Received: 16 February 2022 | Revised: 13 April 2022 | Accepted: 21 May 2022

DOI: 10.1002/dev.22303

Developmental Psychobiology WILEY

RESEARCH ARTICLE

Associations of maternal emotion regulation with child white matter connectivity in Black American mother–child dyads

Chenyang Wang[1] | Dominique L. La Barrie[2] | Abigail Powers[3] | Anais F. Stenson[4] | Sanne J. H. van Rooij[3] | Jennifer S. Stevens[3] | Tanja Jovanovic[4] | Bekh Bradley[3,5] | Robin E. McGee[6] | Negar Fani[3]

[1]Department of Psychiatry, University of Pittsburgh School of Medicine, Pittsburgh, Pennsylvania, USA

[2]Department of Psychology, University of Georgia, Athens, Georgia, USA

[3]Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine, Atlanta, Georgia, USA

[4]Department of Psychiatry and Behavioral Neuroscience, Wayne State University, Detroit, Michigan, USA

[5]Atlanta Veterans Affairs Medical Center, Decatur, Georgia, USA

[6]Rollins School of Public Health, Emory University, Atlanta, Georgia, USA

Correspondence
Negar Fani, Department of Psychiatry and Behavioral Sciences, Emory University School of Medicine, 100 Woodruff Circle, Atlanta, GA 30322, USA.
Email: nfani@emory.edu

Funding information
Emory Medical Care Foundation; Center for Scientific Review, Grant/Award Numbers: AT011267, HD071982, MH100122, MH111682; Emory University Research Council

Abstract

Parental emotion regulation plays a major role in parent-child interactions, and in turn, neural plasticity in children, particularly during sensitive developmental periods. However, little is known about how parental emotion dysregulation is associated with variation in children's brain structure, which was the goal of this study. Forty-five Black American mother–child dyads were recruited from an intergenerational trauma study; emotion regulation in mothers and their children (age 8–13 years) was assessed. Diffusion-weighted images were collected in children; deterministic tractography was used to reconstruct pathways of relevance to emotion regulation. Metrics of white matter connectivity [fractional anisotropy (FA), mean diffusivity (MD)] were extracted for pathways. Socio-economic variables were also included in statistical models. Maternal emotion dysregulation was the strongest predictor of child fornix MD ($r = .35$, $p = .001$), indicating that more severe emotion dysregulation in mothers corresponded with lower fornix connectivity in children. Maternal impulsivity was a strong predictor of child fornix MD ($r = .51$, $p < .001$). Maternal emotion dysregulation may adversely influence connectivity of the child.s fornix, a hippocampal-striatal pathway implicated in reward processes; these associations remained even after accounting for other socio-environmental factors. Dysregulated maternal emotions may uniquely impact children's adaptation to trauma/stress by affecting networks that support appetitive processing.

KEYWORDS
Black American, diffusion tensor imaging, emotion regulation, intergenerational, white matter connectivity

1 | INTRODUCTION

Emotion regulation describes the ability to process, monitor, and manage emotions effectively (Thompson, 1994). These abilities develop throughout childhood and adolescence and into adulthood (Denham et al., 2002; John & Gross, 2004). The awareness and acceptance of emotions, the ability to stay engaged and persist with goal-oriented behavior in the face of negative emotions, and the ability to control impulsive behaviors are all components of adaptive emotion regulation (Gratz & Roemer, 2004). Emotion regulation difficulties, which may manifest as impulsivity, nonacceptance of emotional response, and an inability to persist with goals in the face of distress, have been

Abbreviations: CB, cingulum bundle; CEMS, Child Emotion Management Scale; DERS, Difficulties in Emotion Regulation Scale; FA, fractional anisotropy; HPA, hypothalamus–pituitary–adrenal; MD, mean diffusivity; PFC, prefrontal cortex; SD, standard deviation; TESI, Traumatic Events Screening Inventory; UF, uncinate fasciculus.

associated with mood disruptions and trauma-related disorders, including major depressive disorder and posttraumatic stress disorder (PTSD) (Hofmann et al., 2012; Pencea et al., 2020; Powers et al., 2015). As such, emotion regulation difficulties (often termed emotion dysregulation) are considered a transdiagnostic phenomenon present in various mental health disorders (Bradley et al., 2011).

Emotion regulation processes, including the appraisal of emotional cues, selective inhibition of response, and attentional focus and shifting, are supported by cognitive control and salience neural networks, which include the amygdala, insula, and anterior cingulate cortex (Cole & Schneider, 2007; Phillips et al., 2008; Seeley et al., 2007; Zilverstand et al., 2017). Functional connectivity between these network regions has been linked to specific emotion regulation processes; for example, the amygdala and ventromedial prefrontal cortex (PFC) have been linked to inhibition of threat response (Åhs et al., 2015), and connectivity between the amygdala and dorsal anterior cingulate cortex is associated with the detection of salient stimuli and emotion appraisal (Uddin, 2017; Peters et al., 2016; Etkin et al., 2011). Further, the hippocampus is a key region for contextual processing of emotionally salient stimuli; connectivity of the hippocampus with the striatum, which is essential to reward-related responding, has been implicated in reward-learning processes. As such, hippocampal–striatal pathways are relevant to learning emotional reward in social contexts (Cox & Witten, 2019).

Associational white matter fibers provide connections between these brain regions involved with emotion regulation. Among the paths highlighted as most salient to emotion regulation are the uncinate fasciculus (UF), the cingulum bundle (CB), and fornix (Tan et al., 2020; Cutuli, 2014; Phelps, 2004; Chahal et al., 2021). The UF is a ventrolimbic white matter pathway connecting the ventral PFC with the amygdala that supports various emotional processes, including threat detection and inhibition (Papagno et al., 2010; Oishi et al., 2014; Fujie et al., 2008; Olson et al., 2015). The CB is a dorsolimbic white matter pathway that connects the limbic system with the cingulate cortex and is involved in executive control, episodic memory, and fear extinction (Bubb et al., 2018; Fani et al., 2015; 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 abnormalities in reward learning (Gaffan et al., 1984), and diminished connectivity of this pathway is also linked to anhedonia, suggesting its general relevance to appetitive processes (Harnett et al., 2021). Taken together, diminished connectivity in the CB, UF, and fornix has been linked with impaired emotional management (Barysheva et al., 2013; Baur et al., 2011; Harnett et al., 2018; Korgaonkar et al., 2010; Liao et al., 2014; Manelis et al., 2021), suggesting that structural disruptions in these pathways may result in impaired emotion regulation.

Parents may influence the plasticity of these connections via their role in shaping their children's emotion regulation; this can occur through emotion modeling (Kerr et al., 2019; Morris et al., 2007). Parents with emotion regulation difficulties are less likely to model adaptive emotion regulation strategies. Maternal emotion regulation difficulties have been linked to the presence of emotion dysregulation in children (Morelen et al., 2016; Powers et al., 2022) and negative psychological outcomes overall (Powers et al., 2022). Mothers who report effective emotion regulation 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).

Maternal emotion regulation difficulties may also influence the frequency of negative behaviors toward children (Morelen et al., 2016); these behaviors can represent a chronic stressor for children, with consequences for child neural plasticity. Adverse mother–child interactions can serve as a chronic stressor, activating the stress response system (hypothalamus–pituitary–adrenal [HPA] axis) in children. Chronic HPA activation adversely affects neuroplasticity, particularly for stress-sensitive brain structures (Lupien et al., 2009), with deleterious effects on dendritic and axonal development (Helmeke et al., 2009; Ivy et al., 2010; Liu et al., 2012) in the PFC and limbic system. In rodents exposed to various forms of early life stress, reductions in dendritic complexity and reduced myelination in white matter tracts that connect emotion regulation network regions have been observed (Helmeke et al., 2009; Liu et al., 2012; Muhammad & Kolb, 2011). Further, reduced fractional anisotropy (FA) and increased mean diffusivity (MD), which are indices of white matter microstructural integrity, have been found in the UF, the superior longitudinal fasciculus, the corona radiata, the fornix, the corpus callosum, arcuate fasciculus, and the inferior longitudinal fasciculus of individuals who experienced childhood maltreatment, suggesting that these chronic stressors compromise white matter integrity (Bick et al., 2015; Govindan et al., 2010; Hanson et al., 2013; Huang et al., 2012; McCarthy-Jones et al., 2018).

