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RESEARCH ARTICLE

Emotion regulation and reactivity are associated with cortical thickness in early to mid-childhood

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Abstract

This study explored the neural correlates of emotion regulation and emotional reactivity in early to mid-childhood. A sample of 96 children (70% White, mid-to-high socioeconomic status) aged 3–8 years provided structural neuroimaging data and caregivers reported on emotion regulation and emotional reactivity. The amygdala, insula, inferior frontal gyrus, anterior cingulate cortex, and medial orbitofrontal cortex were explored as a priori regions of interest (ROIs). ROI analyses revealed that emotion regulation was positively associated with cortical thickness in the insula, whereas emotional reactivity was negatively associated with cortical thickness in the inferior frontal gyrus. Exploratory whole-brain analyses suggested positive associations between emotion regulation and both left superior temporal thickness and right inferior temporal thickness, as well as negative associations between emotional reactivity and left superior temporal thickness. There were no significant associations between emotional regulation or reactivity and amygdala volume or cortical surface area. These findings support the notion that surface area and cortical thickness are distinct measures of brain maturation. In sum, these findings suggest that children may rely on a wider set of neural regions for emotion regulation and reactivity than adults, which is consistent with theories of interactive specialization across the life span.

KEYWORDS
amygdala, cortical thickness, early childhood, emotion regulation, emotional reactivity, mid-childhood, surface area

1 | INTRODUCTION

Early socioemotional competencies predict a wide variety of outcomes, including mental health diagnoses, school attitudes, and overall adjustment across development (e.g., Calkins et al., 2002; Denham & Kochanoff, 2002; Eisenberg et al., 2001; Kochanska & Knaack, 2003; O'Neil et al., 1997). Socioemotional competence is built over time via emotional functioning. To fully understand social competence and its related outcomes, it is vital to understand emotional functioning across development (Calkins et al., 2002; Denham & Kochanoff, 2002; Eisenberg et al., 2001; Kochanska & Knaack, 2003; Maszk et al., 1999). For example, emotion dysregulation has been directly linked to various forms of psychopathology, such as anxiety, behavioral, and mood disorders (Calkins, 2021; Christensen et al., 2020; Ehring, 2013; Lewis et al., 2006; McLafferty et al., 2020; Millgram et al., 2020; Stieben et al., 2007). Improved understanding of individual differences in emotional functioning and their underlying neural mechanisms early in life is important, as this knowledge may inform interventions targeting emotional coping and ultimately improve various outcomes in children.

Emotion regulation and emotional reactivity are two components of emotional functioning (Eisenberg et al., 1993, 1994, 1995, 1996). Emotion regulation can best be understood as both internal and external processes that help modulate a child's emotional arousal and allow them to achieve their goals despite various situational demands (Cole et al., 2004; Diamond & Aspinwall, 2003; Gross & John, 2003). Emotion regulation is often conceptualized as lability, flexibility, and situational responsivity (Shields & Cicchetti, 1997). For example, if a child is offered a reward to stop crying, emotion regulation is their ability to downregulate their emotions in order to obtain their reward. Emotional reactivity, however, refers to the sensitivity, intensity, and persistence of emotional responses (Klonsky et al., 2019; Rothbart & Derryberry, 1981; Shields & Cicchetti, 1997). Children with high emotional reactivity may experience more intense, more frequent, and longer lasting feelings of emotion compared to others (Slagt et al., 2019). These individual differences in emotional reactivity are thought to derive primarily from temperamental differences (Murphy et al., 1999; Rothbart & Bates, 2007) but can also be tied to environmental factors like maternal mental health (Spry et al., 2020) or physiological health factors such as poor sleep (Gentzler et al., 2009).

Emotional regulation and reactivity have different developmental timelines. Early in life, infants rely on their caregivers for regulatory assistance (Spangler & Grossmann, 1993; Spangler et al., 1994). Preschool-age children begin making strides in independent emotion regulation; however, emotion regulation proficiencies continue to change across development based on different constraints and co-regulatory opportunities with social partners at different stages of life. In contrast, emotional reactivity is thought to be relatively stable across the life span due to its close ties to temperament (Rothbart & Bates, 2007).

Conceptually, emotion regulation and emotional reactivity may be related. While they are distinct constructs, they cannot be entirely separated from each other and likely influence each other. For example, Tracy et al. (2014) argued that decreased regulation skills might explain increased reactivity. However, it is important to note that this may not always be the case. A child could have high emotional reactivity but also demonstrate proficiencies in emotional regulation. Additionally, previous work has indicated potential sex differences in emotional expression, with girls being more likely to exhibit positive emotions, and in the use of different emotion regulation strategies (Chaplin & Aldao, 2013; Oattes et al., 2018; Sala et al., 2014).

