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The Associations of Maternal Emotion Dysregulation and Parenting Behavior with White Matter Integrity in Children

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An abstract of
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Abstract

Background

Parental emotion regulation plays a significant role in parents' interactions with their children, influencing the development of children's emotional and cognitive processes. These interactions are known to affect neural plasticity, particularly during sensitive periods of development. However, little is known about how parental emotion dysregulation (ED) and parenting style is associated with variation in children's brain structure, including white matter (WM) connectivity, which was the goal of this study.

Methods

Forty-five African American mother-child dyads were recruited from an intergenerational trauma study; mothers were given ED and parenting style (parenting questionnaire, PQ) measures. Diffusion-weighted images were collected, and child emotion regulation measures were administered to children; deterministic tractography was used to reconstruct WM pathways relevant to emotion regulation. Metrics of WM microstructure and connectivity (e.g., fractional anisotropy (FA), mean diffusivity (MD), and radial diffusivity (RD)) were extracted for each pathway.

Results

Maternal ED negatively correlated with connectivity metrics of the right fornix (MD: r=0.48, p=0.001; RD: r=0.46, p=0.002). No significant correlations were observed between child WM indices and the PQ. Parent-report sadness inhibition negatively correlated with MD of the right cingulum bundle (r=-0.44, p=0.003), whereas child-report anger inhibition positively correlated with RD of the right superior longitudinal fasciculus segment 3 (r=0.44, p=0.003).

Conclusion

ED in parents may influence the development of a hippocampal-striatal pathway (e.g., fornix) which is implicated in reward/motivation; child anger and sadness inhibition may influence different white matter tracts implicated in ER and stress response. These data suggest that dysregulated parenting may adversely impact adaptation to trauma/stress in children by affecting the connectivity of these WM pathways.

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Chapter I. Introduction

Emotion regulation (ER) has been described as the ability to monitor, evaluate, and modulate the occurrence, intensity, and expression of emotions (Thompson, 1994). ER is an integrative management system that includes internal components such as neurophysiological, cognitive, behavioral systems, and external components such as social context, and cultural norms (Han & Shaffer, 2013). ER refers to the process that shapes emotional experience and reactions for serving adaptive behavior (Beauchaine, 2015). As a hallmark of many mental disorders, emotion dysregulation is contingent upon the social context where the emotion occurs. Scientists consider the emotional response dysregulatory when it disrupts the normal psychological and behavioral functioning or becomes inappropriate within a certain social context (Han & Shaffer, 2013). Adaptive ER involves awareness and acceptance of emotions, the ability to stay engaged and persist with goal-oriented behaviors in the face of negative emotions, controlling impulsive behaviors, and the ability to use emotion regulation strategies when necessary, to maintain emotions over time (Gratz & Roemer, 2004; Lazarus, 1991).

Emotion dysregulation (ED) is a maladaptive process defined as “a pattern of emotional experience and/or expression that interferes with appropriate goal-directed behavior” (Beauchaine, 2015, p. 876). Psychologists have converged on the consensus that ED is present across disciplines and theoretical frameworks as a transdiagnostic criterion for psychopathology (Aldao, 2012; Kring &Sloan, 2009; Carpenter & Trull, 2012). This recognition is derived from the empirical evidence indicating 1) one or more forms of EDs are observed across psychological diagnoses, including externalizing, internalizing, and psychotic disorders (Beauchaine & Cicchetti, 2019; Gross & Jazaieri,

2014); 2) the neural underpinnings of disrupted modulation of emotion is found through neuroimaging research (Palacios-Barrios & Hanson, 2019); 3) ED is shown to be a risk factor for children to develop psychological disorders in adulthood (Poole et al., 2018, Beauchaine & Cicchetti, 2019). Substantial empirical evidence has demonstrated the association between ED and internalizing and externalizing disorders, suggesting that the development of psychopathology may stem from ED (Zeman et al., 2006; Buckholdt et al., 2014; Beauchaine & Cicchetti, 2019; Aldao et al., 2014). In recent decades, emotion dysregulation (ED) has been recognized as an important construct for understanding various adaptation problems on the developmental trajectory (Beauchaine & Cicchetti, 2019). Cole and Hall (2008) pinpointed four features of ED, emotional interference with behavioral functioning, contextually inappropriate emotional expression, inability to regulate emotions, and inappropriate transition between different emotional states. One assessment of ED evaluates six dimensions of this construct: nonacceptance of emotional responses, difficulties engaging in goal-directed behavior, impulse control difficulties, lack of emotional awareness, limited access to ER strategies, and lack of emotional clarity (Gratz & Roemer, 2004). Emotion dysregulation has been associated with affective disorders and trauma-related disorders, such as depression, anxiety, and post-traumatic stress disorder (Hofmann et al., 2012; Powers et al., 2015).

The neural underpinning of emotional processing and ER have been continually explored in the literature. Emotion regulation processes such as the appraisal of emotional cues, selective inhibition of response, and attentional focus and shifting, are supported by cognitive control and salience neural networks (Phillips et al., 2008; Zilverstand et al., 2017). Key nodes of these networks include: amygdala, hippocampus,

striatum, dorsolateral prefrontal cortex (dlPFC), anterior cingulate cortex (ACC), dorsomedial frontal cortex (dmPFC), ventromedial (vmPFC), ventrolateral (vlPFC) (Phillips et al., 2008). Connectivity between network regions have been linked to specific ER processes: the hippocampus and striatum have been implicated in reward learning (Cox & Witten, 2019); the amygdala and vmPFC pathway have been linked to inhibition of threat response (Åhs et al., 2015); the amygdala and dorsal ACC are involved with salience detection; the amygdala and dmPFC are engaged during appraisal of emotional cues (Peters et al., 2016; Etkin et al., 2011), and the dlPFC and vlPFC engage in executive control functions (Etkin et al., 2015).

Associational and commissural white matter fibers provide connections between these regions involved with ER; among the paths highlighted as most salient to emotion regulation are the uncinate fasciculus (UF), the cingulum (CB), superior longitudinal fasciculus (SLF), inferior longitudinal fasciculus (ILF), corpus callosum (CC), fornix (Tan et al., 2020; Cutuli, 2014; Phelps, 2004; Chahal et al., 2021). Each white matter tract connects different regions of the brain, serving various functions. The UF is a ventrolimbic white matter pathway, connecting the prefrontal cortex with the limbic system. Part of the SLF connects the PFC with the supramarginal gyrus and the temporal lobes, supporting attentional control and shifting (Kamali et al., 2013). The ILF connects the extrastriate cortex of the occipital lobe with the temporal lobe (Herbet et al., 2018). The CB constitutes the dorsolimbic white matter pathway, connecting the limbic system with the cingulate cortex (Versace et al., 2015). The fornix is the major output pathway of the hippocampus, connecting other subcortical limbic structures such as the nucleus accumbens and the striatum. Reduced structural connectivity of the fornix is associated

with a decreased reward learning and processing connectivity of this pathway is also linked to anhedonia, suggesting its general relevance to the experience of positive emotion (Harnett et al., 2020). Lastly, the interhemispheric functional exchange of emotion and cognition is supported by the major commissural tract that connects the left and right hemisphere, the CC, particularly in the prefrontal aspects (Versace et al., 2015).

Parents appear to play a critical role in shaping these connections (Kerr et al., 2019); one pathway through which this may occur is via their impact on the development of ER processes in children. Morris and colleagues (2007) proposed the tripartite model to describe the role of parents on children's ER development. The model includes three processes: 1. Family emotional climate, 2. Parenting practices and 3. Emotional learning and modeling. The model specifies that a parent's emotion regulatory skills, emotion expressiveness, and history of mental health can impact how they model emotion regulation behavior, the emotional climate of the family, and their parenting practices. ED in parents is related to various negative outcomes in children such as ED in children and the onset of internalizing symptoms (Han & Shaffer, 2013). The consistent relationships between parental ED and children's internalizing symptoms have also been identified by other studies (Davis et al., 2014; Crespo et al., 2017; Silva et al., 2018; Powers et al., 2020). Additionally, children exposed to supportive and positive parenting tend to develop healthy ER behaviors, whereas negative parenting is associated with maladaptive ER in children (Morris et al., 2017).

To date, much research effort has investigated the role of various forms of parenting on children (Maag, Phelps, & Kiel, 2020; Clayborne et al., 2020; Vasey & Dadds, 2001). The spectrum of parenting behavior can range from extreme forms of

maltreatment to behaviors that are more common and accepted. Relevant research mainly divides common parenting behaviors into three dimensions: warmth, control, and rejection (Wood et al., 2002). Parental warmth is the use of positive reinforcement such as praising during interactions, responsiveness to children's emotional needs, and acceptance of children's feelings, thoughts, and behaviors (Maccoby, 1992). Besides warmth, positive parenting also includes clear and consistent rules that delineate expectations and boundaries. The rules can help children to learn the socially acceptable ways of expressing emotions and forming their expectations on emotional behaviors (Morris et al., 2017). Parental control is defined as excessive monitoring of children's behaviors, "helicopter" parenting, disregard of children's volition, and dictatorial decision-making (Barber, 1996). Rejection refers to parental withdrawal from child-rearing involvement and hostility toward children (Rapee, 1997). Additionally, corporal punishment for disciplinary purposes is another common phenomenon that is frequently studied in parenting's influence on children that can be seen as both a form of parental control and rejection (Laible et al., 2019).

Guided by Morris' tripartite model, this study aims to examine the relationships of parental ED, parenting behavior, and the white matter connectivity underlying emotion regulation in children. We hypothesize that reduced white matter integrity in children's emotion regulation networks will be associated with more severe maternal ED and negative parenting behaviors. Evidence for this study can lead to increased understanding of potential resilience and risk factors that impact the development of emotion dysregulation and psychopathology among children, guiding resources such as

intervention and prevention efforts to target parent-child relationships and interaction to facilitate better developmental outcomes.

The intergenerational transmission of psychopathological features such as emotion dysregulation presents an ominous implication where adverse outcomes can be continuously cycled and passed out over years across generations. Thus, scientific efforts that seek to elucidate the mechanisms of such transmission become increasingly crucial for intervention and prevention efforts. To date, the majority of relevant research on adverse rearing context has focused on investigating the impact of maltreatment on children's development (McLaughlin et al., 2014). Studies focusing on more accepted and common forms of parenting behavior and its role in shaping children's neurodevelopment are needed (Belsky & de Haan, 2010). There is also a dearth of research exploring the neural mechanisms of the development of emotion dysregulation in children within the context of parenting behavior. The current study seeks to fill this research gap by examining associations between maternal ED, maternal parenting behavior, and white matter connectivity indices in children. The primary objective is to provide more evidence on how maternal emotion regulation may affect structure in relevant neural pathways in children. Secondarily, I examined how these white matter connectivity indices are associated with child emotion regulation.

Chapter II. Literature Review

This thesis project seeks to explore the associations of maternal emotion dysregulation, parenting practices and the white matter integrity of the emotion regulation networks in children. Firstly, the literature review section will focus on the neurobiology of ER, delineating the brain regions implicated in the ER processing. Then, it will move on to describe the theoretical model, and existing empirical evidence supporting the pathways within the model. ER encompasses ED to describe the approach that an individual uses to regulate their emotional experience. ER can be categorized as adaptive or maladaptive, when the regulation becomes maladaptive, such as impulsivity, lack of clarity and acceptance, and inability to engage in other behaviors in the presence of negative emotions, the maladaptive emotion regulatory behaviors are called ED.

The neurobiology of ER

ER engages both the cortical and subcortical regions of the brain to form various pathways of the ER network. The subcortical regions of ER are structurally connected with the cortical regions, thereby influencing each other's functions (Beauchaine & Cicchetti, 2019). Past literature has identified several brain regions as key nodes involved in emotional regulation. These brain regions include 1. the amygdala, 2. the prefrontal cortex, 3. the hippocampus, and 4. the hypothalamic-pituitary-adrenocortical system (HPA axis) (LeDoux & Phelps, 2008; Davidson et al., 2000). We will briefly summarize the function and research findings of each key region.

The Amygdala

The amygdala area is an almond-shaped structure located subcortically in the ventral region of the temporal lobe within both hemispheres. The amygdala plays an important role in emotional processing. A foundational animal study shows that lesions in the amygdala region in rhesus monkeys lessened or erased previously established fear associations (Weiskrantz, 1956). Damage to the amygdala can lead to impairments in the perception of emotionally salient events (Anderson & Phelps, 2001). Based on previous findings, the amygdala is shown to be responsible for threat detection, initiation of defense strategies, and identification of the motivational value of a given stimulus (LeDoux & Phelps, 2008; Costafreda et al., 2008). Scientists believe that the amygdala is the center for detecting the emotional significance of a stimulus. This information is then sent to other subcortical structures to facilitate decision-making (McLaughlin et al., 2019). The amygdala is monitored and regulated by the medial prefrontal cortex (mPFC), which either facilitates emotion attenuation or amplification of fear responses. This fronto-amygdala circuit is thus thought to play a critical role in emotion modulation (McLaughlin et al., 2019).