There are different pathways through which emotion regulation in parents may influence their children's emotion regulation, which is mediated by neuroplastic changes throughout development. However, no extant studies have examined associations between emotion regulation difficulties in parents, specifically mothers, and white matter connectivity in their children. This was our study objective. We included a historically understudied group of individuals—Black American mother–child dyads. The majority of extant intergenerational studies have been conducted with predominantly White middle-class families. Marginalized populations, including people of color (specifically, Black Americans), and economically disadvantaged families have been largely ignored in this line of research. Black communities in the United States face a disproportionately high burden of chronic stress and trauma due to racial discrimination and systemic inequities, which are in turn associated with mental and physical health disparities (Carter et al., 2021; Kirkinis et al., 2021). Despite this fact, Black Americans have been historically ignored in intergenerational research (Barnes et al., 2021).

To our knowledge, this study is the first to investigate associations between maternal emotion regulation and white matter connectivity in children. To examine white matter connectivity, 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, and fornix. Using different white matter connectivity indices (FA and MD), we investigated relationships between these white matter assays in maternal emotion regulation difficulties. Our primary hypothesis was that greater maternal emotion regulation difficulties would be associated with lesser connectivity (lower FA and higher MD) of these pathways in children. Secondarily, we examined relationships between specific aspects of maternal emotion dysregulation (e.g., impulsivity, emotion nonacceptance) and child white matter connectivity. Finally, we conducted exploratory analyses to examine associations of children's white matter connectivity with reported child emotion regulation difficulties. We hypothesized that lesser connectivity of these pathways would be associated with greater emotion regulation difficulties in children. Given that environmental stressors such as economic disadvantage and psychological trauma exposure (which are experienced by a majority of our study population) can also play a role in shaping neural plasticity (Kim et al., 2013; Villalta et al., 2018), we accounted for variance associated with these factors in our analyses.

2 | MATERIALS AND METHODS

2.1 | Procedure

The current study included 45 mother–child dyads from two NIH-funded studies investigating intergenerational trauma in Black American mothers and their children (HD071982) and examined the relationship between trauma exposure and neural plasticity during critical periods of brain development in children (MH111682). All study procedures were approved by the Institutional Review Board of Emory University and Grady Research Oversight Committee. Participant recruitment for these two studies took place in the waiting rooms of a pediatric hospital or the primary care clinic of a publicly funded hospital in Atlanta. After potential participants were approached, mothers completed an initial interview with a trained research assistant to determine eligibility.

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 completed consent to join the study, mother–child dyads were invited to complete individual, separate clinical interviews, and self-report measures to assess their trauma history and emotion regulation. Child participants also completed a magnetic resonance imaging (MRI), which included diffusion-weighted imaging. Clinical and MRI data were collected at different time points across a period of a year and a half, due to the longitudinal nature of the original studies. Prior to each MRI scan, children were given the opportunity to complete a mock scan to ensure participant comfort and reduce the likelihood of motion-related artifacts.

2.2 | Participants

Forty-five Black American mother–child dyads were recruited; complete mother and child emotion regulation data and child white matter data were available for 44 dyads. Inclusion criteria for mothers were as follows: 18–65 years old; identified as Black American/Black; able to provide informed consent; primary caretaker of an 8- to 13-year-old child. Child inclusion criteria were as follows: 8–13 years old; willingness to provide assent/consent. Exclusion criteria for mothers and children were cognitive disability or diagnosis of autism spectrum disorder. The average age for mothers was 37.11 (standard deviation [*SD*] = 8.86, range = 26–59); the average age for children was 10.06 (*SD* = 1.45, range = 8–13; 51% female). Further demographic and clinical characteristics of mothers and their children are provided in Table 1.

2.3 | Clinical measures

Maternal emotion regulation difficulties were measured with the Difficulties in Emotion Regulation Scale (DERS) (Cronbach's $\alpha = .95$) (Gratz & Roemer, 2004), which includes six subscales: nonacceptance of negative affect (Cronbach's $\alpha = .92$); difficulty controlling impulses in the presence of negative affect (Cronbach's $\alpha = .88$); difficulty engaging in goal-directed behavior in the presence of negative emotions (Cronbach's $\alpha = .86$); difficulties with adaptive emotion regulation skills (Cronbach's $\alpha = .92$); difficulties with emotional clarity (Cronbach's $\alpha = .82$); and difficulties with emotional awareness (Cronbach's $\alpha = .82$) (Hallion et al., 2018). Child emotion regulation difficulties were measured using the Children's Emotion Management Scale parent report (CEMS; Cronbach's $\alpha = .75$); dysregulation and inhibition scores for negative emotions (anger, sadness, and worry) were used in analyses. The Traumatic Events Screening Inventory (TESI) was administered to mothers to assess child exposure to various traumatic events (Ghosh-Ippen et al., 2002). A summed score representing exposure to different types of traumatic events was used in analyses. Further details on clinical measures and associations among index scores for these measures are provided in Methods and Results in the Supporting Information and in Table S1.

2.4 | Diffusion tensor imaging acquisition and deterministic tractography

Scanning was conducted with 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 × 2.0 mm thick axial slices, matrix = 106 × 106, field of view = 212 × 180 mm, voxel size = 2 × 2 × 2 mm, TR = 3292 ms, and TE = 96 ms. The diffusion weighting was isotropically distributed along 138 directions using a

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
$1000–1999	29.5
≥$2000	22.7
Difficulties in Emotion Regulation Scale (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)
Child demographic characteristics	Mean (*SD*) (*N* = 45)
Child age (8–13)	10.06 (1.45)
Child gender	%
Female	51.1
Male	46.6
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)

b-value of 1000 s/mm^2. Scan preprocessing steps are detailed in the Supporting Information.

Deterministic tractography was performed using DSI Studio's graphical user interface (http://dsi-studio.labsolver.org). Automated masks were used to generate three-dimensional diffusion tensor imaging volumes, and white matter connectivity indices, FA, and MD values were extracted for the bilateral UF, fornix, and CB using the software's automated system. Example deterministic reconstructions of these tracts are provided in Figure S1.

2.5 | Data analyses

Our primary objective was to examine associations of emotion regulation difficulties in mothers with child white matter connectivity in our tracts of interest (CB, UF, fornix). First, we examined correlations between DERS total score and indices of children's white matter structural connectivity (FA and MD, each averaged across both hemispheres) with maternal emotion regulation difficulties (DERS total). Benjamini–Hochberg correction was applied for each family of tests to adjust for error inflation due to multiple comparisons; where significant associations were observed, we examined correlations of FA and/or MD for each hemisphere with DERS subscales (Impulsivity, Nonacceptance, Clarity, Goals, Awareness, Strategies). Significant findings were also subject to follow-up linear regression analyses to examine specific associations of maternal difficulties in emotion regulation and connectivity of child white matter pathways after accounting for other factors that may affect white matter connectivity, including child sex, economic disadvantage/poverty (monthly household income entered as a binary variable, greater than or less than $1000/month), and child trauma exposure (TESI total). Statistical significance was set at $p < .05$. Secondarily, we conducted exploratory bivariate correlation analyses to examine potential associations between child white matter indices and child difficulties in emotion regulation (CEMS anger, worry, sadness subscales). Analyses were performed with IBM SPSS (version 26) software.

