600 ms before an incorrect response is made and 800 ms following the incorrect

response. These segments are then baseline corrected with the period of -600 to 400 ms prior to the incorrect response (Davies et al., 2004). Eye movement artifacts are removed via regression using the VEOG channel using MATLAB (Segalowitz, 1996), followed by second baseline correction. Segments containing greater than ±100 μV at the midline sites Fz, FCz, Cz, or Pz, are rejected.

For each individual, incorrect trials were averaged together to create an averaged ERP. The scoring was completed in MATLAB utilizing a program that automatically selects the peak and latency of the peaks (Gavin, 2013). The windows for the ERN peak were -10 to 150 ms and -120 to 50 ms for the previous positive component (P3r). This program also allows visual inspection so that if the "peak" is on a slope at the beginning or end of a window, the peak measure can be moved manually to the proper location. For this study, peak-to-peak measurement was used, which is computed by subtracting the P3r amplitude from the ERN amplitude (Gavin et al., 2019).

The Adaptive Woody filter (Harris & Woody, 1969) was used to adjust for trial-to-trial latency variability in the incorrect trial segments (Gavin et al., 2019). In the processing stages described above, the segments are saved and then called into the MATLAB adaptive Woody filter program. Using a template from the individual's averaged ERP, the Adaptive Woody filter program shifts each segment forward and backwards until each segment is maximally aligned to the template which includes the ERN component. Once this alignment has been found, the shifted segments are averaged to obtain a "latency-adjusted" averaged ERP. Finally the "latency-adjusted" ERN peak (-10 to 150 ms) and P3r peak (-120 to 50 ms) are scored using the MATLAB program that automatically selects the peak and latency of the peaks (Gavin, 2013) as described above.

The grand averages obtained from averaging the incorrect trial ERPs for each participant were used to create a topographic map (see Figure A4). The information from this topographic map was used to confirm that a strong ERN signal was at the frontal sites, Fz and FCz, for this study sample. For this study, ERN was scored from the FCz site.

Children whose error rates on the speeded flanker task were > 38% were excluded from analyses of ERN amplitude to exclude those who may have been performing at random, consistent with previous studies (Davies et al., 2004; Gavin et al., 2019). Trial-to-trial variability of the ERN latency was highly correlated with participant age ($r = -.27$, $p < .01$) (see Figure A5). Therefore, Woody filtered ERN scores, which adjust for latency jitter, were used in analyses.

**Hair Cortisol Concentration (HCC)**

Hair samples were collected when hair was at least 3 cm long. Hair samples were cut from the back of the head along the midline as close to the scalp as possible. Hair samples were then stored and shipped in batches to Dr. Kestutis Bendinskas' lab at the State University of New York at Oswego. There, 20-50 mg of hair were mixed with isopropanol and minced into 2 mm pieces and then washed 4 times with .5 mL of isopropanol for 30 seconds to remove any contamination (Blodgett et al., 2017). Cortisol is extracted with 1 mL of methanol overnight, 1 mL acetone for 5 minutes, and then 1 mL of methanol overnight again (Slominski et al., 2015). Any solvent that remains is removed with nitrogen. Samples are then dissolved in an assay diluent, randomly distributed to different plates to avoid any batch effect, and analyzed using Salimetrics cortisol enzyme-linked immunosorbent assay (ELISA) (Blodgett et al., 2017). Five percent of samples are also randomly reanalyzed to ensure reproducibility. The average picogram of cortisol in a milligram of hair was calculated, creating the HCC (pg/mg). Children's HCC was used in analyses. HCC values that were 1.5 × interquartile range (IQR) above Q3 were

excluded from analyses (n = 11) (Bendinskas et al., 2024; Han et al., 2012). HCC was log-transformed to correct for skewness.

To collect data on potential hair cortisol confounds, parents answered questions regarding their child's hair care and medication use, including the child's natural hair color, how frequently and recently the child washed their hair, if they use any hair dye, and if they took any corticosteroid medications in the past 3 months (Merz et al., 2019). Analyses were conducted to examine the associations between child HCC and these potentially confounding factors (Gray et al., 2018). HCC was not significantly associated with hair washing frequency ($p$ = .32), hair washing recency ($p$ = .24), hair color ($p$ = .14), hair dye ($p$ = .84), or corticosteroid medication use ($p$ = .34). Therefore, none of these variables were included as covariates in the regression analyses.

**Statistical Analyses**

All analyses were conducted in R (version 4.3.1). Multiple linear regression models were used to examine associations among socioeconomic factors (parental education, family income-to-needs ratio), family stressor exposure, HCC, and ERN amplitude. Primary models tested whether socioeconomic factors were associated with family stressor exposure; whether family stressor exposure was associated with HCC and ERN amplitude; and whether HCC was associated with ERN amplitude. In supplemental models, perceived stress, negative life experiences, and material hardship were examined separately. All models included covariates relevant to each outcome, including child age and sex, flanker task error rate for ERN models, and assay batch for HCC models. HCC assay batch was dummy coded to correspond to the batch in which hair samples were processed (1–4) and was included as a covariate in analyses that included HCC to account for potential batch-related variability. Child sex was only significantly

associated with HCC ($p$ = .01) but no other main variables. Therefore, sex was only a control variable in analysis including HCC. Child race/ethnicity was significantly associated with family income-to-needs ratio, $F(5, 100) = 3.39$, $p$ = .007), and parental education, $F(5, 101) = 4.25$, $p$ = .002, as expected. However, race/ethnicity was not associated with any of the other main variables and was therefore not included as a covariate in analyses. Statistical significance threshold was set to $p < .05$ for all analyses.