The Prefrontal Cortex

The central relevance of the PFC in emotion processing and regulation has been shown by decades of animal research and human subject studies. Functionally connected to the limbic system, the executive attention network shows activation in response to the perception of basic emotions as well as the reappraisal and suppression of emotional states (Banks et al., 2007). Specifically, the dorsolateral prefrontal cortex (DLPFC), dorsal medial prefrontal cortex (DMPFC), ventrolateral prefrontal cortex (VLPFC),

orbitofrontal cortex (OFC), and anterior cingulate cortex (ACC) are engaged (Banks et al., 2007). Triggered by one's active self-regulation, the engagement of these frontal regions is significantly associated with amygdala activities (Urry et al., 2006). Studies of psychopathologies also converge on the involvement of the fronto-amygdala circuits in ER by revealing the abnormal neural responses in DMPFC, DLPFC, ACC, and/or OFC responding emotional stimuli during cognitive-emotional tasks compared with those who do not have mental disorders (Lanius et al., 2004; New et al., 2007).

During childhood, the development of cognitive strategies and reliance on the executive attention network allows children to acquire ER skills such as emotion identification and effortful control, making them more effective in managing negative and positive emotional responses (Kerr et al., 2019). For adolescents, cognitive reappraisal is considered an important skill used to reduce negative emotions (Kerr et al., 2019). Research has illustrated PFC's involvement during cognitive reappraisal when subjects undergo stress-inducing tasks (Guyer et al., 2016). The increase in socialization, emotionality, and the more refined emotional categories and experiences are accompanied by changes in PFC, ACC, insula, striatum, and amygdala (Guyer et al., 2016).

The Hippocampus

The hippocampus is a subcortical structure that locates deep in the temporal lobe, playing a crucial role in forming episodic memory and learning through long-term potentiation of sensory information across various domains (Dhikav & Anand, 2012). Human lesion studies demonstrated a subject's inability to encode new episodic memory

after hippocampus removal (Penfield & Milner, 1958). The fronto-hippocampal circuit underlies memory consolidation and retrieval of stored information (Euston et al., 2012). Additionally, the connectivity between the amygdala and hippocampus creates an interaction between emotions and memory. Past research has illustrated Amygdala's engagement with memory formation and memory consolidation. Hippocampus is also involved in the regulation of the HPA axis and the termination of its stress response (Jacobson & Sapolsky, 1991). Animal studies showed that that adversity in the rearing environment would lead to a reduction in neuronal dendrites, leading scientists to investigate the influence of childhood maltreatment on hippocampal structure and functioning (Ivy et al., 2010).

The HPA Axis

The HPA axis contains the hypothalamus, the pituitary gland, and the adrenal gland. The HPA axis is the central stress response system. The stress response is characterized by the release of corticotropin-releasing factor (CRF) from the hypothalamus (Smith & Vale, 2006). CRF is a neuropeptide that is a major regulator of the HPA system. Coupled with arginine vasopressin (AVP), CRF is released into the hypothalamo-hypophyseal portal system and binds to the anterior pituitary gland, stimulating the secretion of adrenocorticotropin (ACTH) (Smith & Vale, 2006). CRF triggers a downward cascade of events where ATCH, in turn, stimulates the release of glucocorticoids (cortisol) by binding to the adrenal cortex in response to stress (Smith & Vale, 2006). Hyperactivity and hypoactivity of the HPA axis are often observed in psychopathology. Differences in emotional reactivity tend to be associated with

individual differences in HPA responses, with internalizing symptoms related to greater HPA reactivity, proving HPA's relevance in ER and processing. The dysregulation of the HPA axis can lead to functional and organizational changes in multiple areas of the brain, manifesting in maladaptive behaviors in humans (Stansbury & Gunnar, 1994).

Family influence on Children's ER Development

In recent decades, emotion dysregulation (ED) has been recognized as an important construct for understanding various adaptation problems on the developmental trajectory (Beauchaine & Cicchetti, 2019). According to its functional definition, emotion becomes dysregulated when it prevents one from engaging in meaningful and goal-directed behaviors. A growing body of research has illustrated that early ED may precede later risk of developing psychopathology, suggesting that the development of psychopathology may stem from ED (Zeman et al., 2006; Buckholdt et al., 2014; Beauchaine & Cicchetti, 2019; Aldao et al., 2014). Deficits in children's ability to regulate emotion are shown to be associated with a range of developmental indicators such as internalizing and externalizing problems, and poor social competence (Zalewski et al., 2011). Additionally, it has been observed that children who have distinct forms of psychological difficulties often demonstrate unique patterns of ED (Zeman et al., 2006). Specifically, children who reported more depressive symptoms are more likely to experience intense and volatile emotions, denial, or rumination of negative emotions and maladaptive ER (Silk et al., 2003). Depressed children are also less likely to endorse problem-focused and active distraction strategies and exhibit lower self-efficacy in their ability to successfully regulate their emotions (Garber et al., 1995). Similarly, children

who are diagnosed with anxiety disorders experience more intense and frequent negative emotions, more avoidant and less problem-solving strategies, and fewer abilities in the reappraisal of external stimuli and self-efficacy in ER (Carthy et al., 2010).

The emergence of ER occurs as early as infancy and continues to develop from infancy to adolescence (Calkin & Hill, 2007; Ostlund et al., 2019). Differential emotions theory argues that infants express universal facial expressions that are indicative of their basic emotional states such as anger, fear, and joy as adaptive tools to signal certain needs and discomfort (Zeman et al., 2006). From infancy (3-14 months) to toddlerhood (1-2 years), children acquire abilities to differentiate and label emotional states by recognizing and imitating the facial expressions of their caregivers, other adults, and peers (Pollak & Sinha, 2002). During this time, the regulation of emotion in children primarily relies on the caregivers through caregivers' use of pacifying behaviors (Zeman et al., 2006). Infants of mothers with depression who have emotional withdrawal tend to be less emotionally expressive and less responsive to faces and voices (Field et al., 2009; Lundy et al., 1996). Emotional socialization also begins in infancy. Infants often learn the context-appropriate emotional reactions through observing and receiving adults' responses and emotional expressions as a social reference to guide their behavioral responses in novel situations (Malatesta et al, 1987).

The development of more refined emotional experience and social rules for emotional expressivity continue throughout early childhood. Self-conscious emotions such as shame, pride, and embarrassment occur in toddlerhood. The skills to differentiate and label different emotional experiences will continue to develop from toddlerhood to early elementary age (Zeman et al., 2006). During early elementary age, children will

have an increase in understanding and display of emotional expressions that are consistent with the cultural-specific expectations in a given context (Gnepp & Heiss, 1986). This form of emotion regulation implies a separation between the display of one's emotions and one's true emotion experience either through behavioral amplification or inhibition. Elementary-age children gradually gain an understanding of such an implication to guide their expression and regulation of emotions (Harris et al., 1986). This ability continues to develop throughout middle childhood. By adolescence, children's ability to regulate their emotions becomes more differentiated according to the social contexts, the types of emotion, and the motivation behind the expressions (Zeman & Garber, 1996).

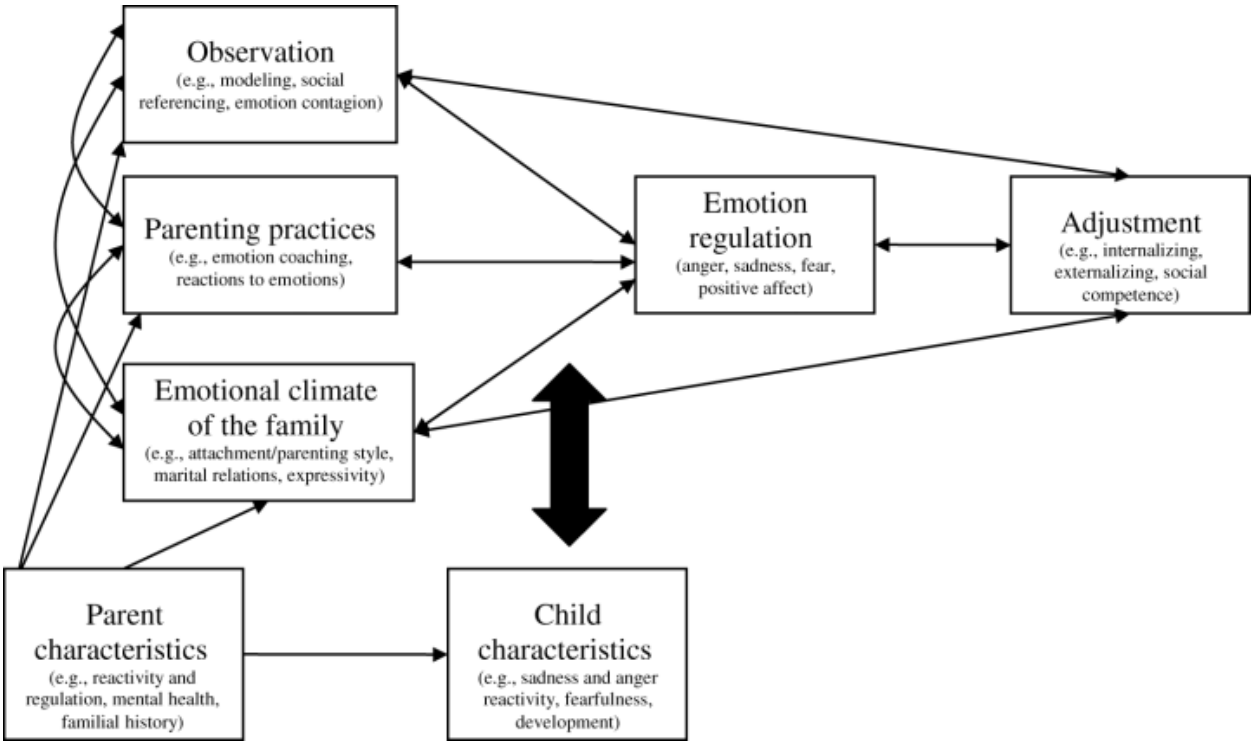

Figure 1. Tripartite Model of the Impact of Family on Children's Emotion Regulation (Morris et al., 2007)

The development of ER in children is constantly shaped by the interactions that they have with their environment. Parents seem to play an important role in shaping the ER development in children. According to the tripartite model (see Figure 1.) proposed by Morris and colleagues (2007), parents exert their influence on children's ER development through 1. Observational learning and modeling, 2. Parenting practices and 3. Family emotional climate. Specifically, they hypothesized that the parent's ER strategies, history of psychopathology, and belief in emotion expressivity will influence how they model ER, their parenting behaviors, and the overall emotional climate of the family (Morris et al., 2007). They also posit a family system view where family and children's ER mutually influence each other throughout the development trajectory (Morris et al., 2007).

The first pathway where parents can influence their children's ER is through observational learning and modeling. Specifically, observations of parents' emotional displays and expressivity can influence the ER development in children as modeling has long been proposed as a learning mechanism of various behaviors in social contexts (Bandura, 1977). In novel situations that are emotion-provoking, children often refer to others' reactions such as parents or peers, to learn how they 'should' react in similar situations (Emde et al., 1991). The free expressions of various emotions in parents can also help children to learn the range of emotional reactions and appropriate displays of emotions in different situations (Denham et al., 1997). However, parents with emotion dysregulation are less likely to model appropriate emotion regulation strategies; Children of mothers with depression are found to have fewer types of emotions expressed and

limited demonstration of ER strategies compared with children of mothers who have never been depressed (Silk et al., 2006).

The second pathway where parents can influence their children's ER is through parenting practices, which can also be impacted by the parents' ER styles and strategies. Conceptually, parental ER and parenting behaviors are two distinct but closely related constructs. The presence of parental ED will likely impede parents' ability to demonstrate adaptive parenting behaviors because it would inhibit a mother's ability to focus and engage their children's needs effectively (Han & Shaffer, 2013). Due to the regulatory nature of most parenting tasks, it is arguable that adaptive self-regulation is fundamental for parents to attend to the regulation of others. Various theoretical frameworks elucidating predictors of parent-child interaction have all highlighted the significant role of maternal ER in contributing to different parenting behaviors (Gottman et al., 1996; Eisenberg et al., 1998; Morris et al., 2007). Thus, understanding parental characteristics that contribute to parenting behaviors becomes important for preventive interventions that target parental mental health and bolster adaptive child development.

There is an abundance of empirical evidence showing that a parent's ability to regulate their emotions has strong associations with their parenting behaviors. Buckholdt and colleagues (2014) revealed a positive association between parental emotion dysregulation and their invalidation of their children's emotional reactions. Lorber and colleagues (2012) distinguished between the varied associations of cognitive reappraisal with positive parenting and suppression with harsh and ineffective disciplinary behaviors in mothers. Additional research found that maternal ED is associated with inattentiveness to adolescents' negative emotions, harsh parenting, and low level of adaptive dyadic

interactions (Jones et al., 2014; Mazursky-Horowitz et al., 2014; Shaffer & Obradović, 2017). These data suggest that a mother who experiences emotion dysregulation may be more likely to criticize their child's emotional expression, teaching the child maladaptive emotion regulation strategies, supporting the hypothesis that stable and positive parenting behavior requires the mother's adaptive ER strategies to effectively interact with children in a supportive manner.