3 | RESULTS

3.1 | Maternal emotion regulation associations with child white matter connectivity indices

Our primary objective was to examine associations of maternal emotion regulation difficulties with child white matter connectivity in our

TABLE 2 Associations of maternal emotion dysregulation (DERS) total scores with child white matter connectivity indices

	DERS total	FA fornix	MD fornix	FA cingulum bundle	MD cingulum bundle	FA uncinate fasciculus	MD uncinate fasciculus
DERS total	–						
FA fornix	−.09	–					
MD fornix	−.35*	−.56**	–				
FA cingulum bundle	.05	.35**	.074	–			
MD cingulum bundle	−.02	−.50**	.27	−.75**	–		
FA uncinate fasciculus	−.02	.19	−.03	.46**	−.55**	–	
MD uncinate fasciculus	−.03	−.32*	.21	−.34*	.43*	−.80**	–

*Correlation is significant at the .05 level (two-tailed).
**Correlation is significant at the .01 level (two-tailed).

TABLE 3 Predictors of child fornix connectivity

		Full model		DERS total	Child sex	Monthly income	Child trauma (TESI)
Tract	Hemi	*F*-statistic (*p*-value)	*R*-value	*β* (*p*-value)			
Fornix	Right						
MD		5.96 (.001)**	.64	.002 (<.001)**	−.005 (.85)	−.063 (.019)*	−.008 (.032)*
Fornix	Left						
MD		2.85 (.039)*	.50	.001 (.14)	−.061 (.030)*	−.064 (.023)*	−.003 (.41)

*$p < .05$;
**$p < .01$.

tracts of interest (CB, UF, fornix). We found that maternal emotion regulation difficulties correlated significantly and positively with MD of the child's fornix ($r = .35$, $p = .001$; Table 2; Figure 1a,b), indicating that higher maternal emotion dysregulation (DERS total) corresponded with lower child fornix connectivity. No other significant correlations were observed at our a priori statistical threshold. To follow up on these analyses, we examined correlations of MD of the fornix for each hemisphere with DERS subscales (Impulsivity, Nonacceptance, Clarity, Goals, Awareness, Strategies). Findings indicated that, among these subscales, maternal impulsivity (DERS Impulsivity subscale) was most strongly correlated with MD of the child's right fornix ($r = .51$, $p < .001$; Table S2). Difficulty engaging in goal-directed behavior in the presence of negative emotions subscale (DERS Goals) was also significantly associated with MD of the child's right fornix ($r = .45$, $p = .003$).

Subsequent regression analyses were conducted to examine unique associations of maternal emotion dysregulation with child fornix connectivity (MD) after accounting for variance associated with child sex, monthly income, and trauma exposure (TESI total; Table 3). We conducted three separate models that included MD of the right and left fornix, separately; all models were statistically significant ($ps < .05$). For MD ($R = .64$, $p = .001$) of the child's right fornix, the predictors collectively accounted for 41% of the variance. Among the predictors in the model, DERS total score predicted the most variance in MD of the child's right fornix ($\beta = .002$, $p < .001$), followed by monthly income ($\beta = -.063$, $p = .019$) and child trauma exposure ($\beta = -.008$, $p = .032$). For MD of the left fornix ($R = .50$, $p = .039$), the predictors accounted for 25% of the variance. DERS total score was not a significant predictor of left fornix MD ($\beta = .001$, $p = .14$); sex significantly predicted variance in MD of the child's left fornix ($\beta = -.061$, $p < .030$), followed by monthly income ($\beta = -.064$, $p = .023$).

3.2 | Child emotion regulation associations with child white matter connectivity indices

Our secondary aim was to examine potential associations between child white matter indices and child difficulties in emotion regulation (CEMS anger, worry, sadness subscales). Child sadness inhibition (CEMS sadness inhibition) was significantly and negatively correlated with MD of the CB ($r = -.40$, $p = .006$; Figure 1c; Table S3). No other significant correlations were observed at our a priori statistical threshold. Follow-up analyses with anterior and posterior CB segments indicated that MD of the right posterior CB ($r = -.45$, $p = .002$), left posterior CB ($r = -.33$, $p = .028$), and right anterior CB ($r = -.32$, $p = .033$) was significantly associated with child sadness inhibition (Table S4).

Given these findings, regression analyses were conducted to examine unique associations of child sadness inhibition with child CB MD after accounting for variance associated with child sex, monthly income, trauma exposure (TESI total), as well as age, given that age was correlated with CB connectivity; correlations among model variables

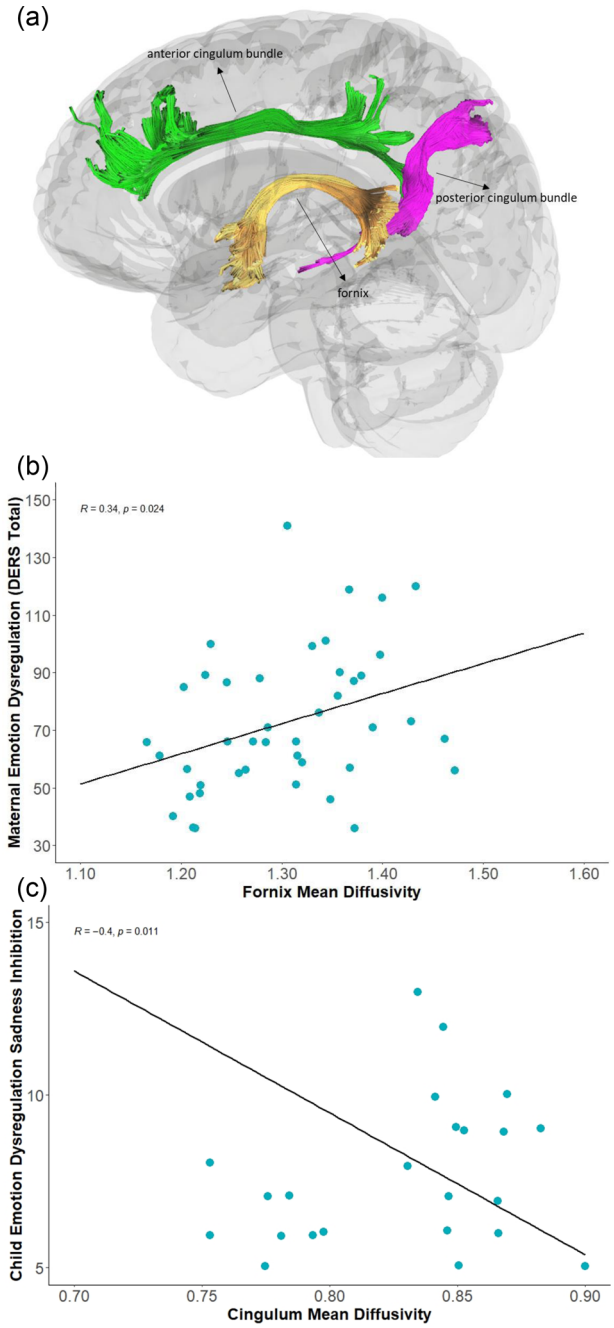

FIGURE 1 (a) Deterministic reconstructions of the cingulum bundle (anterior segment displayed in green, posterior segment displayed in magenta) and fornix (displayed in yellow) of a child's brain. (b) Greater maternal emotion dysregulation (DERS total) corresponds with lower child fornix connectivity (mean diffusivity; $r = .34$, $p = .024$). (c) Greater child sadness inhibition (CEMS sadness inhibition) corresponds with greater child cingulum bundle connectivity (mean diffusivity; $r = -.4$, $p = .011$)

are provided in Table S5 and regressions in Table S6. When separated by hemisphere and by section (anterior/posterior), the predictors accounted for 32% of the variance in child right posterior CB ($R = .56$; $p = .022$) and 29% of the variance in the MD of the child's left posterior CB ($R = .52$; $p = .041$). However, the individual predictors for these models were not statistically significant; child sadness inhibition and child age demonstrated the highest β values.