Mediation analyses were planned to evaluate whether HCC mediated associations between family stressor exposure and ERN amplitude. However, because family stressor exposure was not significantly associated with HCC in children (the hypothesized 'a' path of the mediation model; see Figure A1), mediation analyses were not conducted.

Moderation analyses were conducted using multiple linear regression models including family stressor exposure × positive parenting interaction terms to test whether positive parenting buffered associations between family stressor exposure and HCC or ERN amplitude. Supplemental models also examined perceived stress, negative life experiences, and material hardship separately. Effect sizes ($\eta_p^2$) are presented for significant results, with values of .01, .06, and .14 indicating small, medium, and large effects, respectively (Richardson, 2011).

# RESULTS

## Descriptive Statistics

Descriptive statistics for the main study variables are provided in Table 1, and zero-order correlations between the main study variables are provided in Table 2.

## Associations Between Socioeconomic Factors and Family Stressor Exposure

*Family income-to-needs ratio*

Lower family income-to-needs ratio was significantly associated with greater family stressor exposure when controlling for parent age and sex ($\eta_p^2 = .21$) (see Table 3 and Figure 2). In supplemental analyses, family income-to-needs ratio was significantly associated with perceived stress ($\beta = -.27$, $p < .001$, $\eta_p^2 = .10$), negative life experiences ($\beta = -.24$, $p < .05$, $\eta_p^2 = .05$), and material hardship ($\beta = -.52$, $p < .001$, $\eta_p^2 = .28$).

*Parental education*

Lower parental education was significantly associated with greater family stressor exposure when controlling for parent age and sex ($\eta_p^2 = .18$) (see Table 3 and Figure 2). In supplemental analyses, parental education was significantly associated with perceived stress ($\beta = -.23$, $p < .05$, $\eta_p^2 = .08$), negative life experiences ($\beta = -.25$, $p < .05$, $\eta^2 = .07$), and material hardship ($\beta = -.49$, $p < .001$, $\eta_p^2 = .23$).

## Associations Between Family Stressor Exposure and ERN Amplitude

Family stressor exposure was not significantly associated with ERN amplitude when controlling for age and flanker task error rate ($\beta = -.14$, $p = .14$) (see Table 4). This association remained non-significant when socioeconomic factors (parental education / family income-to-needs ratio) were added as covariates to the model ($\beta = -.09$, $p = .42$ / $\beta = -.13$, $p = .25$). In supplemental analyses, perceived stress ($\beta = .04$, $p = .70$), negative life experiences ($\beta = -.09$, $p = .34$), and material hardship ($\beta = -.07$, $p = .49$) were not significantly associated with ERN amplitude.

## Associations Between Family Stressor Exposure and HCC

Family stressor exposure was not significantly associated with children's HCC when controlling for age, sex, and HCC batch (β = .05, *p* = .66) (see Table 5). This association remained non-significant when socioeconomic factors (parental education / family income-to-needs ratio) were added as covariates to the model (β = .11, *p* = .33 / β = .12, *p* = .35). In supplemental analyses, perceived stress (β = .12, *p* = .27), negative life experiences (β = .06, *p* = .54), and material hardship (β = .04, *p* = .73) were not significantly associated with HCC.

## Associations Between HCC and ERN Amplitude

Higher HCC was significantly associated with smaller (less negative) ERN amplitudes when controlling for age, sex, batch, and flanker task error rate ($\eta_p^2$ = .02) (see Table 6 and Figure 3).

## Positive Parenting as a Moderator

### *HCC*

Positive parenting did not moderate the association between family stressor exposure and HCC when controlling for age, sex, and HCC batch (β = -.08, *p* = .55). The interaction term remained non-significant when additionally controlling for parental education (β = -.06, *p* = .62) and family income-to-needs ratio (β = -.09, *p* = .48). The interaction term was also non-significant in supplemental analyses focused separately on perceived stress (β = -.06, *p* = .63), negative life experiences (β = .05, *p* = .69), and material hardship (β = -.02, *p* = .89).

*ERN amplitude*

Similarly, positive parenting did not moderate the association between family stressor exposure and ERN amplitude when controlling for age and flanker task error rate (β = -.16, $p$ = .07). The interaction term was also non-significant in supplemental analyses focused separately on perceived stress (β = -.17, $p$ = .06), negative life experiences (β = -.16, $p$ = .07), and material hardship (β = -.16, $p$ = .07).

## DISCUSSION

The goal of this study was to examine how SES-related family stress and children's physiological stress processes shape children's capacity to learn from mistakes, and to examine the potential role of positive parenting as a moderator of these associations. Some of my pre-registered hypotheses were supported, whereas others were not. Consistent with prior research, this study replicated the well-established link between socioeconomic disadvantage and greater family stressor exposure in children (K. J. Conger et al., 2000; Evans & Kim, 2013). Importantly, this study disentangled the unique contributions of reported family stressor exposure and physiological stress activation to children's neural error monitoring. Higher HCC was significantly associated with a decreased ERN in children but family stressor exposure was not associated with children's ERN amplitude. These findings suggest that it may not be exposure to stressors alone, but rather children's physiological response to stressors that shapes neural sensitivity to mistakes. This study also found that positive parenting did not moderate the impact of family stressor exposure on HCC or ERN. Together, these findings highlight physiological

stress as a key pathway through which environmental experiences may shape children's neural sensitivity to errors.