Parenting practices are shown to impact the development of emotion regulation in children (Gottman et al., 1996; Morris et al., 2007). The tripartite model emphasizes the significance of parenting in shaping the ER development of children, including parental attention and reactions to children's emotions, validation of children's emotions, teaching and encouraging specific emotional regulation strategies, and coaching of emotion management. Positive parenting practices such as emotional coaching can facilitate the learning of positive ER strategies in the context of negative emotions such as anger and/or sadness (Criss et al., 2016; Gottman et al., 1996). Other positive parenting behaviors such as maternal warmth, acceptance, and responsiveness are associated with children's adaptive behavioral regulation, support-seeking ER strategies, and low level of observed negative emotions (von Suchodoletz et al., 2011; Fabes et al., 1994). Maternal support was found to be associated with children's perception that using ER strategies is more appropriate, and a wider range of ER strategies in children (Hardy et al., 1993). On the other hand, negative parenting practices such as hostility, psychological control, and inattentiveness are associated with maladaptive ER in children. For instance, corporal punishment is associated with an increase in children's anxiety and aggression (Lansford et al., 2014; Kliewer et al., 1996). Emotion invalidation is shown to be associated with

children's expression difficulty in their emotion management (Eisenberg et al., 1996). Morris and colleagues (2002) found that child report of maternal hostility was associated with ED in children. Lastly, harsh parenting from mothers is associated with ED in children whereas harsh parenting from fathers is associated with child aggression (Chang et al., 2003).

The last element proposed by Morris's Tripartite model is the emotional climate of the family, which is largely determined by the parental characteristics (e.g. ER strategies) and relationship qualities (such as attachment and marriage qualities). The frequency of positive and negative emotions expressed in the family is related to emotion regulation in parents (Morris et al., 2007). Specifically, mothers who report less emotion dysregulation are more likely to foster positive family expressiveness in their home environment, which in turn relates to the use of adaptive emotion regulation strategies in children (Are & Shaffer, 2016). Children who are exposed to a negative emotional climate daily are at high risk of becoming emotionally reactive (Morris et al., 2007). Additionally, recent research has indicated that maternal emotion regulation may serve as a greater influence on child emotion regulation development as compared to fathers (Bariola et al., 2011; Silva et al., 2018). This may be explained by children spending more time with their mothers, who often take the primary caregiver role. Research by Silva and colleagues (2018) further demonstrated that this relationship persists into later adolescence and is dependent on the quality of the adolescent-mother relationship.

Adverse interactions between parents and children can serve as a chronic stressor, activating the stress response system (hypothalamus-pituitary-adrenal, or HPA axis) in children; chronic HPA activation adversely affects neuroplasticity, particularly for stress-

sensitive brain structures (Lupien et al., 2009). The activation of the HPA axis triggers the production of glucocorticoids, a steroid hormone that regulates stress responses (Myers et al., 2014). The glucocorticoids exert their influence on our brain by binding to the mineralocorticoid (MRs) glucocorticoid receptors (GRs) in neurons and/or glia. MR is thought to indicate the resting level of glucocorticoid whereas GR is thought to be particularly important in signally stress-level glucocorticoid release (Myers et al., 2014). GR is present throughout the brain, including the limbic system and the PFC, which are implicated in the mediation of stress responses through the paraventricular nucleus of the hypothalamus projecting neurons to the HPA axis (Viho et al., 2019). In animal models, the development of dendrites and axons in the PFC and limbic system is shown to be altered by chronic HPA axis activation related to stress. Both reduction in dendritic complexity and reduced myelination in white matter tracts that connect emotion regulatory network regions have been observed in rodents exposed to various forms of early life stress (Helmeke et al., 2009; Liu et al., 2012; Muhammad & Kolb et al., 2011).

Both animal and human research confirm the role of childhood maltreatment in shaping the dendritic and axonal development through interacting with the HPA axis. However, the extant literature on stress and white matter structure has mostly focused on abuse and deprivation as the extreme form of parenting behaviors. Reduced fractional anisotropy (FA), thought to be an index of white matter microstructural integrity, has been found in the UF, the SLF, the corona radiata, the fornix, the CC, arcuate fasciculus, and the IFOF of individuals who experienced childhood maltreatment, suggesting compromised white matter integrity caused by chronic stress (Bick et al., 2015; Choi et al., 2009; Govindan et al., 2010; Hanson et al., 2013; Huang et al., 2012; McCarthy-Jones

et al., 2018). Outside of the maltreatment contexts, parenting practices have been found to moderate the association between reduced FA values in the white matter around the left thalamus, the right ACC, and the right superior frontal gyrus and high cortisol reactivity to stress in girls (Sheikh et al., 2014). Specifically, the association between FA values in white matters surrounding the right anterior cingulate cortex and superior frontal gyrus and high cortisol reactivity is moderated by positive parental affectivity during parent-child interactions, suggesting that positive parenting styles that demonstrate affectivity may act as a buffer to moderate the impact of stress on the structural development of white matter underlying emotion regulation in children (Sheikh et al., 2014).

In our study, the disruption in white matter microstructure was measured by FA, mean diffusivity (MD), and radial diffusivity (RD). FA is a measure of water diffusion with directional restriction due to axonal myelination; higher FA values suggest more restricted water diffusion along axonal tracts, indicating axonal integrity (Kochunov et al., 2012). MD reflects the average of water molecule mobility by calculating the mean of the three eigenvalues of the diffusion tensor. RD describes water diffusivity perpendicular to the axons by taking the average of second and third eigenvalues (Salat, 2014). Higher MD and RD may suggest demyelination and dysmyelination (Abdel-Aziz & Ciccarelli, 2014; Caeyenberghs & Swinnen, 2015).

There are different pathways through which emotional dysregulation in parents may influence the development of emotion regulation in children, however, no studies have examined associations between emotion regulation in parents, specifically mothers, and indices of white matter microstructure in their children. We used deterministic

tractography to reconstruct pathways that have been repeatedly implicated in basic emotion regulation processes and studies of early adversity, including the UF, CB, SLF, ILF, fornix, and CC. We examined indices of white matter microstructure in these pathways in children, including fractional anisotropy (FA), mean diffusivity (MD), and radial diffusivity (RD), examining relationships between these assays and maternal emotion regulation. We hypothesize lower FA, higher MD, and RD of the abovementioned pathways will be associated with more severe maternal ED. Secondarily, we examined parenting style and its associations with WM. We hypothesize that lower FA, higher MD, and RD of the relevant pathways will be associated with more negative parenting behaviors such as demandingness and corporal punishment, while enhanced higher FA, lower MD, and RD will be associated with more positive parenting behavior such as warmth. Finally, we explored associations of children's WM indices with child emotion regulation, focusing specifically on negative emotions including anger, sadness, and worry. We hypothesize that lower FA, higher MD, and RD in relevant pathways will be associated with a higher level of negative emotion dysregulation among children.

Chapter III. Methodology

Procedure

The current study drew participants recruited from a larger longitudinal NIH-funded study on the transmission of intergenerational trauma and Post-Traumatic-Stress-Disorder (PTSD) in a sample of low-income African American mother-child dyads (Jovanovic et al., 2011; Kamkwalala et al., 2012). This study is a secondary analysis of the originally collected data using only cross-sectional data. All study procedures were approved by the institutional review board of Emory University, School of Medicine, and Grady Memorial Hospital. For the main study, participant recruitment took place in the waiting rooms of a Pediatric hospital, Primary Care, or Obstetrics Gynecology of a publicly funded hospital in Atlanta, GA.

After consenting, mothers completed an interview with a trained research assistant to determine eligibility. Inclusion criteria for mothers were to be of 18-65 years of age, identify as African American/Black, be able to provide consent, and be the primary caretaker of an 8 to 12-year-old child. Child inclusion criteria were to be of 8-12 years of age and willing to provide assent. Exclusion criteria for mothers and children included active psychotic disorder, cognitive disability, and a diagnosis of autism spectrum disorders.

Prior to participating in the study, all mothers provided informed consent and parental permission for children to participate. Assent from child participants was also obtained prior to participating in the initial study visit. Once participants consented to join the study, mother-child dyads were invited to complete individual separate clinical interviews and self-report measures to assess their trauma history, PTSD, emotion

regulation, and parenting skills. Additionally, child participants completed an MRI scanning session which took place at the Facility for Education and Research in Neuroscience at Emory University.

Participants

Participants for this study were 45 African American mother-child dyads. The average age for the mother was 37.11 (SD=8.86, range=26-59); the average age for child participants was 10.07 (SD=9.76, range=8-13) with the majority of children identifying as female (52%). During recruitment, participants were approached by research assistants in the waiting rooms at the Grady Hospital. Participants were asked if they would be interested in participating in this study. Several screening questions were asked during the conversation to ensure the participants eligibility. If the participants were interested in the study, they were asked to provide contact information such as phone number, email address for further contact.

Measures

Difficulties in Emotion Regulation Scale

Maternal ED was measured with the Difficulties in emotion Regulation Scale (DERS) (Gratz & Roemer, 2004). DERS is a well-validated (Cronbach's α=0.95) psychometric scale that contains 36 items (Mekawi et al., 2021). Six subscales were included, 1. non-acceptance of negative affect, 2. difficulty controlling impulses in the presence of negative affect, 3. difficulty engaging in goal-directed behavior in the presence of negative emotions, 4. lack of adaptive emotion regulation skills 5. Lack of emotional clarity, 6. lack of emotional awareness. One item states: "When I'm upset, I

become angry with myself for feeling that way". Responses are in the form of a 5-point Likert scale that ranges from "almost never" to "almost always" based on the statements. Scores were summed up with a higher score indicating a higher level of emotion dysregulation, with scores ranging from 36 to 180. Total scores can also be broken down into 6 different subscales, with scores ranging from 6 to 30.

Parenting Questionnaire

The Parenting Questionnaire (PQ) is a 50-item self-report measure of negative and positive parenting practices including warmth, demandingness, and corporal punishment as three subscales (Powers et al., 2021). The Cronbach's α for our sample is, 0.87 for warmth, 0.71 for demandingness, and 0.74 for corporal punishment. A sample item includes "I expect my child to obey me without questioning me". Responses to the items are on a 5-point Likert scale ranging from 1 (almost never or disagree) to 5 (very often or strongly agree). The score of each subscale is the sum of the responses to all the questions within the subscale. Higher scores represent a higher level of warmth, demandingness, or corporal punishment. The score range for warmth is 22-110, for demandingness is 22-110, and for corporal punishment is 4-20.

The Children's Emotion Management Scale (CEMS)

Child emotion regulation was measured using the Children's Emotion Management Scale (CEMS) is a 33-item self-report measure that has been well-validated (Zeman et al., 2001; Zeman et al., 2010). The CEMS is administered to both parents (CEMS PR) and children (CEMS CR) and includes three primary subscales for specific

emotions: inhibition (i.e., emotion suppression), dysregulation (i.e., inappropriate emotional expression), and coping scales (i.e., adaptive emotion coping skills). Each subscale includes scores for specific emotions including anger, sadness, and worry emotions. CEMS PR and CEMS CR were used separately to reflect emotion dysregulation in children. The Cronbach's alpha for all the subscales ranges from 0.61 to 0.80, demonstrating an adequate general internal consistency (Zeman et al., 2010). Participants respond to the CEMS using a 3-point Likert scale ranging from 1 (*hardly ever*) to 3 (*often*). A sample item includes: "My child does things like slam doors when he/she is mad". Scores were summed with a higher score indicating a higher level of emotion dysregulation in children (Zeman et al., 2001). The score for inhibition subscales ranges from 4 to 12, for dysregulation subscales ranges from 3 to 9.

For this study, only child dysregulation and inhibition scores for anger sadness, and worry were examined. Specifically, our rationale for excluding the coping scale was that it measured a different construct that was outside the scope of this study. Coping has been referred to as the process to manage intense and long-lasting emotions, reflecting the adaptive nature of managing negative emotions. This study aims to examine associations between mother-child emotion dysregulation, focusing on the maladaptive elements of ER. Additionally, there was no direct assessment of maternal emotional coping to map onto child coping skills.