Lastly, given the correlations of children's CEMS with children's white matter connectivity, CEMS dysregulation total, CEMS inhibition total, and DERS total were included as independent variables to predict the MD of the fornix in a multivariate linear regression analysis (Table S7). The overall model was statistically significant ($F = 4.99$, $p = .005$) and accounted for 28% of the variance in MD of the right fornix; only DERS total was a significant predictor in the model ($\beta = .49$, $p = .003$).

4 | DISCUSSION

The primary objective of this study was to examine associations between maternal emotion regulation difficulties and child white matter connectivity in a sample of Black American mothers and their children. Secondarily, we examined how child emotion regulation difficulties related to child white matter connectivity. We observed that children of mothers who reported more difficulties in emotion regulation demonstrated lesser connectivity of the fornix. Even after accounting for environmental (economic disadvantage, trauma) and other factors that may affect white matter connectivity, maternal difficulties in emotion regulation remained the most robust predictor of child fornix connectivity, particularly for the right fornix. Specifically, maternal impulse control dysregulation and difficulty engaging in goal-directed behaviors in the presence of negative emotions were most strongly associated with fornix connectivity in their children. Further, we observed that parent-reported sadness inhibition in children was negatively correlated with MD of the CB, particularly posterior segments, indicating that greater sadness inhibition was associated with greater connectivity of this pathway.

Our findings indicate that disruptions in white matter connectivity of the child's fornix assessed via MD were linked to maternal difficulties in emotion regulation, particularly impulsivity. MD reflects the average of water molecule mobility in axons, equivalent to the mean of the three eigenvalues of the diffusion tensor (Salat, 2014). Higher MD values have been suggestive of demyelination (Abdel-Aziz & Ciccarelli, 2014; Caeyenberghs & Swinnen, 2015) in axons. Although the mechanism was not assessed in the current study, a possible cause of the white matter disruptions observed here is chronically increased glucocorticoid levels secondary to HPA axis activation (Eva et al., 2019; Myers et al., 2014) related to exposure to maternal emotion dysregulation. Glucocorticoids bind to the mineralocorticoid and glucocorticoid receptors in neurons, and oligodendrocytes that produce myelin sheaths contain both mineralocorticoid and glucocorticoid receptors (Bohn et al., 1991); these receptors are expressed throughout the brain, particularly in prefrontal and limbic regions implicated in emotion regulation. Glucocorticoids can affect neural plasticity by inhibiting the proliferation of oligodendrocyte predecessor cells in both white and gray matter (Alonso, 2000). Other chronic stressors, such as childhood maltreatment, are known to impact white matter plasticity, particularly connectivity of the fornix (Choi et al., 2009; McCarthy-Jones

et al., 2018; Lim et al., 2020). Maternal emotion regulation difficulties could likewise represent a chronic stressor that impacts white matter plasticity in this region via HPA axis activation. These effects may be particularly potent in the peripubertal period, during which chronic stress appears to have a more prominent impact (Fani et al., 2021; Callaghan & Tottenham, 2016; Gur et al., 2019; Sumner et al., 2019).

Maternal emotion regulation difficulties may be a particularly impactful stressor to children given that mothers are often their primary attachment figure, the person with whom a child interacts most during development. Emotion regulation in mothers affects attachment with their children (Brake et al., 2020); a child's perception of frequent maternal emotion disruption can influence feelings of instability, leading to an insecure attachment style characterized by anxious or avoidant behaviors. Insecure attachment can influence a child's ability to self-soothe and manage emotional distress; disruptions in these abilities can increase the risk for the development of mood disruptions later in life (Crow et al., 2021). Adaptive emotion regulation is associated with positive parenting practices, and conversely, emotion regulation difficulties are associated with negative parenting practices (Buckholdt et al., 2014; Lorber, 2012; Jones et al., 2014; Mazursky-Horowitz et al., 2014). Thus, maternal difficulties in emotion regulation can pose a significant stressor to children, triggering chronic HPA axis activation through negative interactions and insecure attachment. This, in turn, can lead to changes in white matter microarchitecture, potentially resulting in fornix demyelination over time.

The fornix is the major output fiber tract projecting from the hippocampus to the anterior aspects of the brain. After reaching the anterior commissure, it projects downward, connecting to the hypothalamus, thalamus, ventral striatum, cingulate cortex, and nucleus accumbens (Kazlouski et al., 2011). The nucleus accumbens is a striatal region critical for motivation and the experience of pleasure/reward (Salgado & Kaplitt, 2015). The fornix also plays an important role in emotion regulation, memory, and hippocampal–accumbens-dependent reward processing (Dalgleish, 2004). Fornix lesions have been associated with reward-learning impairments (Gaffan et al., 1984) and disruptions in fornix connectivity have been linked to reward deficits, including anhedonia in the context of PTSD and apathy in the aftermath of ischemic stroke (Harnett et al., 2021; Klimiec-Moskal et al., 2021). Parents are a major source of reward and reward learning throughout their children's development. Rewards such as praise, gifts, and positive interactions, or the lack thereof, likely influence children's expectations of future reward, and similarly, motivation to seek reward. As such, maternal emotion regulation difficulties may affect reward-learning processes in children; however, given that we did not assess reward processing in children, this is merely speculative. Additional research using reward-related tasks is needed to confirm these results.

Further, maternal difficulty engaging in goal-directed behavior in the presence of negative emotion and controlling impulses demonstrated the strongest negative associations with child fornix connectivity. These emotion regulation difficulties may affect a mother's ability to fully attend to her child in the presence of intense negative emotion or similarly may lead to negative and/or inconsistent reactions/behaviors in situations when a child is expecting praise/reward. Such interactions can lead to chronic HPA axis activation, gradually affecting fornix connectivity. Lastly, childhood trauma and economic disadvantage were also negatively associated with child fornix connectivity. This finding is consistent with past literature, which indicates that early childhood adversity is associated with disruptions in white matter connectivity in the fornix and other brain regions, suggesting the contributions of other environmental factors on white matter development beyond mother–child interactions (Choi et al., 2009; McCarthy-Jones et al., 2018; Lim et al., 2020; Yu et al., 2017). Considering this literature, and the fact that our sample consists of Black Americans who face chronic adversity and disadvantage, it is particularly meaningful that maternal emotion dysregulation had the strongest associations with fornix connectivity. These data demonstrate that, even in the context of other significant adversity/disadvantage, maternal–child interactions play a salient role in child neural plasticity.

We also observed that greater sadness inhibition in children was negatively associated with MD of the CB. The CB links the frontal, parietal, and medial temporal regions, with aspects proximal to the hippocampus/parahippocampus (Versace et al., 2015). The CB has various roles in emotion regulation, executive control, and episodic memory (Bubb et al., 2018). Abnormalities in CB connectivity and microstructure have been found across psychiatric disorders, including major depressive disorder, obsessive–compulsive disorder, posttraumatic stress disorder, and schizophrenia (Bubb et al., 2018). Our findings indicate that effortful inhibition of sadness in children corresponded with relatively enhanced connectivity of the CB, which has been involved with inhibitory processes. Inhibition of other emotions, specifically conditioned fear, has been associated with the structural integrity of the CB, which in turn affects the functional connectivity of regions implicated in conditioned fear extinction such as the hippocampus, the PFC, and amygdala (Fani et al., 2015). The present findings extend this research to suggest that the CB may be implicated in the inhibition of other negative emotions as well, including sadness (He et al., 2021). Negative correlations between negative emotionality traits and FA of the CB have been previously observed, indicating that reduced white matter integrity of this pathway is associated with stronger experience of negative emotions (McIntosh et al., 2013). From a functional perspective, the posterior cingulate cortex is activated in the presence of stimuli with sad valence, including sad films and sad facial images (Eugène et al., 2003; Goldin et al., 2005; Habel et al., 2005). However, no other research to date has linked CB connectivity with sadness inhibition. Our findings of greater CB connectivity in association with stronger sadness inhibition in children extend this earlier research, implicating this pathway in negative emotion inhibition more generally. Although we did not observe a relationship between maternal difficulties in emotion regulation and sadness inhibition in children, future studies with larger sample sizes are warranted to investigate this potential relationship.