### Associations Between Socioeconomic Factors and Family Stressor Exposure

In support of my pre-registered hypothesis, indicators of lower SES were significantly associated with increased family stressor exposure. This finding is consistent with extensive previous literature that has linked indicators of lower SES (e.g., income, education, occupation) with higher levels of stressors and negative life experiences for parents and children (Cohen & Janicki-Deverts, 2012; Cundiff et al., 2022; Hatch & Dohrenwend, 2007; Lantz et al., 2005).

### Family Stressor Exposure and ERN Amplitude in Children

Contrary to my pre-registered hypothesis, family stressor exposure was not related to children's ERN amplitude. This result may relate to the few studies that have examined the associations between socioeconomic factors and ERN amplitudes in children, which have yielded inconsistent results (Brooker, 2018; Conejero et al., 2016; Perera-W.A. et al., 2021). These past studies relied on measures of SES such as family income, parental occupation, and parent education. Even work that examined measures of family stressors comparable to those in the present project have also reported inconsistent relationships between stressor exposure and ERN amplitude in children (Lackner et al., 2018; Mehra et al., 2022).

One explanation for these inconsistencies is that the ERN in children may be more sensitive to the accumulation of stressors across development. Longer durations of stressor exposure has been found to have an impact on children's cognitive control (Raver et al., 2013; Rudd et al., 2021), mental health (Schilling et al., 2007; Thurston et al., 2023), and behavioral problems (Appleyard et al., 2005), which all rely on error monitoring. Longitudinal studies have

shown that children's victimization at baseline predicts increases in ERN amplitude two years later (S. L. Cole et al., 2023) and baseline hostile parenting predicts a larger ERN in children 3 years later (A. Meyer et al., 2015). Studies of the ERN in adults has also found that greater severity of childhood adversity was associated with a larger ERN in adulthood (Banica et al., 2022; J. Wu et al., 2021). Together, these findings suggest that error monitoring and ERN functioning may reflect stress accumulated across many years of development, which may not be fully captured by the family stressor exposure composite score used in this study.

The ERN has also been theorized to be especially sensitive to contexts that increase the perceived threat or salience of errors. For example, studies have found that laboratory tasks that increase the threat-value of committing an error such as having the child be observed by a friend (Kim et al., 2005) or a controlling parent (Meyer et al., 2019) increase the ERN significantly more than completing the task in a non-threatening environment. Similar lab based tasks where the child was punished for making a mistake (S. L. Cole et al., 2022) or given a task alongside unpredictable stimuli (Speed et al., 2017) produced a larger ERN than when the child did the same task in a calm, predictable environment. Children's self-reported error sensitivity has also been found to be associated with a larger ERN (Chong & Meyer, 2019) and an intervention focusing on reducing children's feelings of error sensitivity by addressing perfectionism, fear of the social consequences of making mistakes, and over-evaluation of the negative consequences of errors successfully reduced ERN amplitudes in young adults (Meyer et al., 2020). Stressor exposure may heighten children's sensitivity to potential mistakes by shaping expectations about threat, evaluation, or punishment (Tone, 2015). However, this link is unlikely to be uniform across adversity types and stressor contexts, possibly leading to inconsistent results on the impact of stress- or adversity-related variables on the ERN in children. Different forms of stress or

adversity may have differential patterns of associations with neural error processing measured by the ERN. Chronic or acute stressors that explicitly link mistakes with punishment, loss of privileges, criticism, or social threat may be more likely to heighten ERN amplitude. Thus, children exposed to different types of stressors may differ in whether they learn to associate mistakes with threat, which could contribute to variability in ERN findings across studies and help explain inconsistent results for ERN amplitude.

Together, this body of work suggests that alterations in children's ERN amplitude may emerge primarily in the context of prolonged or threat-related adversity exposure, particularly when stressors increase the perceived negative consequences of mistakes. The insignificant association observed in the present study may indicate that broader measures of parent-reported stressor exposure do not uniformly translate into heightened neural sensitivity to errors, as measured by the ERN, in children. Alternatively, this pattern may suggest that such neural alterations have not yet fully emerged in this sample, highlighting a potential window for prevention and early intervention before alterations in error-monitoring processes become more established. Future studies should incorporate longitudinal designs and measures that capture cumulative stressor exposure over time, children's subjective and contextual experience of errors, and adversity dimensions to build on results from the current study.

**Family Stressor Exposure and HCC in Children**

Contrary to my pre-registered hypothesis, family stressor exposure was not associated with children's HCC. This contributes to the current conflicting literature on what impacts children's HCC. For example, lower parental education has been associated with elevated child HCC in some studies (Anand et al., 2020; Merz et al., 2019; Tarullo et al., 2020; Ursache et al., 2017; Vaghri et al., 2013; Vliegenthart et al., 2016) but others have found no association (Bryson

et al., 2019; Liu et al., 2020). Similarly, associations between parental perceived stress and child HCC have been observed in several samples (Babarro et al., 2023; Bates et al., 2017; Bhopal et al., 2019; Bilodeau-Houle et al., 2025; Kang et al., 2024; Li et al., 2023; Ouellet-Morin et al., 2021; Palmer et al., 2013; Shan et al., 2024; Simmons et al., 2016), while others have found no association (Isaac et al., 2023; Koenig et al., 2018; Liu et al., 2016; Olstad et al., 2016; Romero-Gonzalez et al., 2018).