Diffusion Tensor Imaging (DTI) procedures and analyses

The neuroimaging data of the children were collected using a machine called magnetic resonance imaging. A mock scan was conducted before the actual scan to help

children better acclimate to the scanner. Scanning took place on a research-dedicated Siemens 3-Tesla TIM-Trio scanner using a 32-channel head coil. Diffusion-weighted images were acquired in two different phase-encoding directions with the following parameters: 66 x 2.0mm thick axial slices, matrix=106 x 106, field of view=212 x 180mm, voxel size=2 x 2 x 2mm, TR=3292 ms, TE=96 ms. The diffusion weighting was isotopically distributed along 138 directions using a b-value of 1000s/mm^2. For each scan, six normalization images, with no diffusion encoding (b=0), were acquired and averaged for each direction using linear rigid-body registration (FLIRT; Jenkinson and Smith, 2001). All image processing and analysis were conducted using FMRIB Software Library (FSL version 4.1; www.fmrib.ax.ac.uk/fsl; Smith et al, 2004). Data preprocessing and correction for head motion and eddy current distortion was conducted with TOPUP and EDDY toolkits in FSL (www.fsl.fmrib.ox.ac.uk/fsl). Normalization images were skull-stripped using the FSL brain extraction tool (Smith, 2002). Data from three participants were excluded due to motion artifacts or image distortion.

Deterministic Tractography was performed using DSI Studio's graphical user interface (http://dsi-studio.labsolver.org). The NIFTI files were processed by DSI Studio in batch by applying automated masks to generate 3D DTI volumes. Once the volumes were generated, three DTI indices, the FA, MD, and RD values were extracted for the (UF; right and left), (CC), (SLF; three segments I, II, III, right and left), fornix and cingulum bundle (CB; right and left) using the software's automated system. Example pathways for these tracts are provided in Appendix, Figure 2.

Statistical Analysis Methodology

All analyses were performed using SPSS 26 software. Our primary objective was to examine associations of maternal ED with child WM connectivity in our tracts of interest, examining each hemisphere for associational tracts (SLF, ILF, CB, UF, fornix) and the single commissural tract (CC). First, we examined correlations between DERS total score and indices of children's white matter structural connectivity (FA, MD, and RD) with maternal ED (DERS total and subscale scores). A Bonferroni-corrected statistical threshold of $p<.0033$ (for 15 tracts) was used to define significance for each family of tests. Significant findings were subject to more granular analyses; DERS subscales and tract segments (e.g., fornix) were included in post-hoc analyses to examine specific associations of maternal emotion dysregulation and connectivity of WM pathway sections. Secondarily, we examined associations between parenting style (Parenting Questionnaire) and WM indices. Finally, we examined associations between these indices and child emotion dysregulation, focusing on negative emotions (CEMS anger, worry, sadness subscales). Additionally, the relationships among the measures such as the correlations between CEMS and DERS, and DERS with PQ were assessed using correlational analyses to provide an overview of the results.

Chapter IV. Results

For our 45 mother-child dyads, approximately half of the mothers reported their marital status as single (54.5%). Maternal participant education was distributed as follows, 18.2 % >12th grade, 29.5% high school graduate or equivalent, 22.7% some college or technical school, 11.4% technical school graduate, 13.6% college graduate, 4.5% completed a graduate degree. Most maternal participants reported being employed at the time of data collection (62.2%), and most (52.3%) reported a household monthly income of $999 or above. Further detailed demographic and clinical characteristics of mothers and their children are provided in Table 1.

Maternal Emotion Dysregulation Associations with Child White Matter Connectivity Indices

The mean for DERS total among mothers is 72.77 (sd=24.66), 10.98 (sd=5.10) for non-acceptance, 12.30 (sd=4.82) for difficulty engaging in goal-directed behaviors, 11.84 (sd=5.43) for impulsivity, 13.25 (sd=5.26) for awareness, 14.74 (sd=6.18) for emotion regulatory strategies, and 9.65 (sd=4.45) for emotional clarity. The results of correlation analyses among the variables are included in Table 2, significant correlations are shown in figure 3. Consistent with our hypothesis, ED (DERS total) was positively associated with the MD (r=0.48, p=0.001) and RD (r=0.46, p=0.002) of the right fornix. DERS inability to engage in goal-directed behavior in the presence of negative emotions subscale (DERS GOAL) was associated with MD of the right fornix (r=0.45, p=0.003). DERS Impulsivity subscale was associated with MD (r=0.51, p<0.001) and RD (r=0.51, p=0.001) of the right fornix. No significant associations were observed between DERS and FA of the white matter tracts.

Parenting Questionnaire Associations with Child White Matter Connectivity Indices

The mean for parental warmth is 89.26 (sd=9.99), 74.79 (sd=10.46), 9.58 (sd=2.84) for corporal punishment. For the parenting practices, none of the associations was statistically significant after the Bonferroni correction of the p-value. For results that approach our threshold, parental demandingness was associated with FA of the left fornix (r=-0.37, p=0.015). Corporal punishment was associated with RD (r=0.32, p=0.037) of the right CB.

Child Emotion Dysregulation Associations with Child White Matter Connectivity Indices

The mean for CEMS PR anger inhibition is 5.67 (sd=1.90), 5.27 (sd=1.60) for anger dysregulation, 7.31 (sd=2.42) for sadness inhibition, 6.04 (sd=1.68) for sadness dysregulation, 6.27 (sd=1.81) for worry inhibition, and 4.40 (sd=1.44) for worry dysregulation. The mean for CEMS CR anger inhibition is 7.36 (sd=2.32), 6.13 (sd=1.97) for anger dysregulation, 8.13 (sd=2.38) for sadness inhibition, 4.93 (sd=1.54) for sadness dysregulation, 8.49 (sd=1.90) for worry inhibition and 4.47 (sd=1.50) for worry dysregulation.

The mean for CEMS PR is 56.11 (sd=9.03), and 58.78 (sd=7.29) for CEMS CR. The results of correlation analyses among the variables are included in Table 3, significant associations are shown in Figure 4. Consistent with our hypothesis, parent-report sadness inhibition (CEMS sadness inhibition) is associated with the MD (r=-0.44, p=0.003) of the CB of the right hemisphere. However, contrary to our hypothesis, CEMS cr anger inhibition subscale is significantly associated with the RD of the right hemisphere SLF III (r=0.44, p=0.003). The analysis results of the correlations of CEMS with DERS, and CEMS with PQ can be found in Table 4. and Table 5.

Chapter V. Conclusions

The current study is to our knowledge, the first to investigate the associations between maternal ED with the white matter microstructure in their children. We used deterministic tractography to reconstruct the pathways that have been implicated in emotion regulation processes and studies of early life stress. Lower FA, Higher MD and RD values indicate disruptions in the microstructural integrity of the white matter. This disruption may be caused by demyelination, dysmyelination and reduced dendritic complexity. Our primary results indicate associations between maternal emotion dysregulation and white matter microstructure disruption of the fornix of the right hemisphere. Specifically, overall emotion dysregulation, difficulty engaging in goal-driven behaviors in the presence of negative emotions, and impulsivity in mothers demonstrated the strongest associations with MD and RD indices of the right fornix in their children.

Our findings on maternal ED and white matter connectivity of the right fornix in children are consistent with previous literature implicating the fornix as part of the emotion regulation network. The fornix, emerging as the fimbria-fornix, is the major output fiber tract projecting from the hippocampus to the anterior part of the brain. After reaching the anterior commissure, it projects downward, connecting to various regions such as the hypothalamus, thalamus, ventral striatum, nucleus accumbens, and the cingulate cortex (Kazlouski et al., 2011). As part of the Papez Circuit in the limbic system, the fornix is engaged in ER, reward learning, and memory (Dalgleish, 2004). Monkeys with fornix lesions demonstrated the inability to pair spatial locations with

reward (Gaffan et al., 1984). Lesions of the fornix in rodents also resulted in disruptions of the contextual fear conditioning (Phillips & LeDoux, 1995). Thus, disrupted white matter microstructure of the fornix in children could indicate an altered reward learning process due to exposure to maternal ED. Parents are a major source of reward learning during the child's growth. The reward such as praises, gifts, and positive interactions, or punishment such as criticism or disapproval that children receive from their parents can influence the possibility of children engaging in similar behavior in the future. ED in a mother can prevent her from properly responding to her child's behavior. In our results, the ED subscales assessing the inability to engage in goal-driven behaviors and impulsivity demonstrated the strongest associations with the disrupted white matter microstructure of the fornix. These results may suggest that in the presence of negative emotions, a mother can fail to pay attention to her child or react negatively to behaviors that the child is expecting a reward. This inconsistency in reactions to the child's behavior can create confusion, stress, and frustration in a child. The inconsistent and negative experience that a child had during interaction with their mothers can chronically activate the HPA axis, gradually altering the structure of the fornix. Additionally, the lack of consistent reward learning experience can also create difficulties when trying to retrieve relevant memory, further implicating the fornix in the process.

ED in mothers could be reflected through the parenting practices that they adopt when interacting with their children. Our results found that higher level of ED in mothers is associated with lower level of warm, and high level of demandingness and corporal punishment (Table 5.). However, for our second analysis, no significant results were found between parenting practices and white matter structural indices. The lack of

significance between parenting and white matter indices may suggest that based on our Tripartite model, the emotional learning and modeling, the direct influence of maternal characteristics to the child as well as the overall family climate as more relevant in the process where parents influence the development of emotion regulation neural networks in their children. Additionally, the lack of significant findings could also be due to the problems of the measure. PQ was originally created and validated in a sample of middle-class mothers who were predominantly white. Using PQ to assess parenting practices in our sample may not account for the cultural background of the population, limiting the validity of the measure.

Our third analysis explores associations of ED in children (with the CEMS anger, worry, sadness subscales) and their white matter structural connectivity. The parent-report sadness inhibition in children is negatively associated with MD of the right CB, whereas the child-report anger inhibition is negatively associated with RD of the right SLF III. CB links the frontal, parietal, and medial temporal regions, also connecting the cingulate gyrus with the limbic system (Versace et al., 2015). The CB is engaged in various brain functioning such as emotional processing and regulation, executive control, and episodic memory (Bubb et al., 2018). Abnormal CB has also been found in various psychopathologies such as depression, obsessive-compulsive disorder, post-traumatic stress disorder, and schizophrenia (Bubb et al., 2018). SLF III interconnects the PFC, the ventral premotor area, and the supramarginal gyrus of the parietal lobe, implicated in language, motor control, attentional control, and spatial awareness (Nakajima et al., 2019). Findings suggest that a lower level of sadness inhibition is associated with reduced white matter integrity in these regions. This may indicate that effortful inhibition of

sadness requires top-down regulation of the emotional responses. Enhanced myelination of the white matter tracts can enhance the efficiency of the inhibition processes. However, there is a positive correlation between the SLF III and anger inhibition, indicating that a higher level of anger inhibition is associated with a higher level of reduced white matter integrity in the SLF III. The difference in the two results could reflect the qualitative differences between two emotions. Unlike anger, sadness is not a defensive response. It is more related to an individual's expressivity and introspection. Results could indicate that the two white matter tracts are involved in regulation of different emotions. Additionally, the development of the neural networks implicated in emotion regulation processing is more protracted in children, SLF is more involved in other cognitive functioning and may mature earlier than the CB. Therefore, SLF may suffer from white matter disruptions earlier than CB on the developmental trajectory.

Our findings are consistent with previous literature implicating the CB with pediatric ED (Bertocci et al., 2016; Hung et al., 2020; Versace et al., 2015). ED was found to be associated with increased RD and decreased FA in the cingulum callosal bundles among children 6 to 17 years old (Huang et al., 2020). In addition, axial diffusivity (AD) is another white matter index with a lower value reflecting higher axonal injury. Increases in AD were associated with the severity of manic symptoms among adolescents with ED (Versace et al., 2015). Among adolescents with ED, greater CB fiber length predicted lower scores on mania compared with controls (Bertocci et al., 2016). Lastly, all the significant findings are found in the right hemisphere. These findings may reflect the lateralization of emotion processing, particularly negative emotions in the right hemisphere (Zeman et al., 2021). Past studies have repeatedly

demonstrated the right hemispheric responsiveness to emotion processing (Adolphs et al., 1996; Schwartz et al., 1975; Borod et al., 1998; Ross & Monnot, 2008).

The differences in associations between parent-report and child-report pediatric ED can be due to differences in parent-perceived and self-perceived ED in children. It is possible that mothers who have ED symptoms are more likely to perceive the behavior of their children to be a sign of dysregulation, or that mothers with ED are more aware of the similarities between children's exhibition of ED with mothers' ED behaviors. Children, on the other hand, may perceive their behaviors as normal due to a lack of understanding of their emotions, or a skewed understanding of appropriate emotional behaviors due to long-term exposure to maternal ED. The stronger associations of DERS with CEMS pr subscales compared with CEMS cr subscales further support this hypothesis.

The current study also has its strengths and limitations. Our study focuses on a low-income minority population that has been historically overlooked in research. Our findings can provide insight on the unique challenges that African American Families often encounter when raising children. As for the limitations, firstly, due to the high plasticity of children's brains, the findings that we observed at a certain age might not be present in a different age range. Future studies could adopt a longitudinal design to see if these associations stay on the same path of development as the kids go into adolescence. Secondly, we only examined the white matter in children. Future studies should also assess the maternal white matter connectivity of relevant tracts to examine the similarities and differences of neural patterns between the mother and the child. Lastly, other environmental factors such as trauma, socioeconomic status, family structure were not

included in the analyses. Future studies should consider including above-mentioned factors as covariates due to the significant impact that the environment can have on the development of children.