We acknowledge several limitations to this study. Our sample size likely limited statistical power to detect more subtle associations between maternal emotion regulation and child white matter connectivity in other pathways linked to emotion regulation. The

cross-sectional design precluded our ability to make causal inferences about the relationship between maternal emotion regulation difficulties and child white matter connectivity. Additionally, given that children in this peripubertal period are known to demonstrate significant neural plasticity (Laube et al., 2020), the strength and direction of these findings may change in children assessed at a later age; as such, longitudinal assessment can provide a more complete understanding of the trajectory of these associations across later stages of development. Another limitation is the collection of clinical and neuroimaging data across different days, which may introduce a source of error variance in findings. Further, we did not have sufficient data in mothers to compare white matter connectivity in these pathways in mother–child dyads and assess relationships with other factors, such as genetics; this merits consideration in future research.

In sum, in this sample of Black American mothers and their children, we observed that maternal emotion regulation difficulties, particularly, difficulties with goal-directed behavior in the presence of negative emotion and controlling impulses, were associated with lower fornix connectivity in children. These associations were stronger than those related to other types of adversity, including trauma exposure and economic disadvantage. Given the role of the fornix in reward learning, it is possible that these aspects of maternal emotion dysregulation may affect appetitive processes in children via effects on white matter plasticity. These findings suggest specific links between maternal emotion regulation difficulties and white matter development in children, representing a pathway through which maternal difficulties in emotion regulation may impact emotion regulation processes in children.

ACKNOWLEDGMENTS

This study was supported by NIH funding (MH100122 and MH111682 to TJ, HD071982 to BB, AT011267 to NF) as well as the Emory Medical Care Foundation and Emory University Research Foundation. We would like to thank Rebecca Hinrichs, Allen Graham, Angelo Brown, Alexander Vance, Ye Ji Kim, Dorthie Cross, Vasiliki Michopoulos, and the many staff and volunteers of the Grady Trauma Project for their assistance with the conduct of this study. The data that support the findings of this study are available from the corresponding author upon reasonable request.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

DATA AVAILABILITY STATEMENT

De-identified aggregate data will be available upon request.

ORCID

Chenyang Wang https://orcid.org/0000-0003-1640-2277
Anais F. Stenson https://orcid.org/0000-0002-8214-8580
Negar Fani https://orcid.org/0000-0002-7720-252X

REFERENCES

Abdel-Aziz, K., & Ciccarelli, O. (2014). Rationale for quantitative MRI of the human spinal cord and clinical applications. In J. Cohen-Adad & C. A. M. Wheeler-Kingshott (Eds.), *Quantitative MRI of the spinal cord* (pp. 3–21). Academic Press. https://doi.org/10.1016/b978-0-12-396973-6.00001-0

Åhs, F., Kragel, P. A., Zielinski, D. J., Brady, R., & Labar, K. S. (2015). Medial prefrontal pathways for the contextual regulation of extinguished fear in humans. *Neuroimage*, *122*, 262–271. https://doi.org/10.1016/j.neuroimage.2015.07.051

Alonso, G. (2000). Prolonged corticosterone treatment of adult rats inhibits the proliferation of oligodendrocyte progenitors present throughout white and gray matter regions of the brain. Glia, *31*(3), 219–231. https://doi.org/10.1002/1098-1136(200009)31:3<219::aid-glia30>3.0.co;2-r

Are, F., & Shaffer, A. (2016). Family emotion expressiveness mediates the relations between maternal emotion regulation and child emotion regulation. *Child Psychiatry & Human Development*, *47*(5), 708–715. https://doi.org/10.1007/s10578-015-0605-4

Barnes, L., Duarte, A., & Sheridan, M. (2021). Toward a more representative brain: The importance and absence of diversity in human neuroscience research across the lifespan. *Frontiers in Human Nueroscience*. https://doi.org/10.3389/978-2-88966-883-0

Barysheva, M., Jahanshad, N., Foland-Ross, L., Altshuler, L. L., & Thompson, P. M. (2013). White matter microstructural abnormalities in bipolar disorder: A whole brain diffusion tensor imaging study. *NeuroImage: Clinical*, *2*, 558–568. https://doi.org/10.1016/j.nicl.2013.03.016

Baur, V., Hänggi, J., Rufer, M., Delsignore, A., Jäncke, L., Herwig, U., & Beatrix Brühl, A. (2011). White matter alterations in social anxiety disorder. *Journal of Psychiatric Research*, *45*(10), 1366–1372. https://doi.org/10.1016/j.jpsychires.2011.05.007

Bick, J., Zhu, T., Stamoulis, C., Fox, N. A., Zeanah, C., & Nelson, C. A. (2015). Effect of early institutionalization and foster care on long-term white matter development. *JAMA Pediatrics*, *169*(3), 211–219. https://doi.org/10.1001/jamapediatrics.2014.3212

Bohn, M. C., Howard, E., Vielkind, U., & Krozowski, Z. (1991). Glial cells express both mineralocorticoid and glucocorticoid receptors. *The Journal of Steroid Biochemistry and Molecular Biology*, *40*(1-3), 105–111. https://doi.org/10.1016/0960-0760(91)90173-3

Bradley, B., Defife, J. A., Guarnaccia, C., Phifer, J., Fani, N., Ressler, K. J., & Westen, D. (2011). Emotion dysregulation and negative affect. *The Journal of Clinical Psychiatry*, *72*(05), 685–691. https://doi.org/10.4088/jcp.10m06409blu

Brake, E., Berle, D., Reilly, N. M., & Austin, M. (2020). The relationship between emotion dysregulation and postnatal attachment in women admitted to a mother baby unit. *Australian Journal of Psychology*, *72*(3), 283–292. https://doi.org/10.1111/ajpy.12289

Bubb, E. J., Metzler-Baddeley, C., & Aggleton, J. P. (2018). The cingulum bundle: Anatomy, function, and dysfunction. *Neuroscience & Biobehavioral Reviews*, *92*, 104–127. https://doi.org/10.1016/j.neubiorev.2018.05.008

Buckholdt, K. E., Parra, G. R., & Jobe-Shields, L. (2014). Intergenerational transmission of emotion dysregulation through parental invalidation of emotions: Implications for adolescent internalizing and externalizing behaviors. *Journal of Child and Family Studies*, *23*(2), 324–332.