These inconsistencies may reflect variability in susceptibility rather than the absence of stress-related biological effects. While a meta-analysis found that studies using cumulative indices of adversity were more likely to detect associations with higher child HCC compared to studies examining individual stressors (Bryson et al., 2021), this association may be moderated by factors such as protective resources and the duration of stress exposure. Ling and colleagues (2020) found that parental perceived stress was associated with children's HCC only among parents reporting low coping capacity, indicating that caregiving resources may determine whether environmental stress translates into sustained physiological activation. Ouellet-Morin et al. (2021) reported non-linear associations between cumulative adversity and children's HCC, with cortisol levels remaining relatively stable across low-to-moderate adversity and increasing primarily at the highest levels of cumulative risk. This suggests that high levels of prolonged exposure to stressors may be required to impact HCC levels. Consistent with this, parent-reported child lifetime exposure to stressors, but not past-year stressful life events, predicted children's HCC, highlighting the importance of longer-term exposure (DePasquale et al., 2021). Prior work using salivary cortisol further demonstrates that each additional year of poverty is associated with greater HPA axis dysregulation (Evans & Kim, 2007). Given the wide age range in this sample, children likely differed substantially in the duration of adversity they experienced.

If HCC is more sensitive to prolonged or cumulative exposure, as suggested by previous work, then a past-year stress measure may obscure meaningful variability in chronic stress burden on the HPA axis. Together, these findings suggest that stressor exposure may not produce uniform increases in HCC across children; rather, associations may emerge primarily as a function of cumulative life experience, particularly under conditions of heightened vulnerability or more severe, prolonged adversity.

Methodological factors may also have attenuated detectable associations. Meta-analytic evidence indicates that associations between stressful life events, perceived stress, and HCC are strongest when reported stress measures are temporally aligned with the hair cortisol sampling window (Li et al., 2023). Although the composite stress measure in the current study aimed to capture proximal stress exposure, its components varied in temporal scope (past year and past month), potentially introducing misalignment with HCC, which reflected cortisol accumulation over approximately three months in this study. In addition, Li et al. (2023) reported that associations between stressor exposure and HCC vary by assay method, with significant effects observed for LC-MS/MS but not ELISA or CLIA (Slominski et al., 2015).

Taken together, the present findings highlight that children's HCC may not reflect family stressor exposure in a uniform or linear manner. Instead, HCC appears sensitive to specific contextual and methodological conditions, including caregiving resources, cumulative risk severity, temporal alignment between stress exposure and cortisol accumulation, and assay methodology. Within this framework, the null association observed here may reflect both heterogeneity in children's susceptibility to stress and considerations inherent in capturing dynamic stress processes with a single biomarker.

**Associations Between HCC and ERN Amplitude in Children**

In support of my pre-registered hypothesis, higher HCC was associated with a smaller (less negative) ERN in children. This result is consistent with previous work that found associations between acute cortisol reactivity and ERN amplitude in adults (Compton et al., 2013; Hu et al., 2019; Tops & Boksem, 2011). Importantly, the present findings extend this work by demonstrating that similar associations are observed when cortisol is indexed over a longer timescale in children, suggesting that sustained cortisol exposure may have effects on neural performance-monitoring systems.

This interpretation is further supported by other research that has examined the impact of cortisol on the functioning of the ACC, which produces the ERN (Bush et al., 2000). Specifically, prolonged exposure to glucocorticoids (such as cortisol) is associated with morphological alterations in brain regions involved in the regulation of HPA activity such as medial PFC and ACC regions (Treadway et al., 2009). Similarly, studies find that those with high cortisol reactivity have deactivation in the ACC during stressful conditions (Dedovic et al., 2009; Pruessner et al., 2008), providing a plausible neural mechanism through which chronic cortisol output may blunt ERN amplitude.

Previous work has relied on parent- and self-report measures of stressor exposure when examining the impact of stress on the ERN in children, yielding inconsistent results (Brooker, 2018; Conejero et al., 2016; Lackner et al., 2018; Loman et al., 2013; Mehra et al., 2022; Perera-W.A. et al., 2021). This is the first study to examine the associations between HCC and the ERN in children, with HCC providing a measure of prolonged cortisol exposure. By using HCC, the present findings extend prior work by capturing sustained activation of the HPA axis, which may

be particularly relevant for children growing up in lower-SES contexts often characterized by chronic and cumulative stressors.

Importantly, the divergence between stressor exposure and HCC in predicting ERN amplitude suggests that environmental stressors and physiological stress embedding may represent distinct pathways influencing neural performance monitoring. Whereas environmental measures capture the presence of stressors, HCC indexes sustained biological activation of the HPA axis stress response system. This distinction may help explain inconsistencies across prior studies, as not all children exposed to stressors may exhibit altered HPA-axis activation. Together, these findings suggest that exposure to family stressors and physiological responses to stress may have differential effects on the ERN in children.