Taken together, our study found that higher maternal ED was associated with weakened microstructural integrity in the right fornix. Pediatric ED, particularly higher sadness and anger inhibition are associated with reduced disruption of the white matter structure in SLF III and the CB. consistent with the Tripartite model, our finding suggests that the emotional learning and modeling, the direct influence of maternal characteristics to the child as well as the overall family climate could be more relevant in the process where parents influence the development of emotion regulation neural networks in their children. Our findings also suggest that disrupted white matter microstructure of certain tracts underlying emotion regulation processing may represent the developmental risk factors for psychopathologies characterized by ED as one of the core symptomatology, prompting future efforts to identify clinical biomarkers of ED and children who are at risk of developing mental health conditions. This work can also inspire the development of intervention and prevention strategies for pediatric psychological disorders as well as providing a tool for treatment monitoring and outcome evaluation.

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Appendix

Table 1. Participant Demographic Information	
Maternal Demographic Characteristics	
	Mean (SD) *N*= 45
Maternal Age	37.11 (8.86)
Relationship Status	**%**
Single	54.5
Married	9.1
Divorced	18.2
Separated	9.1
Widowed	4.5
Domestic Partner	4.5
Maternal Education	**%**
< 12th	18.2
Highschool Graduate/Equivalent	29.5
Some College/Technical School	22.7
Technical School Graduate	11.4
College Graduate	13.6

Graduate School	4.5
Maternal Employment	**%**
Employed	62.2
Unemployed	35.6
Household Monthly Income	**%**
$0 - 249	9.1
$250 - 499	9.1
$500 - 999	29.5
$1,000 - 1,999	29.5
$2,000 ≥	22.7
Difficulties in Emotion Regulation (DERS)	**Mean (SD) *N*= 45**
Total	72.77 (24.66)
Nonacceptance	10.98 (5.10)
Goals	12.30 (4.82)
Impulse	11.84 (5.43)
Awareness	13.25 (5.26)
Strategies	14.74 (6.18)

Clarity	9.65 (4.45)
Parenting Questionnaire (PQ)	**Mean (SD) *N*= 43**
Parental Warmth	89.26 (9.99)
Parental Demandingness	74.79 (10.46)
Parental Corporal Punishment	9.58 (2.84)
Child Demographic Characteristics	
	Mean (SD) *N*= 45
Child Age	10.07 (9.67)
Child Gender	
Female	52.3
Male	47.7
Child Emotion Management Scale	**Mean (SD) *N*= 45**
CEMS PR Subscales	
Anger Inhibition	5.67 (1.90)
Anger Dysregulation	5.27(1.60)
Sadness Inhibition	7.31 (2.42)
Sadness Dysregulation	6.04 (1.68)

Worry Inhibition	6.27 (1.81)
Worry Dysregulation	4.40 (1.44)
CEMS CR Subscales	
Anger Inhibition	7.36 (2.32)
Anger Dysregulation	6.13 (1.97)
Sadness Inhibition	8.13 (2.38)
Sadness Dysregulation	4.93 (1.54)
Worry Inhibition	8.49 (1.90)
Worry Dysregulation	4.47 (1.50)

Table 2 DERS associations with white matter tract indices

2a. DERS total associations with white matter tract indices

Correlations	DER Total (*r* value)
FA Right Cingulum	0.155
MD Right Cingulum	0.02
RD Right Cingulum	-0.077
FA Left Cingulum	0.182
MD Left Cingulum	-0.131
RD Left Cingulum	-0.185
FA Right Superior Longitudinal Fasciculus 1	0.052
MD Right Superior Longitudinal Fasciculus 1	0.025
RD Right Superior Longitudinal Fasciculus 1	0.011
FA Right Superior Longitudinal Fasciculus 2	0.226
MD Right Superior Longitudinal Fasciculus 2	-0.083
RD Right Superior Longitudinal Fasciculus 2	-0.142
FA Right Superior Longitudinal Fasciculus 3	0.19
MD Right Superior Longitudinal Fasciculus 3	0.098
RD Right Superior Longitudinal Fasciculus 3	-0.003
FA Left Superior Longitudinal Fasciculus 1	-0.09
MD Left Superior Longitudinal Fasciculus 1	0.121
RD Left Superior Longitudinal Fasciculus 1	0.154
FA Left Superior Longitudinal Fasciculus 2	-0.049
MD Left Superior Longitudinal Fasciculus 2	0.036
RD Left Superior Longitudinal Fasciculus 2	0.058
FA Left Superior Longitudinal Fasciculus 3	0.175
MD Left Superior Longitudinal Fasciculus 3	-0.034
RD Left Superior Longitudinal Fasciculus 3	-0.087

FA Right Inferior Longitudinal Fasciculus	-0.088
MD Right Inferior Longitudinal Fasciculus	-0.005
RD Right Inferior Longitudinal Fasciculus	0.044
FA Left Inferior Longitudinal Fasciculus	-0.145
MD Left Inferior Longitudinal Fasciculus	0.042
RD Left Inferior Longitudinal Fasciculus	0.141
FA Right Uncinate Fasciculus	0.077
MD Right Uncinate Fasciculus	-0.094
RD Right Uncinate Fasciculus	-0.087
FA Left Uncinate Fasciculus	-0.117
MD Left Uncinate Fasciculus	0.027
RD Left Uncinate Fasciculus	0.055
FA Right Fornix	-0.237
MD Right Fornix	.479***
RD Right Fornix	.455***
FA Left Fornix	0.084
MD Left Fornix	0.124
RD Left Fornix	0.079
FA Corpus Callosum	-0.042
MD Corpus Callosum	0.092
RD Corpus Callosum	0.095
****. Correlation is significant at the 0.0033 level (2-tailed).*	
***. Correlation is significant at the 0.01 level (2-tailed).*	
**. Correlation is significant at the 0.05 level (2-tailed).*	

2b. Maternal Emotion Dysregulation Subscales Associations with Child White Matter Connectivity Indices of the Right Fornix

	FA Right Fornix	MD Right Fornix	RD Right Fornix	DERS Nonacceptance	DERS Goals	DERS Impulse	DERS Awareness	DERS Strategies	DERS Clarity
FA Right Fornix	--								
MD Right Fornix	-.550**	--							
RD Right Fornix	-.688**	.982**	--						
DERS Nonacceptance	-0.143	0.288	0.275	--					
DERS Goals	-0.155	.446***	.407**	.600**	--				
DERS Impulse	-.337*	.514***	.506***	.676**	.741**	--			
DERS Awareness	-0.029	0.213	0.185	0.294	0.17	0.173	--		
DERS Strategies	-0.184	.412**	.388*	.711**	.729**	.871**	0.187	--	
DERS Clarity	-0.269	.366*	.368*	.610**	.415**	.563**	.529**	.559**	--
	***. *Correlation is significant at the 0.0033 level (2-tailed).*								
	**. *Correlation is significant at the 0.01 level (2-tailed).*								
	*. *Correlation is significant at the 0.05 level (2-tailed).*								

Table 3. CEMS Subscale Associations with Child White Matter Indices
3a. CEMS Child Report (CR) Anger Subscale Associations with Child White Matter Connectivity Indices

Correlations	CEMS CR Anger Inhibition (r value)	CEMS CR Anger Dysregulation (r value)
FA Right Cingulum	-0.09	-0.042
MD Right Cingulum	-0.001	0.041
RD Right Cingulum	0.057	0.06
FA Left Cingulum	0.058	-0.118
MD Left Cingulum	-0.038	0.097
RD Left Cingulum	-0.058	0.102
FA Right Superior Longitudinal Fasciculus 1	-.331*	0.241
MD Right Superior Longitudinal Fasciculus 1	0.11	-0.133
RD Right Superior Longitudinal Fasciculus 1	0.232	-0.22
FA Right Superior Longitudinal Fasciculus 2	-0.121	-0.067
MD Right Superior Longitudinal Fasciculus 2	0.268	0.053
RD Right Superior Longitudinal Fasciculus 2	0.233	0.063
FA Right Superior Longitudinal Fasciculus 3	-.361*	0.055
MD Right Superior Longitudinal Fasciculus 3	.391**	-0.094
RD Right Superior Longitudinal Fasciculus 3	.435***	-0.113
FA Left Superior Longitudinal Fasciculus 1	-0.251	0.071
MD Left Superior Longitudinal Fasciculus 1	0.057	0.144
RD Left Superior Longitudinal Fasciculus 1	0.178	0.028
FA Left Superior Longitudinal Fasciculus 2	-0.171	0.058
MD Left Superior Longitudinal Fasciculus 2	0.165	0.062
RD Left Superior Longitudinal Fasciculus 2	0.221	0.017
FA Left Superior Longitudinal Fasciculus 3	-0.165	0.093
MD Left Superior Longitudinal Fasciculus 3	0.166	0.048
RD Left Superior Longitudinal Fasciculus 3	0.201	-0.019
FA Right Inferior Longitudinal Fasciculus	0.127	-0.055
MD Right Inferior Longitudinal Fasciculus	-0.155	0.058
RD Right Inferior Longitudinal Fasciculus	-0.147	0.057
FA Left Inferior Longitudinal Fasciculus	-0.121	-0.006
MD Left Inferior Longitudinal Fasciculus	0.036	-0.039
RD Left Inferior Longitudinal Fasciculus	0.097	-0.023
FA Right Uncinate Fasciculus	0.083	-0.225

MD Right Uncinate Fasciculus	0.046	0.091
RD Right Uncinate Fasciculus	-0.012	0.15
FA Left Uncinate Fasciculus	-0.085	0.116
MD Left Uncinate Fasciculus	0.207	-0.011
RD Left Uncinate Fasciculus	0.172	-0.059
FA Right Fornix	-0.204	-0.088
MD Right Fornix	0.257	0.03
RD Right Fornix	0.257	0.038
FA Left Fornix	-0.003	-0.094
MD Left Fornix	0.228	0.182
RD Left Fornix	0.205	0.185
FA Corpus Callosum	0.064	-0.07
MD Corpus Callosum	-0.038	0.094
RD Corpus Callosum	-0.032	0.104
****. Correlation is significant at the 0.0033 level (2-tailed).*		
***. Correlation is significant at the 0.01 level (2-tailed).*		
**. Correlation is significant at the 0.05 level (2-tailed).*		

3b. CEMS CR Sadness Subscale Associations with Child White Matter Connectivity Indices

Correlations	CEMS CR Sadness Inhibition (*r* value)	CEMS CR Sadness Dysregulation (*r* value)
FA Right Cingulum	0.186	-0.039
MD Right Cingulum	0.013	-0.135
RD Right Cingulum	-0.126	-0.087
FA Left Cingulum	0.189	-0.032
MD Left Cingulum	-0.037	-0.121
RD Left Cingulum	-0.163	-0.071
FA Right Superior Longitudinal Fasciculus 1	0.122	0.066
MD Right Superior Longitudinal Fasciculus 1	-0.037	-0.236
RD Right Superior Longitudinal Fasciculus 1	-0.063	-0.218
FA Right Superior Longitudinal Fasciculus 2	0.06	0.051
MD Right Superior Longitudinal Fasciculus 2	0.054	-0.119
RD Right Superior Longitudinal Fasciculus 2	0.011	-0.092
FA Right Superior Longitudinal Fasciculus 3	0.178	-0.031
MD Right Superior Longitudinal Fasciculus 3	0.056	-0.105
RD Right Superior Longitudinal Fasciculus 3	-0.022	-0.07

FA Left Superior Longitudinal Fasciculus 1	-0.005	0.173
MD Left Superior Longitudinal Fasciculus 1	-0.054	-0.129
RD Left Superior Longitudinal Fasciculus 1	-0.032	-0.178
FA Left Superior Longitudinal Fasciculus 2	0.038	0.112
MD Left Superior Longitudinal Fasciculus 2	0.008	-0.276
RD Left Superior Longitudinal Fasciculus 2	0.02	-0.263
FA Left Superior Longitudinal Fasciculus 3	0.155	0.223
MD Left Superior Longitudinal Fasciculus 3	-0.069	-0.209
RD Left Superior Longitudinal Fasciculus 3	-0.103	-0.239
FA Right Inferior Longitudinal Fasciculus	0.185	.330*
MD Right Inferior Longitudinal Fasciculus	-0.097	-0.283
RD Right Inferior Longitudinal Fasciculus	-0.141	-.331*
FA Left Inferior Longitudinal Fasciculus	0.082	0.014
MD Left Inferior Longitudinal Fasciculus	-0.01	-0.205
RD Left Inferior Longitudinal Fasciculus	-0.043	-0.133
FA Right Uncinate Fasciculus	0.224	0.29
MD Right Uncinate Fasciculus	-.295*	-0.178
RD Right Uncinate Fasciculus	-0.294	-0.242
FA Left Uncinate Fasciculus	0.014	0.076
MD Left Uncinate Fasciculus	-0.002	-0.161
RD Left Uncinate Fasciculus	-0.022	-0.147
FA Right Fornix	0.038	0.03
MD Right Fornix	0.143	-0.057
RD Right Fornix	0.101	-0.062
FA Left Fornix	0.145	0.174
MD Left Fornix	0.161	0.03
RD Left Fornix	0.112	-0.013
FA Corpus Callosum	0.212	.310*
MD Corpus Callosum	-0.093	-.309*
RD Corpus Callosum	-0.137	-.322*
****. Correlation is significant at the 0.0033 level (2-tailed).*		
***. Correlation is significant at the 0.01 level (2-tailed).*		
**. Correlation is significant at the 0.05 level (2-tailed).*		