Caeyenberghs, K., & Swinnen, S. P. (2015). Neural correlates of motor deficits in young patients with traumatic brain injury. *Brain Mapping*, *2*, 461–468. https://doi.org/10.1016/b978-0-12-397025-1.00025-7

Callaghan, B. L., & Tottenham, N. (2016). The stress acceleration hypothesis: Effects of early-life adversity on emotion circuits and behavior. *Current Opinion in Behavioral Sciences*, *7*, 76–81. https://doi.org/10.1016/j.cobeha.2015.11.018

Chahal, R., Kirshenbaum, J. S., Ho, T. C., Mastrovito, D., & Gotlib, I. H. (2021). Greater age-related changes in white matter morphometry following early life stress: Associations with internalizing problems in adolescence. *Developmental Cognitive Neuroscience*, *47*, 100899. https://doi.org/10.1016/j.dcn.2020.100899

Carter, S., Gibbons, F., & Beach, S. (2021). Measuring the biological embedding of racial trauma among Black Americans utilizing the RDoC

approach. *Development and Psychopathology*, *33*(5), 1849–1863. https://doi.org/10.1017/S0954579421001073

Choi, J., Jeong, B., Rohan, M. L., Polcari, A. M., & Teicher, M. H. (2009). Preliminary evidence for white matter tract abnormalities in young adults exposed to parental verbal abuse. *Biological Psychiatry*, *65*(3), 227–234. https://doi.org/10.1016/j.biopsych.2008.06.022

Cole, M. W., & Schneider, W. (2007). The cognitive control network: Integrated cortical regions with dissociable functions. *Neuroimage*, *37*(1), 343–360. https://doi.org/10.1016/j.neuroimage.2007.03.071

Cox, J., & Witten, I. B. (2019). Striatal circuits for reward learning and decision-making. *Nature Reviews Neuroscience*, *20*(8), 482–494. https://doi.org/10.1038/s41583-019-0189-2

Crow, T. M., Levy, K. N., Bradley, B., Fani, N., & Powers, A. (2021). The roles of attachment and emotion dysregulation in the association between childhood maltreatment and PTSD in an inner-city sample. *Child Abuse & Neglect*, *118*, 105139. https://doi.org/10.1016/j.chiabu.2021.105139

Cutuli, D. (2014). Cognitive reappraisal and expressive suppression strategies role in the emotion regulation: An overview on their modulatory effects and neural correlates. *Frontiers in Systems Neuroscience*, *8*, 175. https://doi.org/10.3389/fnsys.2014.00175

Dalgleish, T. (2004). Cognitive approaches to posttraumatic stress disorder: The evolution of multirepresentational theorizing. *Psychological Bulletin*, *130*(2), 228–260. https://doi.org/10.1037/0033-2909.130.2.228

Denham, S. A., Caverly, S., Schmidt, M., Blair, K., Demulder, E., Caal, S., Hamada, H., & Mason, T. (2002). Preschool understanding of emotions: Contributions to classroom anger and aggression. *Journal of Child Psychology and Psychiatry*, *43*(7), 901–916. https://doi.org/10.1111/1469-7610.00139

Etkin, A., Egner, T., & Kalisch, R. (2011). Emotional processing in anterior cingulate and medial prefrontal cortex. *Trends in Cognitive Sciences*, *15*(2), 85–93. https://doi.org/10.1016/j.tics.2010.11.004

Eugène, F., Lévesque, J., Mensour, B., Leroux, J.-M., Beaudoin, G., Bourgouin, P., & Beauregard, M. (2003). The impact of individual differences on the neural circuitry underlying sadness. *Neuroimage*, *19*(2), 354–364. https://doi.org/10.1016/s1053-8119(03)00121-6

Eva, J., Mahfouz, A., Lisa, L., Houtman, R., Hazel, K. J., & Onno (2019). Corticosteroid action in the brain: The potential of selective receptor modulation. *Neuroendocrinology*, *109*(3), 266–276. https://doi.org/10.1159/000499659

Fani, N., King, T. Z., Brewster, R., Srivastava, A., Stevens, J. S., Glover, E. M., Norrholm, S. D., Bradley, B., Ressler, K. J., & Jovanovic, T. (2015). Fear-potentiated startle during extinction is associated with white matter microstructure and functional connectivity. *Cortex; A Journal Devoted to the Study of the Nervous System and Behavior*, *64*, 249–259. https://doi.org/10.1016/j.cortex.2014.11.006

Fani, N., Stenson, A. F., Rooij, S. J. H., La Barrie, D. L., & Jovanovic, T. (2021). White matter microstructure in trauma-exposed children: Associations with pubertal stage. *Developmental Science*, *24*(6), e13120. https://doi.org/10.1111/desc.13120

Fujie, S., Namiki, C., Nishi, H., Yamada, M., Miyata, J., Sakata, D., Sawamoto, N., Fukuyama, H., Hayashi, T., & Murai, T. (2008). The role of the uncinate fasciculus in memory and emotional recognition in amnestic mild cognitive impairment. *Dementia and Geriatric Cognitive Disorders*, *26*, 432–439. https://doi.org/10.1159/000165381

Gaffan, D., Saunders, R. C., Gaffan, E. A., Harrison, S., Shields, C., & Owen, M. J. (1984). Effects of fornix transection upon associative memory in monkeys: Role of the hippocampus in learned action. *The Quarterly Journal of Experimental Psychology Section B*, *36*(3b), 173–221. https://doi.org/10.1080/14640748408402203

Ghosh-Ippen, C., Ford, J., Racusin, R., Acker, M., Bosquet, K., Rogers, C., & Edwards, J. (2002). *Trauma events screening inventory—Parent report revised*. The Child Trauma Research Project of the Early Trauma Network and The National Center for PTSD Dartmouth Child Trauma Research Group.

Goldin, P. R., Hutcherson, C. A. C., Ochsner, K. N., Glover, G. H., Gabrieli, J. D. E., & Gross, J. J. (2005). The neural bases of amusement and sadness: A comparison of block contrast and subject-specific emotion intensity regression approaches. *Neuroimage*, *27*(1), 26–36. https://doi.org/10.1016/j.neuroimage.2005.03.018

Govindan, R. M., Behen, M. E., Helder, E., Makki, M. I., & Chugani, H. T. (2010). Altered water diffusivity in cortical association tracts in children with early deprivation identified with Tract-Based Spatial Statistics (TBSS). *Cerebral Cortex*, *20*(3), 561–569. https://doi.org/10.1093/cercor/bhp122

Gratz, K. L., & Roemer, L. (2004). Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the difficulties in emotion regulation scale. *Journal of Psychopathology and Behavioral Assessment*, *26*(1), 41–54. https://doi.org/10.1023/b:joba.0000007455.08539.94

Gur, R. E., Moore, T. M., Rosen, A. F. G., Barzilay, R., Roalf, D. R., Calkins, M. E., Ruparel, K., Scott, J. C., Almasy, L., Satterthwaite, T. D., Shinohara, R. T., & Gur, R. C. (2019). Burden of environmental adversity associated with psychopathology, maturation, and brain behavior parameters in youths. *JAMA Psychiatry*, *76*(9), 966–975. https://doi.org/10.1001/jamapsychiatry.2019.0943

Habel, U., Klein, M., Kellermann, T., Shah, N. J., & Schneider, F. (2005). Same or different? Neural correlates of happy and sad mood in healthy males. *Neuroimage*, *26*(1), 206–214. https://doi.org/10.1016/j.neuroimage.2005.01.014

Hallion, L. S., Steinman, S. A., Tolin, D. F., & Diefenbach, G. J. (2018). Psychometric properties of the Difficulties in Emotion Regulation Scale (DERS) and its short forms in adults with emotional disorders. *Frontiers in Psychology*, *9*, 539. https://doi.org/10.3389/fpsyg.2018.00539

Hanson, J. L., Adluru, N., Chung, M. K., Alexander, A. L., Davidson, R. J., & Pollak, S. D. (2013). Early neglect is associated with alterations in white matter integrity and cognitive functioning. *Child Development*, *84*(5), 1566–1578. https://doi.org/10.1111/cdev.12069

Harnett, N. G., Ference, E. W., & Knight, A. J., & Knight, D. C. (2018). White matter microstructure varies with post-traumatic stress severity following medical trauma. *Brain Imaging and Behavior*, *14*(4), 1012–1024. https://doi.org/10.1007/s11682-018-9995-9

Harnett, N. G., Stevens, J. S., Rooij, S. J. H., Ely, T. D., Michopoulos, V., Hudak, L., Jovanovic, T., Rothbaum, B. O., Ressler, K. J., & Fani, N. (2021). Multimodal structural neuroimaging markers of risk and recovery from posttrauma anhedonia: A prospective investigation. *Depression and Anxiety*, *38*(1), 79–88. https://doi.org/10.1002/da.23104