The finding that children with higher levels of HPA-axis activation exhibit a reduced ability to monitor errors has important implications for academic achievement and mental health outcomes. Prior research has consistently shown that children from lower socioeconomic backgrounds are at elevated risk for academic difficulties and internalizing and externalizing problems, outcomes that have been linked to disruptions in cognitive control and performance-monitoring processes (Cuartas et al., 2022; McNeilly et al., 2021; Micalizzi et al., 2019; Rakesh et al., 2025; Yang et al., 2022). Blunted error monitoring has been found to relate to difficulties with learning from mistakes, behavioral adjustment following errors, and vulnerability to psychopathology in children. Thus, this work suggests that chronic physiological stress may interfere with children's ability to use mistakes as learning signals, potentially contributing to downstream academic and emotional difficulties. This highlights the importance of interventions that both reduce chronic stressor exposure and support the development of adaptive performance monitoring. For example, interventions targeting family stress (e.g., reducing economic strain or

supporting caregiver coping), as well as school-based programs that promote error recognition and tolerance, feedback sensitivity, and adaptive responses to mistakes, may help buffer against stress-related disruptions in learning and self-regulation. Identifying children with elevated physiological stress may also provide an opportunity for early intervention before these difficulties become more entrenched across development.

**The Moderating Role of Positive Parenting**

Contrary to my hypotheses, positive parenting did not moderate the relationship between family stress exposure and HCC or family stress exposure and ERN. Previous work has found that parenting quality moderates the association between SES and HPA axis function, as measured by salivary cortisol (Blair et al., 2008; Blair, Granger, et al., 2011; Blair, Raver, et al., 2011; Brown et al., 2020; Johnson et al., 2018; Zalewski et al., 2012). Only one study has examined the role of positive parenting on HCC in children from low SES backgrounds. This study found that positive parenting reduced HCC in children from disadvantaged backgrounds (Simmons et al., 2019); however, it did not directly test whether parenting quality interacted with level of disadvantage to alter the strength of the association between socioeconomic risk and chronic HPA activity. Similarly, very few studies have examined the impact that positive parenting has on child HCC in any context (Bryson et al., 2021; Ouellette et al., 2015; Rickmeyer et al., 2017; Schloß et al., 2019). The role of positive parenting may be shaping children's moment-to-moment regulation of stress responses rather than the overall physiological demand placed on the HPA axis across development (Morris et al., 2017; Zimmer-Gembeck et al., 2022). Parenting may attenuate acute cortisol reactivity without substantially altering the cumulative cortisol burden indexed by HCC.

While previous work has consistently found that positive parenting protects against the negative effects of socioeconomic disadvantage on neural function associated with emotion regulation and cognitive control (Brody et al., 2019; Hyde et al., 2020; Merz et al., 2024; Whittle et al., 2017), few studies have examined the role of parenting on ERN development. In one study, only young children from higher SES households with positive parenting showed normative ERN development (Brooker, 2018). This previous work may suggest that caregiving quality may be particularly influential during early developmental periods when performance-monitoring systems are emerging. In contrast, the children in the current sample were older. It is therefore possible that positive parenting exerts its strongest influence on ERN development earlier in life, with more limited capacity to modify individual differences in ERN magnitude later in childhood. This is supported by previous research that found that negative parenting moderated the relationship between ERN and anxiety symptoms, but only in younger children (Chong et al., 2020). Therefore, to find a moderating effect of positive parenting on the relationship between family stress and ERN, another study may need to focus on a younger sample of children.

Finally, there was a restricted range of positive parenting in the subsample with positive parenting data, which was already smaller than the full dataset. Results suggested possible limited representation of lower levels of positive parenting. This restriction of range likely reduced variability in the moderator and limited power to detect interaction effects (Cohen et al., 2013). Additionally, the relatively small sample sizes for these analyses may have reduced statistical power to detect moderation effects.

# LIMITATIONS

There are limitations to this study that need to be considered when interpreting the findings. Because of the cross-sectional, correlational design, no casual inferences can be made based on these findings. Future studies utilizing longitudinal designs are needed to better understand how the relationship between these variables may change over time (D. A. Cole & Maxwell, 2003). Parenting quality was solely measured using parents' self-report completed by one parent. As such, we do not know if parenting quality may differ for the parent who did not complete the questionnaire, or how the children would rate their parent's parenting style. This sample is largely white, non-Hispanic/Latine. The compounding stress of low socioeconomic status and exposure to racial/ethnic discrimination disproportionately impacts people of color (Strait, 2001). Having a majority white sample limits us from making conclusions about these associations among non-white children. This sample also had a large age range (5 – 13 years), and while all analyses did control for age, narrowing the age range in future studies may elucidate patterns of neural activity that are more prevalent for younger or older children. Finally, family stressor exposure was a composite score that summed different stressor/adversity types. Different results may be found if adversity dimensions (e.g. threat, deprivation, uncertainty, unpredictability) were measured and analyzed separately.

# CONCLUSION

While this study did not find family stressor exposure to be associated with children's ERN amplitude or HCC, it did find that children's physiological stress, indexed by hair cortisol, was significantly related to blunted neural error monitoring. This pattern is consistent with the broader literature demonstrating substantial variability in how stress is experienced, perceived, and buffered, and suggests that the impact of adversity on neural systems may be difficult to detect when relying solely on environmental or self-report indicators that are shaped by coping skills, caregiving contexts, and measurement timeframes. By contrast, HCC may capture the downstream physiological consequences of diverse stressors, regardless of their specific source, and thus provide a more integrative index of the cortisol burden that ultimately affects neural functioning. The finding that higher chronic cortisol output was associated with a reduced ERN extends prior work on acute stress reactivity and error monitoring and highlights that sustained HPA-axis activation may associate with altered function of performance-monitoring systems in childhood. Including both environmental stressor exposures and physiological responses allowed for consideration of distinct stress-related processes, suggesting that ERN alterations in children may be more closely linked to HPA axis regulation than to family stressor exposure.