3c. CEMS CR Worry Subscale Associations with Child White Matter Connectivity Indices

Correlations	CEMS CR Worry Inhibition (*r* value)	CEMS CR Worry Dysregulation (*r* value)

FA Right Cingulum	0.060	-0.140
MD Right Cingulum	0.140	-0.043
RD Right Cingulum	0.015	0.036
FA Left Cingulum	0.254	-0.093
MD Left Cingulum	0.017	-0.095
RD Left Cingulum	-0.150	-0.013
FA Right Superior Longitudinal Fasciculus 1	-0.103	0.022
MD Right Superior Longitudinal Fasciculus 1	0.121	-0.264
RD Right Superior Longitudinal Fasciculus 1	0.128	-0.224
FA Right Superior Longitudinal Fasciculus 2	0.032	-0.091
MD Right Superior Longitudinal Fasciculus 2	0.062	-0.067
RD Right Superior Longitudinal Fasciculus 2	0.017	-0.015
FA Right Superior Longitudinal Fasciculus 3	-0.084	-0.018
MD Right Superior Longitudinal Fasciculus 3	0.270	-0.179
RD Right Superior Longitudinal Fasciculus 3	0.214	-0.103
FA Left Superior Longitudinal Fasciculus 1	0.050	0.070
MD Left Superior Longitudinal Fasciculus 1	0.010	-0.101
RD Left Superior Longitudinal Fasciculus 1	-0.031	-0.097
FA Left Superior Longitudinal Fasciculus 2	-0.121	0.011
MD Left Superior Longitudinal Fasciculus 2	0.168	-0.273
RD Left Superior Longitudinal Fasciculus 2	0.185	-0.223
FA Left Superior Longitudinal Fasciculus 3	-0.130	0.095
MD Left Superior Longitudinal Fasciculus 3	0.076	-0.203
RD Left Superior Longitudinal Fasciculus 3	0.089	-0.162
FA Right Inferior Longitudinal Fasciculus	-0.092	0.211
MD Right Inferior Longitudinal Fasciculus	0.288	-0.212

RD Right Inferior Longitudinal Fasciculus	0.228	-0.253
FA Left Inferior Longitudinal Fasciculus	0.021	-0.050
MD Left Inferior Longitudinal Fasciculus	.306*	-0.230
RD Left Inferior Longitudinal Fasciculus	0.170	-0.110
FA Right Uncinate Fasciculus	0.027	0.105
MD Right Uncinate Fasciculus	0.017	-0.086
RD Right Uncinate Fasciculus	-0.008	-0.107
FA Left Uncinate Fasciculus	0.072	-0.120
MD Left Uncinate Fasciculus	0.003	-0.068
RD Left Uncinate Fasciculus	-0.029	-0.010
FA Right Fornix	0.011	0.070
MD Right Fornix	0.263	-0.196
RD Right Fornix	0.220	-0.193
FA Left Fornix	-0.060	0.073
MD Left Fornix	0.211	-0.016
RD Left Fornix	0.183	-0.037
FA Corpus Callosum	-0.159	0.053
MD Corpus Callosum	0.276	-0.068
RD Corpus Callosum	0.245	-0.081
****. Correlation is significant at the 0.0033 level (2-tailed).*		
***. Correlation is significant at the 0.01 level (2-tailed).*		
**. Correlation is significant at the 0.05 level (2-tailed).*		

3d. CEMS PR Anger Subscale Associations with Child White Matter Connectivity Indices

Correlations	CEMS PR Anger Inhibition (*r* value)	CEMS PR Anger Dysregulation (*r* value)
FA Right Cingulum	0.143	0.188
MD Right Cingulum	-0.260	-0.146
RD Right Cingulum	-0.229	-0.205
FA Left Cingulum	-0.055	0.189
MD Left Cingulum	-0.048	-0.109
RD Left Cingulum	0.002	-0.178
FA Right Superior Longitudinal Fasciculus 1	0.152	0.272
MD Right Superior Longitudinal Fasciculus 1	-0.174	-0.110
RD Right Superior Longitudinal Fasciculus 1	-0.184	-0.204
FA Right Superior Longitudinal Fasciculus 2	-0.041	0.223
MD Right Superior Longitudinal Fasciculus 2	-0.076	-0.082
RD Right Superior Longitudinal Fasciculus 2	-0.022	-0.130
FA Right Superior Longitudinal Fasciculus 3	-0.070	0.011
MD Right Superior Longitudinal Fasciculus 3	-0.105	0.120
RD Right Superior Longitudinal Fasciculus 3	-0.038	0.086
FA Left Superior Longitudinal Fasciculus 1	-0.032	0.099
MD Left Superior Longitudinal Fasciculus 1	-0.189	0.076
RD Left Superior Longitudinal Fasciculus 1	-0.101	0.002
FA Left Superior Longitudinal Fasciculus 2	0.145	0.133
MD Left Superior Longitudinal Fasciculus 2	-0.197	-0.140
RD Left Superior Longitudinal Fasciculus 2	-0.205	-0.156

FA Left Superior Longitudinal Fasciculus 3	0.082	0.005
MD Left Superior Longitudinal Fasciculus 3	-0.159	0.001
RD Left Superior Longitudinal Fasciculus 3	-0.128	-0.019
FA Right Inferior Longitudinal Fasciculus	-0.035	0.062
MD Right Inferior Longitudinal Fasciculus	-0.138	0.083
RD Right Inferior Longitudinal Fasciculus	-0.093	0.062
FA Left Inferior Longitudinal Fasciculus	-0.152	0.033
MD Left Inferior Longitudinal Fasciculus	-0.083	0.065
RD Left Inferior Longitudinal Fasciculus	0.043	0.046
FA Right Uncinate Fasciculus	-0.232	-0.040
MD Right Uncinate Fasciculus	0.057	0.070
RD Right Uncinate Fasciculus	0.143	0.069
FA Left Uncinate Fasciculus	-0.041	0.044
MD Left Uncinate Fasciculus	0.025	0.018
RD Left Uncinate Fasciculus	0.067	0.011
FA Right Fornix	-0.053	-0.124
MD Right Fornix	-0.188	.344*
RD Right Fornix	-0.139	.323*
FA Left Fornix	0.112	0.055
MD Left Fornix	-0.159	-0.029
RD Left Fornix	-0.137	-0.040
FA Corpus Callosum	0.172	-0.210
MD Corpus Callosum	-0.266	.337*
RD Corpus Callosum	-0.226	.338*
****. Correlation is significant at the 0.0033 level (2-tailed).*		
***. Correlation is significant at the 0.01 level (2-tailed).*		

*. *Correlation is significant at the 0.05 level (2-tailed).*

3e. CEMS PR Sadness Subscale Associations with Child White Matter Connectivity Indices

Correlations	CEMS PR Sadness Inhibition (*r* value)	CEMS PR Sadness Dysregulation (*r* value)
FA Right Cingulum	.320*	-0.158
MD Right Cingulum	-.435**	-0.028
RD Right Cingulum	-.421***	0.049
FA Left Cingulum	0.038	0.087
MD Left Cingulum	-.313*	-0.035
RD Left Cingulum	-0.196	-0.074
FA Right Superior Longitudinal Fasciculus 1	.352*	-0.108
MD Right Superior Longitudinal Fasciculus 1	-.368*	-0.019
RD Right Superior Longitudinal Fasciculus 1	-.389**	0.016
FA Right Superior Longitudinal Fasciculus 2	0.113	0.040
MD Right Superior Longitudinal Fasciculus 2	-0.196	0.002
RD Right Superior Longitudinal Fasciculus 2	-0.148	-0.004
FA Right Superior Longitudinal Fasciculus 3	0.149	-0.202
MD Right Superior Longitudinal Fasciculus 3	-0.202	0.136
RD Right Superior Longitudinal Fasciculus 3	-0.185	0.184
FA Left Superior Longitudinal Fasciculus 1	0.183	-0.125
MD Left Superior Longitudinal Fasciculus 1	-.374*	0.113
RD Left Superior Longitudinal Fasciculus 1	-.325*	0.155
FA Left Superior Longitudinal Fasciculus 2	0.113	-0.032

MD Left Superior Longitudinal Fasciculus 2	-0.225	-0.113
RD Left Superior Longitudinal Fasciculus 2	-0.207	-0.091
FA Left Superior Longitudinal Fasciculus 3	0.272	-0.254
MD Left Superior Longitudinal Fasciculus 3	-.316*	0.086
RD Left Superior Longitudinal Fasciculus 3	-.299*	0.163
FA Right Inferior Longitudinal Fasciculus	0.068	-0.103
MD Right Inferior Longitudinal Fasciculus	-0.278	0.001
RD Right Inferior Longitudinal Fasciculus	-0.218	0.048
FA Left Inferior Longitudinal Fasciculus	-0.069	-0.161
MD Left Inferior Longitudinal Fasciculus	-0.269	0.152
RD Left Inferior Longitudinal Fasciculus	-0.104	0.202
FA Right Uncinate Fasciculus	-0.031	-0.081
MD Right Uncinate Fasciculus	-0.168	0.240
RD Right Uncinate Fasciculus	-0.066	0.196
FA Left Uncinate Fasciculus	0.159	-0.265
MD Left Uncinate Fasciculus	-0.16	0.162
RD Left Uncinate Fasciculus	-0.142	0.227
FA Right Fornix	0.019	-0.105
MD Right Fornix	-0.164	0.128
RD Right Fornix	-0.143	0.132
FA Left Fornix	0.22	0.094
MD Left Fornix	-0.294	-0.108
RD Left Fornix	-0.276	-0.130
FA Corpus Callosum	0.218	-0.206
MD Corpus Callosum	-0.291	0.253
RD Corpus Callosum	-0.235	0.249

***. *Correlation is significant at the 0.0033 level (2-tailed).*
**. *Correlation is significant at the 0.01 level (2-tailed).*
*. *Correlation is significant at the 0.05 level (2-tailed).*

3f. CEMS PR Worry Subscale Associations with Child White Matter Connectivity Indices

Correlations	CEMS PR Worry Inhibition (*r* value)	CEMS PR Worry Dysregulation (*r* value)
FA Right Cingulum	-0.158	-0.158
MD Right Cingulum	-0.028	-0.028
RD Right Cingulum	0.049	0.049
FA Left Cingulum	0.087	0.087
MD Left Cingulum	-0.035	-0.035
RD Left Cingulum	-0.074	-0.074
FA Right Superior Longitudinal Fasciculus 1	-0.108	-0.108
MD Right Superior Longitudinal Fasciculus 1	-0.019	-0.019
RD Right Superior Longitudinal Fasciculus 1	0.016	0.016
FA Right Superior Longitudinal Fasciculus 2	0.040	0.040
MD Right Superior Longitudinal Fasciculus 2	0.002	0.002
RD Right Superior Longitudinal Fasciculus 2	-0.004	-0.004
FA Right Superior Longitudinal Fasciculus 3	-0.202	-0.202
MD Right Superior Longitudinal Fasciculus 3	0.136	0.136
RD Right Superior Longitudinal Fasciculus 3	0.184	0.184
FA Left Superior Longitudinal Fasciculus 1	-0.125	-0.125
MD Left Superior Longitudinal Fasciculus 1	0.113	0.113

RD Left Superior Longitudinal Fasciculus 1	0.155	0.155
FA Left Superior Longitudinal Fasciculus 2	-0.032	-0.032
MD Left Superior Longitudinal Fasciculus 2	-0.113	-0.113
RD Left Superior Longitudinal Fasciculus 2	-0.091	-0.091
FA Left Superior Longitudinal Fasciculus 3	-0.254	-0.254
MD Left Superior Longitudinal Fasciculus 3	0.086	0.086
RD Left Superior Longitudinal Fasciculus 3	0.163	0.163
FA Right Inferior Longitudinal Fasciculus	-0.103	-0.103
MD Right Inferior Longitudinal Fasciculus	0.001	0.001
RD Right Inferior Longitudinal Fasciculus	0.048	0.048
FA Left Inferior Longitudinal Fasciculus	-0.161	-0.161
MD Left Inferior Longitudinal Fasciculus	0.152	0.152
RD Left Inferior Longitudinal Fasciculus	0.202	0.202
FA Right Uncinate Fasciculus	-0.081	-0.081
MD Right Uncinate Fasciculus	0.240	0.240
RD Right Uncinate Fasciculus	0.196	0.196
FA Left Uncinate Fasciculus	-0.265	-0.265
MD Left Uncinate Fasciculus	0.162	0.162
RD Left Uncinate Fasciculus	0.227	0.227
FA Right Fornix	-0.105	-0.105
MD Right Fornix	0.128	0.128
RD Right Fornix	0.132	0.132
FA Left Fornix	0.094	0.094
MD Left Fornix	-0.108	-0.108
RD Left Fornix	-0.130	-0.130
FA Corpus Callosum	-0.206	-0.206