He, Y., Li, K., Li, J., Wang, J., Cheng, N., Xiao, J., & Jiang, T. (2021). Cingulum white matter integrity as a mediator between harm avoidance and hostility. *Neuroscience*, *461*, 36–43. https://doi.org/10.1016/j.neuroscience.2021.02.031

Helmeke, C., Seidel, K., Poeggel, G., Bredy, T. W., Abraham, A., & Braun, K. (2009). Paternal deprivation during infancy results in dendrite- and time-specific changes of dendritic development and spine formation in the orbitofrontal cortex of the biparental rodent *Octodon degus*. *Neuroscience*, *163*(3), 790–798. https://doi.org/10.1016/j.neuroscience.2009.07.008

Hofmann, S. G., Sawyer, A. T., Fang, A., & Asnaani, A. (2012). Emotion dysregulation model of mood and anxiety disorders. *Depression and Anxiety*, *29*(5), 409–416. https://doi.org/10.1002/da.21888

Huang, H., Gundapuneedi, T., & Rao, U. (2012). White matter disruptions in adolescents exposed to childhood maltreatment and vulnerability to psychopathology. *Neuropsychopharmacology*, *37*(12), 2693–2701. https://doi.org/10.1038/npp.2012.133

Ivy, A. S., Rex, C. S., Chen, Y., Dube, C., Maras, P. M., Grigoriadis, D. E., Gall, C. M., Lynch, G., & Baram, T. Z. (2010). Hippocampal dysfunction and cognitive impairments provoked by chronic early-life stress involve excessive activation of CRH receptors. *Journal of Neuroscience*, *30*(39), 13005–13015. https://doi.org/10.1523/jneurosci.1784-10.2010

John, O. P., & Gross, J. J. (2004). Healthy and unhealthy emotion regulation: Personality processes, individual differences, and life span

development. *Journal of Personality*, *72*(6), 1301–1334. https://doi.org/10.1111/j.1467-6494.2004.00298.x

Jones, J. D., Brett, B. E., Ehrlich, K. B., Lejuez, C. W., & Cassidy, J. (2014). Maternal attachment style and responses to adolescents' negative emotions: The mediating role of maternal emotion regulation. *Parenting*, *14*(3-4), 235–257. https://doi.org/10.1080/15295192.2014.972760

Kazlouski, D., Rollin, M. D. H., Tregellas, J., Shott, M. E., Jappe, L. M., Hagman, J. O., Pryor, T., Yang, T. T., & Frank, G. K. W. (2011). Altered fimbria-fornix white matter integrity in anorexia nervosa predicts harm avoidance. *Psychiatry Research: Neuroimaging*, *192*(2), 109–116. https://doi.org/10.1016/j.pscychresns.2010.12.006

Kerr, K. L., Ratliff, E. L., Cosgrove, K. T., Bodurka, J., Morris, A. S., & Kyle Simmons, W. (2019). Parental influences on neural mechanisms underlying emotion regulation. *Trends in Neuroscience and Education*, *16*, 100118. https://doi.org/10.1016/j.tine.2019.100118

Kim, P., Evans, G. W., Angstadt, M., Ho, S. S., Sripada, C. S., Swain, J. E., Liberzon, I., & Phan, K. L. (2013). Effects of childhood poverty and chronic stress on emotion regulatory brain function in adulthood. *Proceedings of the National Academy of Sciences of the United States of America*, *110*(46), 18442–18447. https://doi.org/10.1073/pnas.1308240110

Kirkinis, K., Pieterse, A. L., Martin, C., Agiliga, A., & Brownell, A. (2021). Racism, racial discrimination, and trauma: A systematic review of the social science literature. *Ethnicity & Health*, *26*(3), 392–412.

Klimiec-Moskal, E., Karcz, P., Kowalska, K., Slowik, A., Herman-Sucharska, I., & Dziedzic, T. (2021). Magnetisation transfer imaging revealed microstructural changes related to apathy symptoms after ischaemic stroke. *International Journal of Geriatric Psychiatry*, *36*(8), 1264–1273. https://doi.org/10.1002/gps.5520

Korgaonkar, M. S., Grieve, S. M., Koslow, S. H., Gabrieli, J. D. E., Gordon, E., & Williams, L. M. (2010). Loss of white matter integrity in major depressive disorder: Evidence using tract-based spatial statistical analysis of diffusion tensor imaging. *Human Brain Mapping*, *32*(12), 2161–2171. https://doi.org/10.1002/hbm.21178

Laube, C., Van Den Bos, W., & Fandakova, Y. (2020). The relationship between pubertal hormones and brain plasticity: Implications for cognitive training in adolescence. *Developmental Cognitive Neuroscience*, *42*, 100753. https://doi.org/10.1016/j.dcn.2020.100753

Liao, M., Yang, F., Zhang, Y., He, Z., Su, L., & Li, L. (2014). White matter abnormalities in adolescents with generalized anxiety disorder: A diffusion tensor imaging study. *BMC Psychiatry*, *14*(1), 41. https://doi.org/10.1186/1471-244x-14-41

Lim, L., Howells, H., Radua, J., & Rubia, K. (2020). Aberrant structural connectivity in childhood maltreatment: A meta-analysis. *Neuroscience & Biobehavioral Reviews*, *116*, 406–414. https://doi.org/10.1016/j.neubiorev.2020.07.004

Liu, J., Dietz, K., Deloyht, J. M., Pedre, X., Kelkar, D., Kaur, J., Vialou, V., Lobo, M. K., Dietz, D. M., Nestler, E. J., Dupree, J., & Casaccia, P. (2012). Impaired adult myelination in the prefrontal cortex of socially isolated mice. *Nature Neuroscience*, *15*(12), 1621–1623. https://doi.org/10.1038/nn.3263

Lorber, M. F. (2012). The role of maternal emotion regulation in overreactive and lax discipline. *Journal of Family Psychology*, *26*(4), 642–647. https://doi.org/10.1037/a0029109

Lupien, S. J., McEwen, B. S., Gunnar, M. R., & Heim, C. (2009). Effects of stress throughout the lifespan on the brain, behaviour and cognition. *Nature Reviews Neuroscience*, *10*(6), 434–445. https://doi.org/10.1038/nrn2639

Manelis, A., Soehner, A., Halchenko, Y. O., Satz, S., Ragozzino, R., Lucero, M., Swartz, H. A., Phillips, M. L., & Versace, A. (2021). White matter abnormalities in adults with bipolar disorder type-II and unipolar depression. *Scientific Reports*, *11*(1), 7541. https://doi.org/10.1038/s41598-021-87069-2

Mazursky-Horowitz, H., Felton, J. W., MacPherson, L., Ehrlich, K. B., Cassidy, J., Lejuez, C. W., & Chronis-Tuscano, A. (2014). Maternal emotion regulation mediates the association between adult attention-deficit/hyperactivity disorder symptoms and parenting. *Journal of Abnormal Child Psychology*, *43*(1), 121–131. https://doi.org/10.1007/s10802-014-9894-5

McCarthy-Jones, S., Oestreich, L. K. L., Lyall, A. E., Kikinis, Z., Newell, D. T., Savadjiev, P., Shenton, M. E., Kubicki, M., Pasternak, O., & Whitford, T. J. (2018). Childhood adversity associated with white matter alteration in the corpus callosum, corona radiata, and uncinate fasciculus of psychiatrically healthy adults. *Brain Imaging and Behavior*, *12*(2), 449–458. https://doi.org/10.1007/s11682-017-9703-1