Future research should use longitudinal designs to track how the timing, severity, type, and duration of stressor exposure relate to changes in both HCC and ERN across development. Clarifying when environmental stressors translate into altered physiological regulation, and when buffering factors such as positive parenting interrupt this process, will be critical for identifying which types of interventions are most effective and when they should be implemented.

Specifically, this work can inform the timing and design of interventions, more effectively supporting families and children in the future.

TABLES

**Table 1**

*Descriptive statistics for sample characteristics and main study variables*

| | *N* | *M* | *SD* | *Range* | *Skew* |
|---|---|---|---|---|---|
| Age (years) | 108 | 8.7 | 2.5 | 5.1 – 13.9 | .39 |
| Parental education (years) | 108 | 16.4 | 2.8 | 11 - 22 | .33 |
| Family income (U.S. dollars) | 107 | 115,574 | 67,519 | 500 - 300,000 | .22 |
| HCC | 86 | 7.60 | 5.45 | 1.4 – 25.71 | 1.28 |
| ERN amplitude (μV) | 95 | -19.31 | 8.56 | -47.3 – -1.2 | -0.94 |
| Parental Perceived stress | 107 | 20.98 | 7.07 | 5 – 38 | -0.10 |
| Negative life events | 106 | 9.00 | 13.19 | 0 – 102 | 5.00 |
| Material hardship | 107 | 1.06 | 2.05 | 0 – 8 | 2.14 |
| Positive parenting | 85 | 70.59 | 5.61 | 56 – 80 | -0.49 |
| | | *n* | *%* | | |
| Sex | | | | -- | -- |
| Male | | 62 | 57 | | |
| Female | | 46 | 43 | | |
| Race/ethnicity | | | | -- | -- |
| White | | 79 | 73 | | |
| Black or African American | | 2 | 2 | | |
| Asian | | 10 | 9 | | |
| Hispanic/Latine | | 14 | 13 | | |
| American Indian or Alaska Native | | 1 | 1 | | |
| Other | | 1 | 1 | | |
| Unknown | | 1 | 1 | | |

**Table 2**

*Zero-order Correlations Between Main Study Variables*

| Variable | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 |
|---|---|---|---|---|---|---|---|---|
| 1. Parental education | — | | | | | | | |
| 2. Income-to-needs ratio | .67*** | — | | | | | | |
| 3. Family stressor exposure | -.53*** | -.58*** | — | | | | | |
| 4. Parental perceived stress | -.36** | -.39*** | .75*** | — | | | | |
| 5. Negative life experiences | -.32** | -.34** | .77*** | .34** | — | | | |
| 6. Material hardship | -.53*** | -.60*** | .78*** | .43*** | .36** | — | | |
| 7. HCC | .08 | .03 | .00 | .04 | -.06 | .02 | — | |
| 8. ERN amplitude (μV) | .02 | -.04 | -.05 | .13 | -.24* | .04 | .31** | — |

Note. Correlations are Pearson's r. * $p < .05$. ** $p < .01$. *** $p < .001$.

**Table 3**

*Associations between socioeconomic factors and family stressor exposure*

| | *β* | *p* |
|---|---|---|
| Model 1: Family income-to-needs ratio | | |
| Family income-to-needs ratio | -.45 | < .01 |
| Parent Age | .00 | = .99 |
| Parent Sex | .07 | = .44 |
| Model 2: Parental education | | |
| Parental education | -.40 | < .01 |
| Parent Age | -.05 | = .56 |
| Parent Sex | .05 | = .56 |

**Table 4**

*Associations between family stressor exposure and ERN amplitude*

| | *β* | *p* |
|---|---|---|
| Family stressor exposure | -.09 | .42 |
| Age | .01 | .95 |
| Flanker task error rate | .53 | < .01 |
| Parental education | .12 | .29 |

**Table 5**

*Associations between family stressor exposure and HCC*

| | *β* | *p* |
|---|---|---|
| Family stressor exposure | .17 | .16 |
| Age | -.25 | .02 |
| Sex | -.25 | .03 |
| Parental education | .05 | .71 |

*Note*. Analyses additionally controlling for HCC batch yielded the same pattern of non-significant results.

**Table 6**

*Associations between HCC and ERN amplitude in children*

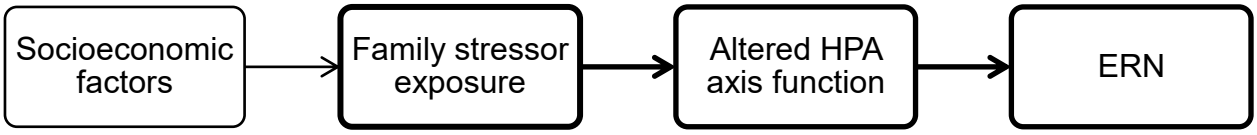
| | *β* | *p* |
|---|---|---|
| HCC | .23 | < .05 |
| Age | .04 | .70 |
| Sex | -.02 | .89 |
| Flanker task error rate | .45 | < .01 |

*Note*. Analyses additionally controlling for HCC batch yielded the same pattern of results.