While the preschool period is an important time for the development of self-regulatory strategies necessary for emotional functioning, it is also an essential period for brain development. Magnetic resonance imaging (MRI) measurements of brain volume increase dramatically during the first decade of life (Brown & Jernigan, 2012). The amygdala, for example, increases in volume through late adolescence (Giedd et al., 1996; Guo et al., 2007; Ostby et al., 2009), but the most substantial growth rate occurs during the first 10 years of life (Uematsu et al., 2012). Additionally, evidence suggests that there is continued neurogenesis and maturation of glial populations postnatally that are concurrent with regressive changes (i.e., tissue loss) associated with synaptic pruning (Brown & Jernigan, 2012).

Some of these neural changes can be explored via structural MRI by examining the volume of neural structures or cortical volume. Cortical thickness and surface area are two different measures thought to be reflective of cortical volume, with unique developmental trajectories (Raznahan et al., 2011; Wierenga et al., 2014). Cortical thickness measurements are believed to reflect dendritic arborization (i.e., the process through which neurons branch out to create new synapses) and pruning (i.e., eliminating redundant neurons and synaptic connections; Huttenlocher, 1990). In contrast, surface area measurements are thought to reflect folding and gyrification (i.e., the process through which sulci and gyri folding patterns develop on the surface of the brain; Chenn & Walsh, 2002; Rakic, 2009). Cortical thickness has been shown to decrease linearly from early childhood through adolescence, whereas surface area typically increases linearly until age 10, after which decreases begin to emerge and continue through adulthood (Brown & Jernigan, 2012; Sussman et al., 2016). Cortical thickness and surface area are valuable measurements of brain maturation in conjunction with volumetric data, as they have been shown to be more sensitive to individual differences (Hutton et al., 2009; Wallace et al., 2015).

The Model of The Cognitive Control of Emotion (MCCE) argues that brain regions related to emotional functioning can be separated into two categories—those that generate emotion and those that support emotion regulatory strategies (Ochsner et al., 2012). For example, the amygdala, ventral striatum, ventromedial prefrontal cortex (vmPFC), and insula have been linked to emotion generation in both humans and animals (Ochsner et al., 2012). In contrast, regions associated with emotion regulation include the prefrontal and cingulate cortices that are thought to support control processes that modulate activity in the aforementioned emotion generation regions (Ochsner & Gross, 2005; Ochsner et al., 2012). Additional work investigating neural bases of emotion regulation has suggested that the inferior frontal gyrus (IFG) (located within the prefrontal cortex) and several temporal cortical regions are associated with self-directed inner speech in adolescents and adults. This could create an emergent emotion regulation process involved in evaluating stimuli significance (Geva et al., 2011; Girbau, 2007; Jones & Fernyhough, 2007; Lieberman et al., 2011; Morin, 2011; Shergill et al., 2003). In summary, the MCCE suggests a top-down model where neural responses to emotional stimuli in the ventral striatum, amygdala, and insula are downregulated by prefrontal and potentially temporal regions (Kober et al., 2010; Phillips et al., 2008; Wager et al., 2008). Although work investigating the neural basis of emotional reactivity is limited, one might expect that the ventral striatum, amygdala, and insula might relate to emotional reactivity and how responsive one is to generated emotions.

Previous research has investigated both the structure and function of regions implicated in the MCCE. Although brain structure and function are not directly analogous measures, there are several reasons that prior research on both structural and functional correlates of emotion regulation may be relevant to this study. First, brain structure and functional networks show a symbiotic or highly interdependent relationship (Rubinov et al., 2009). Over fast time scales, structure can relate the emergence of complex neural networks (Basset et al., 2006). However, over slower time scales, neural functioning can reshape structure through activity-dependent dendritic development (Butz et al., 2009; Chklovskii et al., 2004; Knott & Holtmaat, 2008). Second, structural connectivity in the brain has been shown to relate to aspects of functional connectivity, specifically while the brain is in a resting state (Batista-García-Ramó & Fernández-Verdecia, 2018). Structural MRI is often more practical for younger populations prone to movement in the scanner environment and for whom completing tasks of emotion regulation is difficult. Thus, structural MRI measures allow us to investigate brain–behavior relations in populations, like young children, for whom functional magnetic resonance imaging (fMRI) is challenging or impossible. Even though structural and fMRI findings yield distinct neural measures, both are relevant to the present research. As such, we summarize below prior research using both structural and functional measures to investigate emotion regulation in children and adults.