MD Corpus Callosum	0.253	0.253
RD Corpus Callosum	0.249	0.249
***. *Correlation is significant at the 0.0033 level (2-tailed).*		
**. *Correlation is significant at the 0.01 level (2-tailed).*		
*. *Correlation is significant at the 0.05 level (2-tailed).*		

Table 4. Associations between DERS and CEMS

4a. Associations between DERS and CEMS CR

	DERS total	DERS Nonacceptance	DERS Goals	DERS Impulse	DERS Awareness	DERS Strategies	DERS Clarity	CEMS CR anger inhibition.	CEMS CR anger dysregulation	CEMS CR sadness inhibition	CEMS CR sadness dysregulation	CEMS CR worry inhibition	CEMS CR worry dysregulation
DERS total	--												
DERS Nonacceptance	.841**	--											
DERS Goals	.787**	.600**	--										
DERS Impulse	.872**	.676**	.741**	--									
DERS Awareness	.492**	0.294	0.17	0.173	--								
DERS Strategies	.884**	.711**	.729**	.871**	0.187	--							
DERS Clarity	.775**	.610**	.415**	.563**	.529**	.559**	--						
CEMS CR anger inhibit	0.175	.320*	0.051	0.116	0.118	0.14	0.044	--					

ion.													
CEMS CR anger dysreg ulation	0.035	-0.076	0.02	0.014	0.197	-0.041	0.074	-0.07	--				
CEMS CR sadnes s inhibit ion	0.294	.409**	0.131	0.209	0.201	0.213	0.199	.589**	0.083	--			
CEMS CR sadnes s dysreg ulation	0.048	0.081	-0.018	0.036	0.235	-0.063	-0.05	.344*	.391**	.330*	--		
CEMS CR worry inhibit ion	0.037	-0.059	0.124	0.11	-0.078	0.085	-0.013	0.238	-0.127	.417**	-0.174	--	
CEMS CR worry dysreg ulation	0.188	0.195	0.1	0.195	0.25	0.128	-0.016	0.291	0.14	0.256	.504**	-0.153	--
	***. Correlation is significant at the 0.01 level (2-tailed).*												

	**. Correlation is significant at the 0.05 level (2-tailed).*

4b. Associations between DERS and CEMS PR

	DERS total	DERS Nonacceptance	DERS Goals	DERS Impulse	DERS Awareness	DERS Strategies	DERS Clarity	CEMS PR anger inhibition.	CEMS PR anger dysregulation	CEMS PR sadness inhibition	CEMS PR sadness dysregulation	CEMS PR worry inhibition	CEMS PR worry dysregulation
DERS total	--												
DERS Nonacceptance	.841**	--											
DERS Goals	.787**	.600**	--										
DERS Impulse	.872**	.676**	.741**	--									
DERS Awareness	.492**	0.294	0.17	0.173	--								
DERS Strategies	.884**	.711**	.729**	.871**	0.187	--							
DERS Clarity	.775**	.610**	.415**	.563**	.529**	.559**	--						
CEMS PR	-0.004	0.044	0.044	-0.078	-0.044	-0.088	0.138	--					

anger inhibition													
CEMS PR anger dysregulation	0.247	0.185	0.259	0.228	0.115	0.221	0.137	-0.15	--				
CEMS PR sadness inhibition	0.152	0.127	0.119	-0.065	0.186	0.082	0.298	.747**	0.048	--			
CEMS PR sadness dysregulation	.373*	0.286	.436**	.387*	0.173	.407**	0.006	-0.024	.571**	-0.003	--		
CEMS PR worry inhibition	.321*	.318*	0.161	0.133	.413**	0.112	.401**	.377*	0.061	.576**	-0.056	--	
CEMS PR worry dysregulation	.384*	.381*	0.251	.394**	0.089	.447**	0.179	0.05	.486**	0.009	.662**	0.028	--
	***. Correlation is significant at the 0.01 level (2-tailed).*												

	**. Correlation is significant at the 0.05 level (2-tailed).*

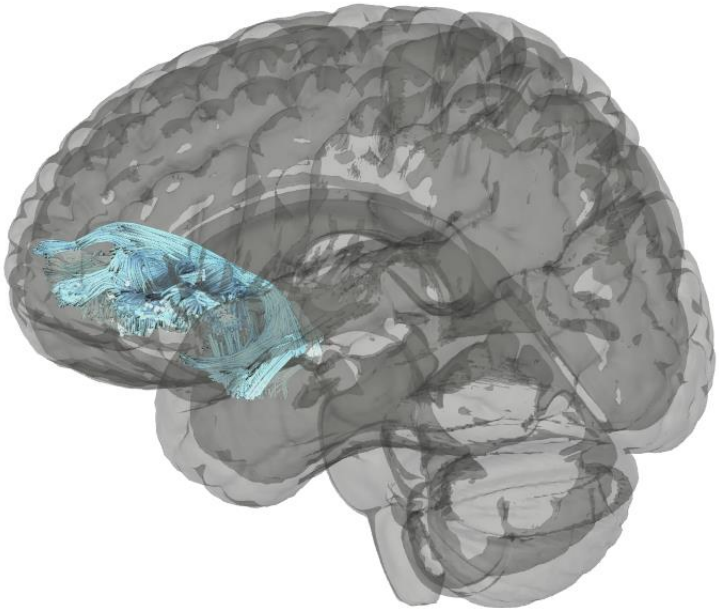
Table 5. Associations between DERS and PQ

	DERS total	DERS NOCCEPTANCE	DERS GOALS	DERS IMPULSE	DERS AWARENESS	DERS STRATEGIES	DERS CLARITY	Warmth	Demanding	Corporal Punishment
DERS total	--									
DERS NOCCEPTANCE	.841**	--								
DERS GOALS	.787**	.600**	--							
DERS IMPULSE	.872**	.676**	.741**	--						
DERS AWARENESS	.492**	0.294	0.17	0.173	--					
DERS STRATEGIES	.884**	.711**	.729**	.871**	0.187	--				
DERS CLARITY	.775**	.610**	.415**	.563**	.529**	.559**	--			
Warmth	-.419**	-0.304	-0.275	-.383*	-.361*	-0.288	-.332*	--		
Demandi	-0.292	-0.199	-0.232	-0.146	-.306*	-0.159	-.342*	.490**	--	

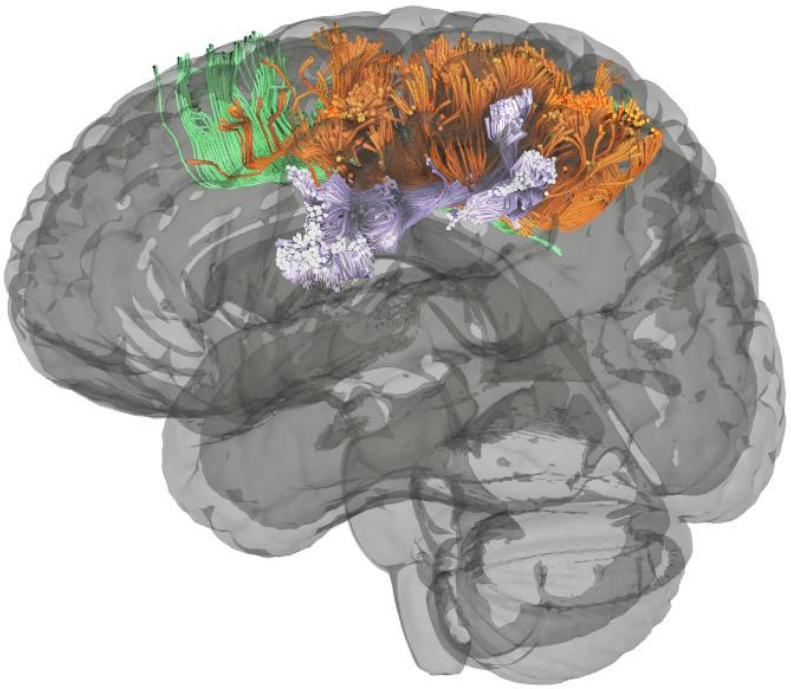
ng										
Corporal Punishment	.354*	0.297	0.084	.359*	.323*	0.253	.314*	-.521**	-0.194	--
**. Correlation is significant at the 0.01 level (2-tailed).										
*. Correlation is significant at the 0.05 level (2-tailed).										

Figure 2. Example deterministic pathways

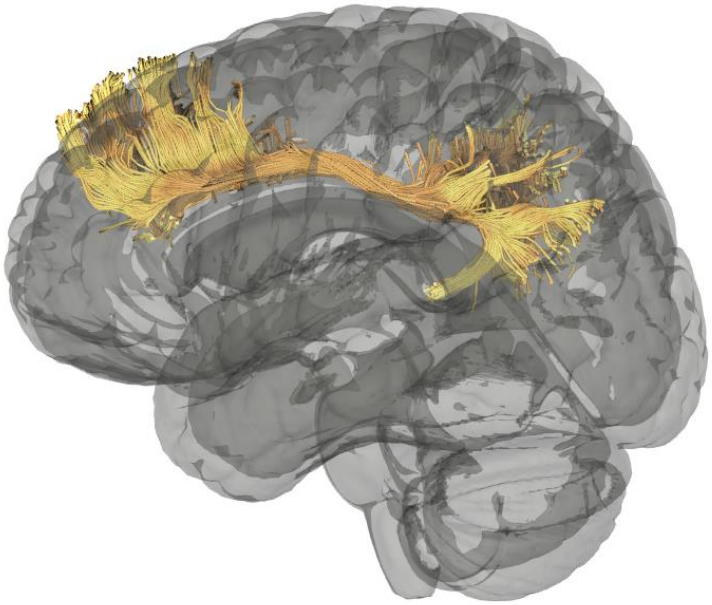
2a. Uncinate fasciculus (light blue)



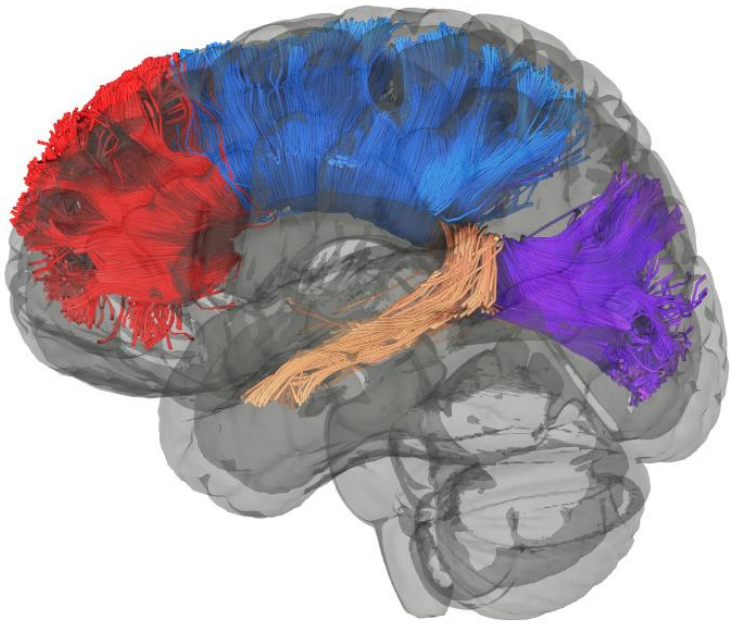
2b. Superior longitudinal fasciculus (Segment 1: violet blue, Segment 2: dark orange, Segment 3: light green)



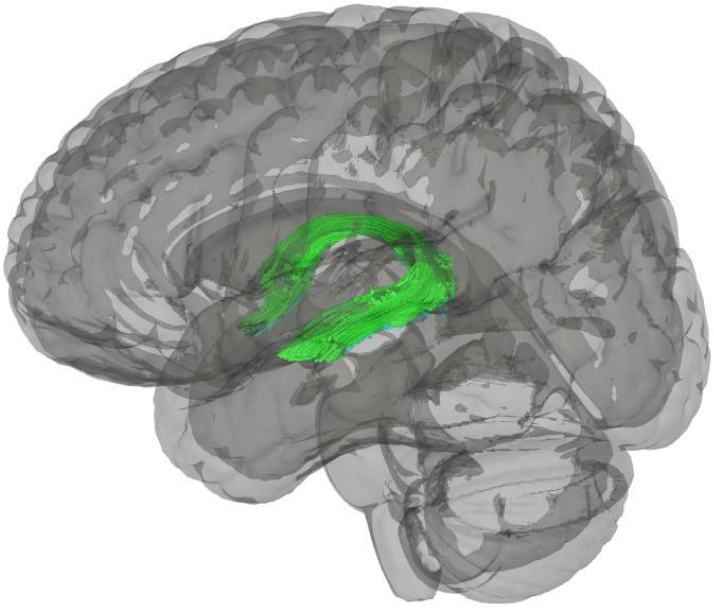
2c. Cingulum Bundle (yellow)