FIGURES

**Figure 1**

*Conceptual Mechanistic Model Guiding Primary Aims*



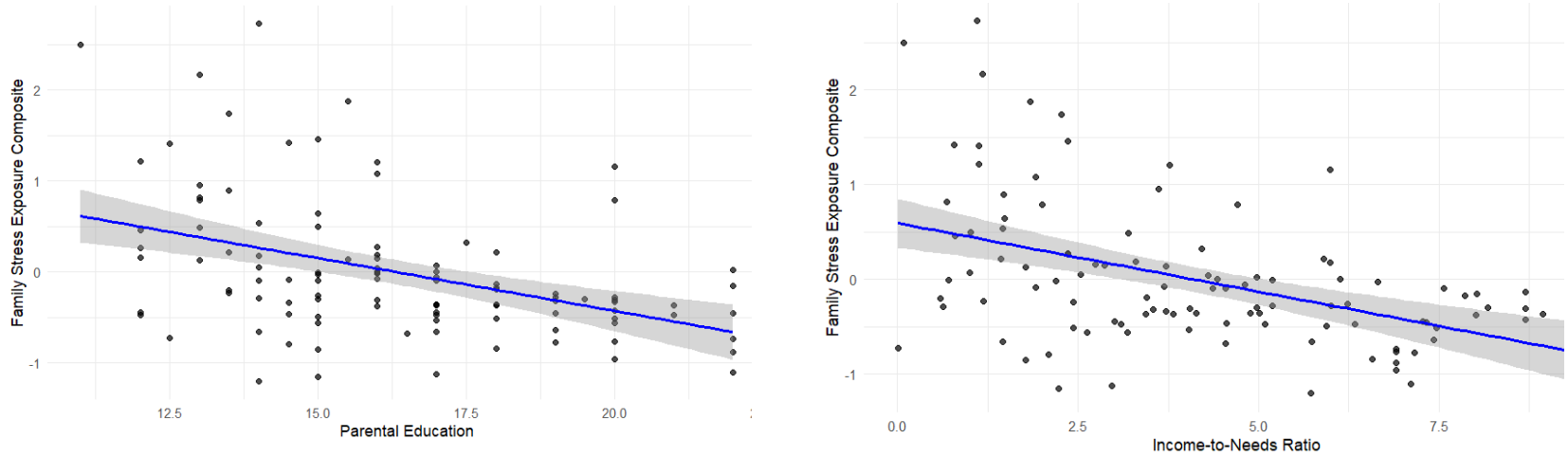

*Note*: HPA, hypothalamic-pituitary-adrenal; ERN, error-related negativity

[a] This figure shows the full mechanistic model that is the basis for the primary aims of this study. The bolded parts of the figure show the more specific focus of the primary aims of this study.

**Figure 2**

*Correlations Between Socioeconomic Factors and Family Stressor Exposure*

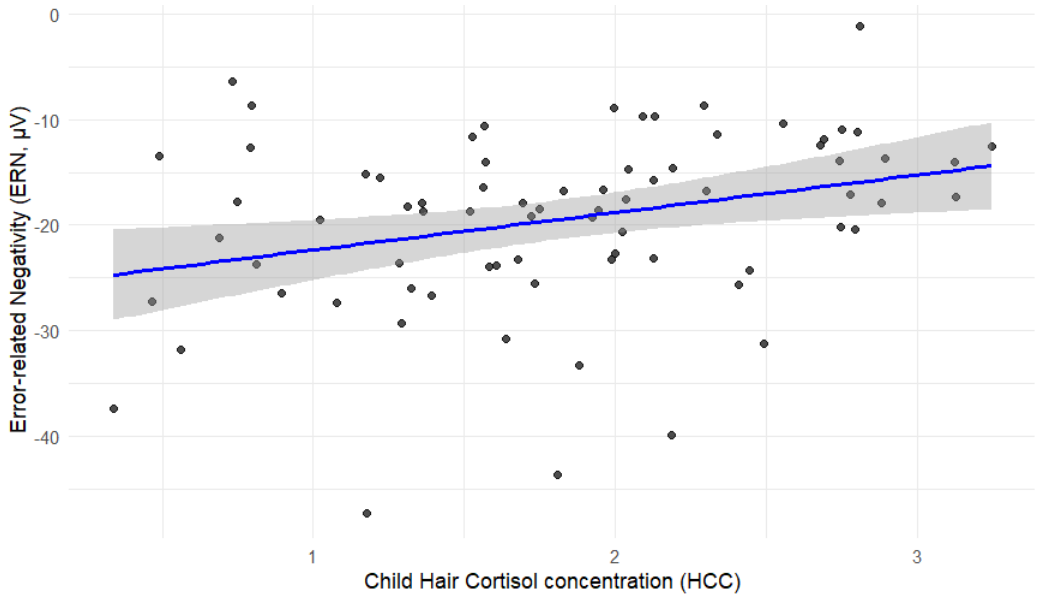
**a)** **b)**




[a] This figure shows zero-order correlations between family stressor exposure and parental education and between family stressor exposure and family income-to-needs ratio.

**Figure 3**

*Correlation Between Hair Cortisol Concentration (HCC) and Error-Related Negativity (ERN) Amplitude in Children*




[a] This figure shows the zero-order correlation between HCC and ERN amplitude in children.