MCCE theory has been well-documented in adult humans (e.g., Morawetz et al., 2016; Terasawa et al., 2011). Emotion generation has been linked to increased brain activation in the amygdala (McRae et al., 2012; Ochsner et al., 2009), whereas emotion regulation has been linked to activation of the prefrontal cortex (Johnstone et al., 2007; Urry et al., 2006). Specifically, Morawetz et al. (2020) identified four large-scale brain networks related to emotion regulation and emotion generation/appraisal. They suggested that convergent activation of several neural regions, including the IFG, right insula, dorsolateral prefrontal cortex, ventrolateral prefrontal cortex, cingulate gyrus, and medial temporal gyrus, was related to emotion regulation. In contrast, convergent activation of several neural regions, including the amygdala, fusiform gyrus, parahippocampal gyrus, left insula, and posterior cingulate cortex, was related to emotion generation (Morawetz et al., 2020). Of further interest is the anterior cingulate cortex (ACC) that connects the “emotional” limbic system to the “cognitive” prefrontal cortex and has therefore been suggested to mediate cognitive influences of emotion (Stevens et al., 2011). Structurally, individual differences in gray matter volume and cortical thickness in prefrontal and limbic structures are associated with emotion regulation skills in adolescents and adults (Ferschmann et al., 2021; Kuhn et al., 2011; Vijayakumar et al., 2014). Specifically, these strategies, such as expressive suppression, are related with larger volumes of the anterior insula and the dorsomedial prefrontal cortex (Giuliani et al., 2011a; Hermann et al., 2014; Kuhn et al., 2011). Strategies that involve the appraisal of emotion, which theoretically may happen during the generation stage of emotion outlined by the MCCE, are associated with larger ACC and amygdala volumes (Giuliani et al., 2011b; Hermann et al., 2014; Kuhn et al., 2011).

However, it is unclear if this top-down model of regulation is functioning at an earlier developmental age when frontal regions are less developed or if additional brain systems are activated during early to mid-childhood when both emotion regulation and neural structures are still developing. Research linking emotion regulation and neurodevelopment in childhood, specifically, is only just beginning to emerge. Pagliaccio et al. (2014) examined if brain structure could predict concurrent and longitudinal measures of emotion regulation skills in healthy school-age children. In their sample of 60 children (ages 8–12 years), they found that larger insula volume predicted better emotion regulation skills over approximately 18 months. In addition, in 105, 8- to 10-year-old children, Whittle et al. (2020) found that reduced cortical thickness of the medial orbitofrontal cortex (mOFC) was associated with higher internalizing symptoms. While internalizing symptoms and emotion regulation are separate constructs, greater internalizing symptoms, such as anxiety, depression, or withdrawal, are thought to relate to deficits in emotion processing and regulation (Whittle et al., 2020).

Despite studies demonstrating links between these regions of interest (ROIs) and emotion regulation, there are still gaps in the literature. For example, developmental studies have yet to investigate cortical variations in the regions posited by the MCCE. Additionally, studies have focused on investigating emotion regulation; thus, the neural bases of emotional reactivity in early to mid-childhood are still unclear. Emotion regulation is a broad construct composed of several competencies and strategies; accordingly, the neurobiology related to emotion is also complex. Therefore, the present study invesrtigated if four a priori ROIs (insula, IFG, ACC, and mOFC), chosen because of their being previously implicated in the MCCE in adult research, would relate to emotion regulation and emotional reactivity in a sample of 3- to 8-year-old children. Due to the novelty of this question, we initially explored relations of both emotion regulation and emotional reactivity with all ROIs, then conducted a whole-brain vertex-by-vertex investigation.

Hypotheses for associations with cortical thickness were formed on the known developmental trajectories (i.e., by this age point, cortical thickness will begin the thinning process, therefore we may assume that thinner cortex may be more developmentally mature cross-sectionally and therefore relate to a better ability to self-regulate). We predicted that reduced thickness in the insula, IFG, ACC, and mOFC would be associated with increased emotion regulation, and increased thickness in the ROIs would be associated with increased emotional reactivity. However, we did not predict the nature of relations of the surface area of the regions with emotion regulation and emotional reactivity due to the lack of literature linking surface area, specifically with emotion regulation and reactivity. After these analyses and a thorough literature review, we further investigated associations with the amygdala to be consistent with the MCCE model. A nonsignificant association between amygdala volume and emotion regulation had previously been investigated within a subset of this sample. However, it was unclear if this nonsignificant finding was due to a lack of statistical power or a true finding of developmental differences in the neural basis of emotion regulation. Therefore, we did not hypothesize about the nature of the association between amygdala volume and emotional functioning prior to the analyses for this study. Finally, we used an exploratory whole-brain analysis to investigate whether relations existed with cortical thickness or surface area of any additional brain regions.