2d. Corpus Callosum (Forcep minor: red, body: dark blue, Tapetum: light orange, Forcep major: purple)



2e. Fornix (dark green)



2f. Inferior Longitudinal Fasciculus (pink)

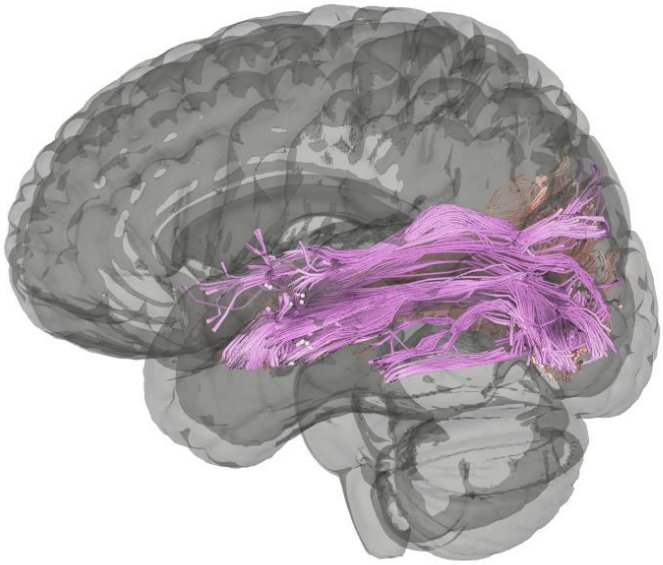
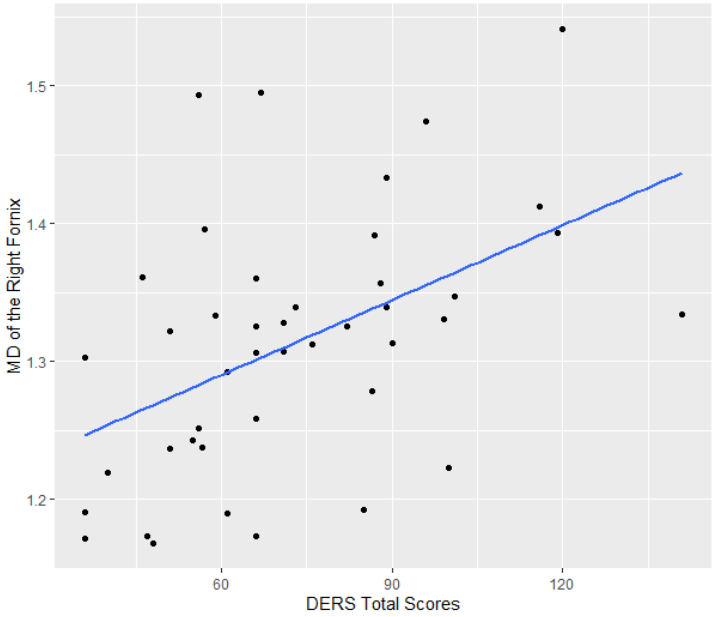
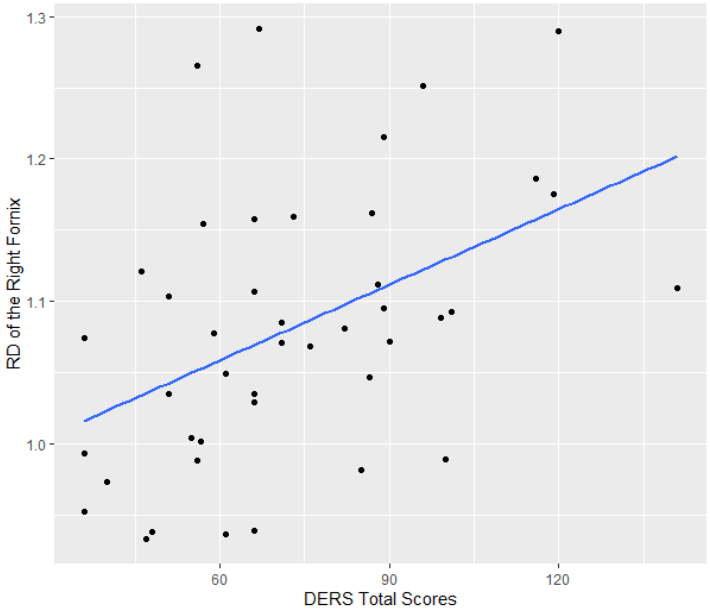


Figure 3. Scatterplots of DERS and white matter indices

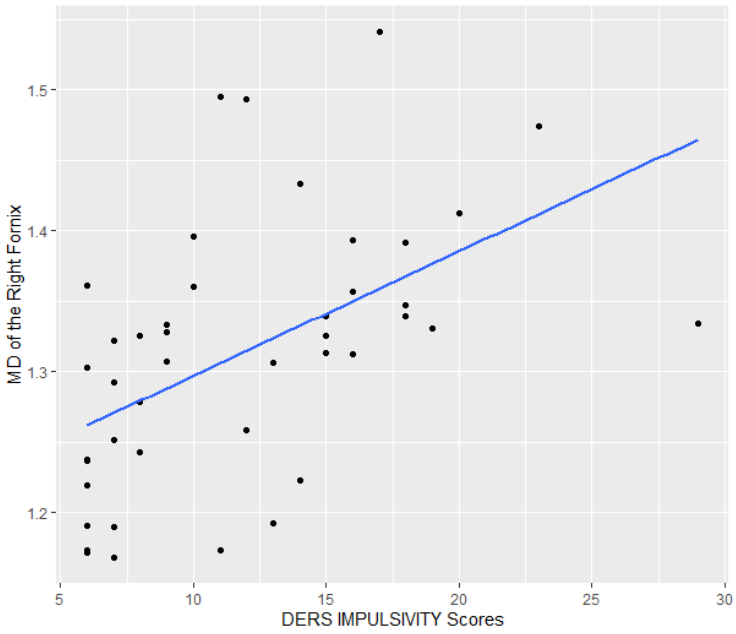
3a.



3b.


RD of the Right Fornix
1.3
1.2
1.1
1.0
60
90
120
DERS Total Scores

3c.


MD of the Right Fornix
1.5
1.4
1.3
1.2
5
10
15
20
25
30
DERS IMPULSIVITY Scores

3d.

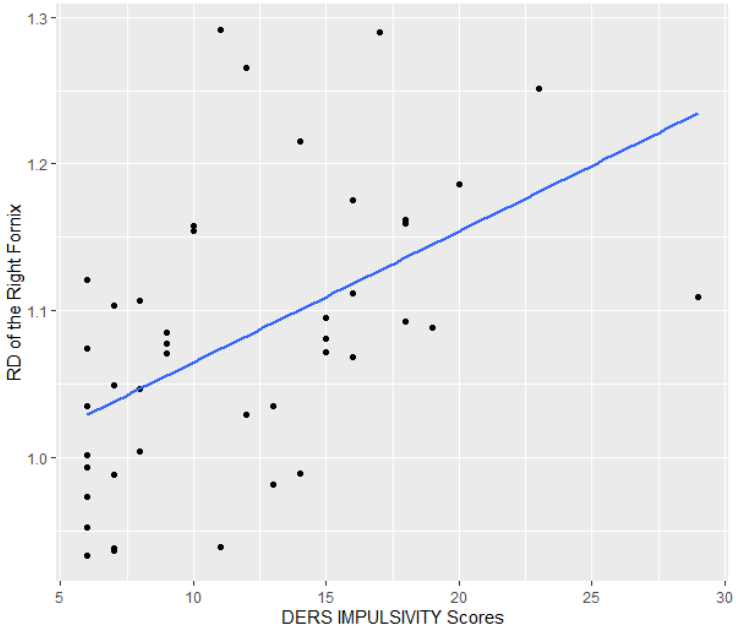
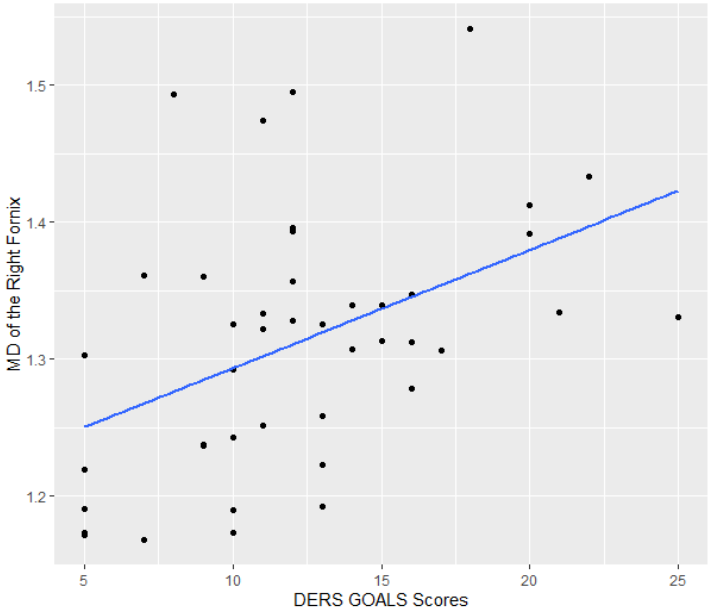
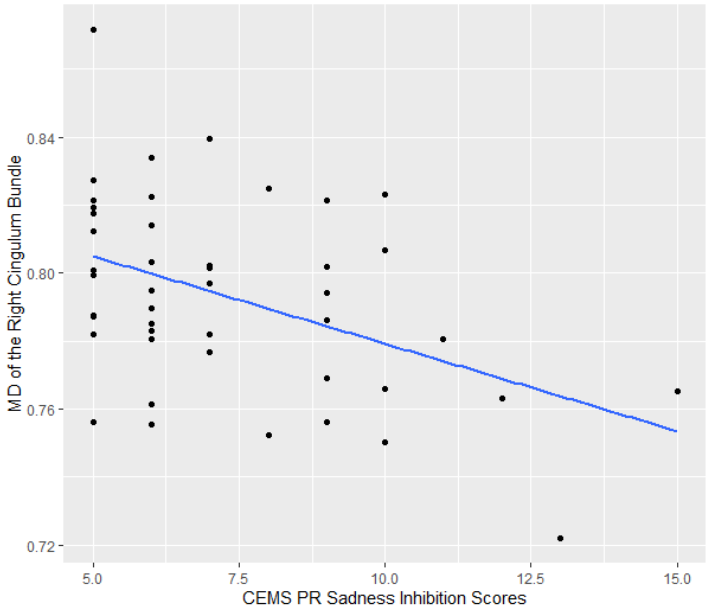


Figure 4. Scatterplots of CEMS and white matter indices

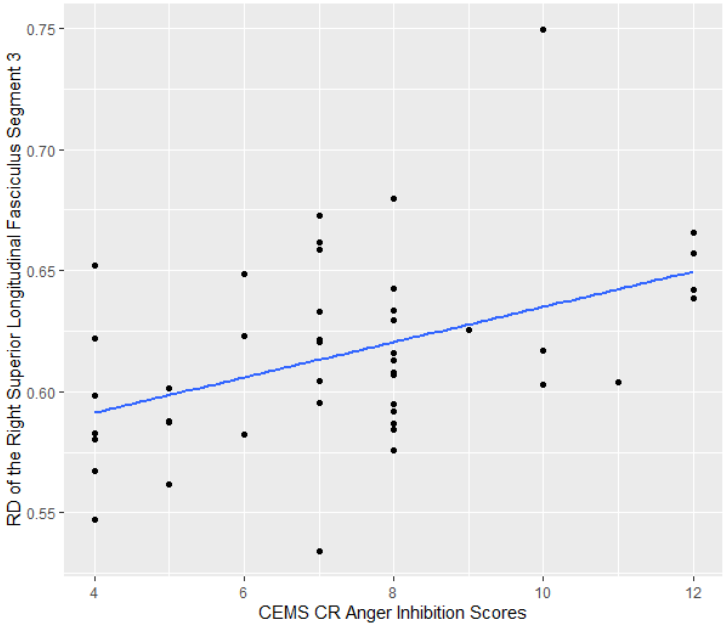
4a.



4b.


MD of the Right Cingulum Bundle
0.84
0.80
0.76
0.72
5.0
7.5
10.0
12.5
15.0
CEMS PR Sadness Inhibition Scores

4c.


RD of the Right Superior Longitudinal Fasciculus Segment 3
0.75
0.70
0.65
0.60
0.55
4
6
8
10
12
CEMS CR Anger Inhibition Scores