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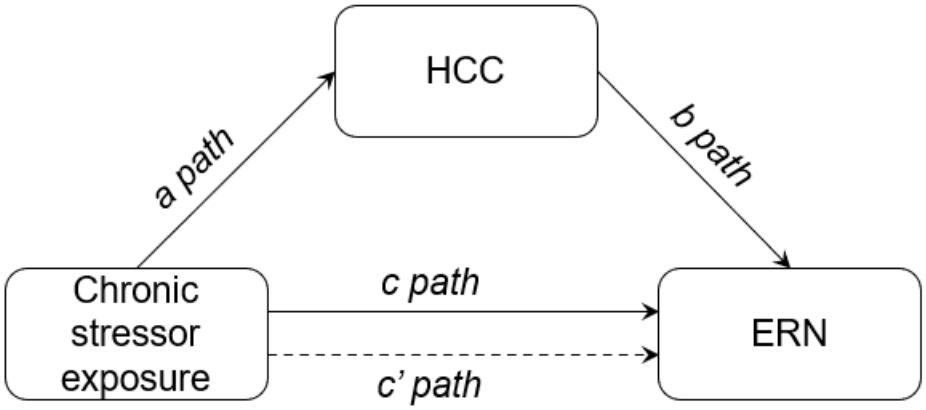
APPENDIX

**Figure A1**

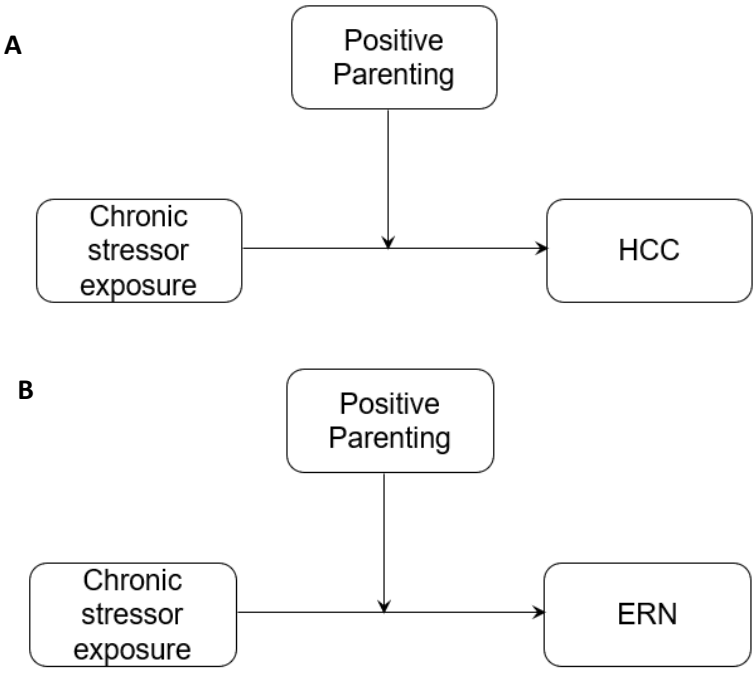
*Hypothesized Mediation Model*




*Note*: HCC, hair cortisol concentration; ERN, error-related negativity

**Figure A2**

*Hypothesized Moderation Model for HCC and ERN Amplitude*




*Note*: HCC, hair cortisol concentration

**Figure A3**

*Speeded Arrow Flanker Task Examples*

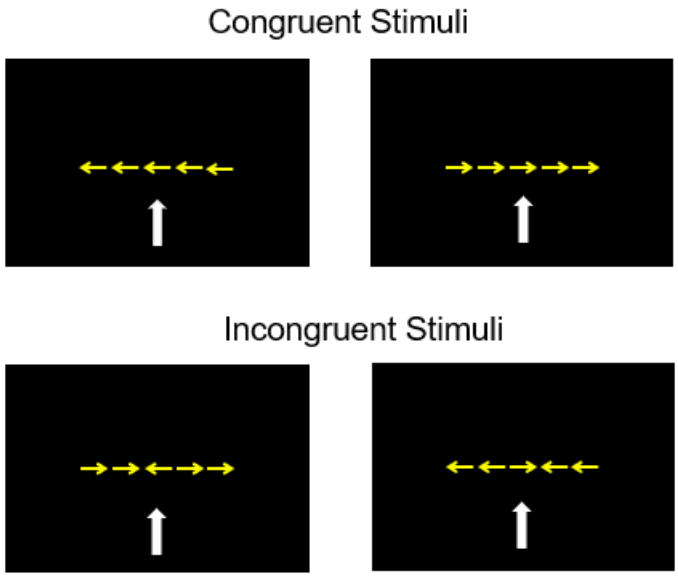



**Figure A4**

*Grand Average of Incorrect Trial ERNs*

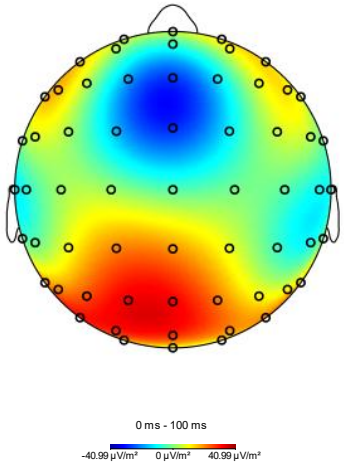


**Figure A5**

*Age and trial-to-trial variability of the ERN latency*