McIntosh, A. M., Bastin, M. E., Luciano, M., Maniega, S. M., Valdes Hernández, M. C., Royle, N. A., Hall, J., Murray, C., Lawrie, S. M., Starr, J. M., Wardlaw, J. M., & Deary, I. J. (2013). Neuroticism, depressive symptoms and white-matter integrity in the Lothian Birth Cohort 1936. *Psychological Medicine*, *43*(6), 1197–1206. https://doi.org/10.1017/S003329171200150X

Morelen, D., Shaffer, A., & Suveg, C. (2016). Maternal emotion regulation. *Journal of Family Issues*, *37*(13), 1891–1916. https://doi.org/10.1177/0192513x14546720

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development*, *16*(2), 361–388. https://doi.org/10.1111/j.1467-9507.2007.00389.x

Muhammad, A., & Kolb, B. (2011). Maternal separation altered behavior and neuronal spine density without influencing amphetamine sensitization. *Behavioural Brain Research*, *223*(1), 7–16. https://doi.org/10.1016/j.bbr.2011.04.015

Myers, B., McKlveen, J. M., & Herman, J. P. (2014). Glucocorticoid actions on synapses, circuits, and behavior: Implications for the energetics of stress. *Frontiers in Neuroendocrinology*, *35*(2), 180–196. https://doi.org/10.1016/j.yfrne.2013.12.003

Oishi, K., Faria, A. V., Hsu, J., Tippett, D., Mori, S., & Hillis, A. E. (2014). Critical role of the right uncinate fasciculus in emotional empathy. *Annals of Neurology*, *77*(1), 68–74. https://doi.org/10.1002/ana.24300

Olson, I. R., Von Der Heide, R. J., Alm, K. H., & Vyas, G. (2015). Development of the uncinate fasciculus: Implications for theory and developmental disorders. *Developmental Cognitive Neuroscience*, *14*, 50–61.

Papagno, C., Miracapillo, C., Casarotti, A., Romero Lauro, L. J., Castellano, A., Falini, A., Casaceli, G., Fava, E., & Bello, L. (2010). What is the role of the uncinate fasciculus? Surgical removal and proper name retrieval. *Brain*, *134*(2), 405–414. https://doi.org/10.1093/brain/awq283

Pencea, I., Munoz, A. P., Maples-Keller, J. L., Fiorillo, D., Schultebraucks, K., Galatzer-Levy, I., Rothbaum, B. O., Ressler, K. J., Stevens, J. S., Michopoulos, V., & Powers, A. (2020). Emotion dysregulation is associated with increased prospective risk for chronic PTSD development. *Journal of Psychiatric Research*, *121*, 222–228. https://doi.org/10.1016/j.jpsychires.2019.12.008

Peters, S. K., Dunlop, K., & Downar, J. (2016). Cortico-striatal-thalamic loop circuits of the salience network: A central pathway in psychiatric disease and treatment. *Frontiers in Systems Neuroscience*, *10*, 104. https://doi.org/10.3389/fnsys.2016.00104

Phelps, E. A. (2004). Human emotion and memory: Interactions of the amygdala and hippocampal complex. *Current Opinion in Neurobiology*, *14*(2), 198–202. https://doi.org/10.1016/j.conb.2004.03.015

Phillips, M. L., Ladouceur, C. D., & Drevets, W. C. (2008). A neural model of voluntary and automatic emotion regulation: Implications for understanding the pathophysiology and neurodevelopment of bipolar disorder. *Molecular Psychiatry*, *13*(9), 833–857. https://doi.org/10.1038/mp.2008.65

Powers, A., Cross, D., Fani, N., & Bradley, B. (2015). PTSD, emotion dysregulation, and dissociative symptoms in a highly traumatized sample. *Journal of Psychiatric Research*, *61*, 174–179. https://doi.org/10.1016/j.jpsychires.2014.12.011

Powers, A., Hunnicutt, K., Stenson, A., Jovanovic, T., Kaslow, N., & Bradley, B. (2022). Associations between emotion dysregulation dimensions and parenting behaviors in trauma-exposed African American

mothers. *Child Maltreatment*, *27*(1), 43–52. https://doi.org/10.1177/1077559520988352

Salat, D. H. (2014). Diffusion tensor imaging in the study of aging and age-associated neural disease. In H. Johansen-Berg & T. E. J. Behrens (Eds.), *Diffusion MRI* (2nd ed., pp. 257–281). Academic Press. https://doi.org/10.1016/B978-0-12-396460-1.00012-3

Salgado, S., & Kaplitt, M. G. (2015). The nucleus accumbens: A comprehensive review. *Stereotactic and Functional Neurosurgery*, *93*(2), 75–93. https://doi.org/10.1159/000368279

Seeley, W. W., Menon, V., Schatzberg, A. F., Keller, J., Glover, G. H., Kenna, H., Reiss, A. L., & Greicius, M. D. (2007). Dissociable intrinsic connectivity networks for salience processing and executive control. *Journal of Neuroscience*, *27*(9), 2349–2356. https://doi.org/10.1523/jneurosci.5587-06.2007

Sumner, J. A., Colich, N. L., Uddin, M., Armstrong, D., & McLaughlin, K. A. (2019). Early experiences of threat, but not deprivation, are associated with accelerated biological aging in children and adolescents. *Biological Psychiatry*, *85*(3), 268–278. https://doi.org/10.1016/j.biopsych.2018.09.008

Tan, P. Z., Oppenheimer, C. W., Ladouceur, C. D., Butterfield, R. D., & Silk, J. S. (2020). A review of associations between parental emotion socialization behaviors and the neural substrates of emotional reactivity and regulation in youth. *Developmental Psychology*, *56*(3), 516–527. https://doi.org/10.1037/dev0000893

Thompson, R. A. (1994). Emotion regulation: A theme in search of definition. *Monographs of the Society for Research in Child Development*, *59*(2-3), 25–52. https://doi.org/10.1111/j.1540-5834.1994.tb01276.x

Uddin, L. Q. (2017). *Salience network of the human brain*. Academic Press.

Versace, A., Acuff, H., Bertocci, M. A., Bebko, G., Almeida, J. R., Perlman, S. B., Leemans, A., Schirda, C., Aslam, H., Dwojak, A., Bonar, L., Travis, M., Gill, M. K., Demeter, C., Diwadkar, V. A., Sunshine, J. L., Holland, S. K., Kowatch, R. A., Birmaher, B., … Phillips, M. L. (2015). White matter structure in youth with behavioral and emotional dysregulation disorders: A probabilistic tractographic study. *JAMA Psychiatry*, *72*(4), 367–376. https://doi.org/10.1001/jamapsychiatry.2014.2170

Villalta, L., Smith, P., Hickin, N., & Stringaris, A. (2018). Emotion regulation difficulties in traumatized youth: A meta-analysis and conceptual review. *European Child & Adolescent Psychiatry*, *27*(4), 527–544. https://doi.org/10.1007/s00787-018-1105-4

Yu, S.-T., Lee, K.-S., & Lee, S.-H. (2017). Fornix microalterations associated with early trauma in panic disorder. *Journal of Affective Disorders*, *220*, 139–146. https://doi.org/10.1016/j.jad.2017.05.043

Zilverstand, A., Parvaz, M. A., & Goldstein, R. Z. (2017). Neuroimaging cognitive reappraisal in clinical populations to define neural targets for enhancing emotion regulation. A systematic review. *Neuroimage*, *151*, 105–116. https://doi.org/10.1016/j.neuroimage.2016.06.009

SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

How to cite this article: Wang, C., La Barrie, D. L., Powers, A., Stenson, A. F., van Rooij, S. J. H., Stevens, J. S., Jovanovic, T., Bradley, B., McGee, R. E., & Fani, N. (2022). Associations of maternal emotion regulation with child white matter connectivity in Black American mother–child dyads. *Developmental Psychobiology*, *64*, e22303. https://doi.org/10.1002/dev.22303