TABLE 1 Demographic information.

Variable name		Study 1 (*N* = 82)	Study 2 (*N* = 20)	Combined (*N* = 102)
Child age in years, *M* (*SD*)		6.543 (1.276)	3.975 (0.426)	6.040 (1.546)
Child race				
	Asian	3.70%	0.00%	2.90%
	White	65.90%	90.00%	70.60%
	Black	7.30%	5.00%	6.90%
	Multiracial	19.50%	5.00%	16.70%
	Did not disclose	3.70%	0.00%	2.90%
Child ethnicity				
	Hispanic/Latino	7.30%	10.00%	7.80%
Responding parents' education (%) with at least a 4-year college degree		80.50%	85.00%	81.40%
Nonresponding parents' education (%) with at least a 4-year college degree		79.30%	80.00%	80.20%
Household income (%) higher than $75,000		87.80%	85.00%	89.00%
Responding parent occupational prestige, *M* (*SD*)		5.281 (1.958)	3.053 (1.433)	4.861 (2.059)
Nonresponding parents' occupational prestige, *M* (*SD*)		5.074 (1.311)	3.737 (0.991)	4.820 (1.359)

2 | METHOD

2.1 | Participants

The current study used a combined dataset drawn from two larger longitudinal studies. Study 1 investigated memory and brain development in 4- to 8-year-old children and yielded a subsample of 82 children (47 female, *M* = 6.543, *SD* = 1.276). Study 2 investigated sleep-dependent memory development in 3- to 4-year-old children and yielded a subsample of 20 children (11 female, *M* = 3.975, *SD* = 0.426). Inclusion and exclusion criteria for both studies were identical with two exceptions: (1) the target ages of children in the studies and (2) since Study 2 was focused on sleep-dependent memory development, children with a history of sleep problems or disorders were excluded from that sample. We combined these datasets to increase statistical power and encompass an early to mid-childhood age range. Detailed descriptions of demographics for both studies and the combined sample are included in Table 1.

Individuals included in the present report were selected if they had data for the Emotion Regulation Checklist (ERC) and structural MRI. No prior publication with this sample has explored relations between the variables included here (i.e., ERC, amygdala volume, cortical thickness, and cortical surface area), although see Fitter et al. (2022) for a study exploring relations between attachment security and brain structure from this sample.

Prior to data collection, all methods were approved by the University's Institutional Review Board. Participants were recruited through a faculty-maintained database of families interested in participating in research, flyers, and online advertisements. Screening involved parents completing a short questionnaire either online or over the phone to ensure they met inclusion criteria. Inclusion criteria required the child to be between 3 and 8 years of age and typically developing. Exclusion criteria included a history of head trauma, abnormal circadian function, history of a brain abnormality, a neurological disorder, premature birth, a diagnosis of ADHD or other learning disability, a diagnosis of a psychiatric disorder, a history of a developmental delay or disorder, a family history of autism spectrum disorder, metal in the body, or any other contraindications for MRI.

Six of the acquired T1 scans were deemed unusable due to motion artifacts (such as significant banding). Thus, the final combined sample included 96 children (55 female, *M* = 6.114 years, *SD* = 1.514).

2.2 | Measures

2.2.1 | Emotion regulation and reactivity

Emotion regulation and reactivity were assessed through the 24-item ERC (Shields & Cicchetti, 1997). The ERC has previously been validated for assessing emotion regulatory processes in children (Cohen & Mendez, 2009; Graziano et al., 2007; Izard et al., 2008). Parents completed the checklist electronically at the time of the study. Included in the checklist are both positively and negatively weighted items on a 4-point Likert scale (1 = *Rarely/Never*, 2 = *Sometimes*, 3 = *Often*, and 4 = *Almost Always*). Example items include "Exhibits wide mood swings" and "Is a cheerful child."

The Emotion Regulation subscale indexes situationally appropriate affective displays, emotional self-awareness, and empathy (Shields & Cicchetti, 1997). The subscale includes eight items (Cronbach's alpha = .83), such as "displays appropriate negative affect in response to hostile, aggressive, or intrusive acts by peers" and "can say when s/he is feeling angry" (Shields & Cicchetti, 1997). The Emotional Lability and Negativity subscale (i.e., Emotional Reactivity) indexes a lack of flexibility, mood lability, and dysregulated negative affect (Shields & Cicchetti, 1997). It includes 16 items (Cronbach's alpha = .96), such as "is easily frustrated" and "exhibits wide mood swings" (Shields & Cicchetti, 1997).

TABLE 2 Descriptive statistics for study variables ($n = 96$).

	Variable name	*M* (*SD*)	Minimum	Maximum
Emotion Regulation Checklist	Emotion regulation	0.409 (0.075)	0.22	0.5
	Negativity/lability	0.129 (0.031)	0.07	0.2
Cortical thickness	IFG	3.123 (0.165)	2.75	3.54
	ACC	3.355 (0.184)	2.94	3.88
	mOFC	3.027 (0.175)	2.6	3.42
	Insula	3.545 (0.142)	3.25	3.91
Surface area	IFG	1217.411 (149.306)	834	1572.83
	ACC	685.629 (113.469)	442.75	1048
	mOFC	1774.184 (224.406)	1276	2329
	Insula	2127.121 (260.721)	1682.5	3059
Volume	Amygdala	1534.879 (187.683)	1149.15	2008.5

Abbreviations: ACC, anterior cingulate cortex; IFG, inferior frontal gyrus; mOFC, medial orbitofrontal cortex.

Subscales were calculated so that higher scores reflected greater emotional reactivity and better emotional regulation. Scores were averaged for each individual subscale if the parent had completed 75% or more of the required questions in the scale. Means and standard deviations are included in Table 2.

2.2.2 | MRI acquisition

To prepare for the MRI scan, children first took part in a mock scan in which they practiced lying still and were given motion feedback by an experimenter. The children then completed the MRI scan. Padding was placed around the child's head to minimize head movements and mitigate the effects of motion. Participants were scanned in a Siemens 3.0T scanner (MAGNETOM Trio Tim System; Siemens Medical Solutions) with a 32-channel coil. Children were permitted to watch a movie of their choice during the scan to enhance compliance. A standard-resolution (0.9 mm^3) T1-magnetization-prepared rapid gradient-echo (MPRAGE) sequence consisting of 176 contiguous sagittal slices (0.9 mm isotropic; 1900 ms repetition time; 2.32 ms echo time; 900 ms inversion time; 9° flip angle; pixel matrix = 256 × 256) was acquired. If the image quality was deemed to be too low (based on visual inspection of banding or a significantly blurred image), the scan was repeated when possible ($n = 4$).

2.2.3 | MRI analysis

Standard procedures, including cortical surface reconstruction, and cortical and subcortical segmentation, were conducted using FreeSurfer versions 5.1 and 6.0 for Study 1 and Study 2, respectively.[1] FreeSurfer is a standard automatic segmentation program (surfer.nmr.mgh.harvard.edu; Fischl, 2012; Fischl et al., 2002). Preprocessing of structural T1-weighted images consists of skull stripping, image registration, motion correction, smoothing, and subcortical segmentation.

To obtain amygdala volumes, reconstruction and volumetric calculations were automated. To obtain measures of cortical thickness and surface area, two experienced editors inspected the boundary lines separating gray/white and pial surfaces to ensure accuracy. In the case of errors, such as the pial boundary containing portions of the skull or slices where the gray/white matter boundary extended into or past the skull, further investigation was conducted by editors. If errors were visible in more than seven consecutive slices, editors corrected the errors by first changing the watershed value within FreeSurfer and then editing manually, if necessary (Ducharme et al., 2016). An experienced reviewer then completed a final quality check. Cortical thickness was then calculated by measuring the distance from the gray/white matter boundary to the pial boundary (Fischl & Dale, 2000). The Desikan–Killiany Atlas was used for cortical parcellation (Desikan et al., 2006). To minimize the number of comparisons, bilateral ROIs were created. The amygdala, insula, and mOFC were created by averaging these regions' left and right hemisphere values. The ACC was created by averaging left and right hemisphere values for both rostral and caudal ACC. Finally, the IFG was created by averaging the left and right hemisphere values for pars triangularis, pars orbitalis, and pars opercularis. FreeSurfer was also used to extract total gray matter volume (Fischl et al., 2002). Means and standard deviations are included in Table 2.

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[1] Previous research has suggested that different versions of FreeSurfer produce nominally different values for cortical thickness and subcortical volumes (e.g., Gronenschild et al., 2012); however, these variations do not change outcomes in correlational research (Bigler et al., 2020; Chepkoech et al., 2016). In our data, we had 20 cases originally processed in Freesurfer version 6.0. To investigate if there were significant effects of using different versions of Freesurfer within our data, we reprocessed those cases in version 5.1 and ran correlational analyses on those 20 cases between the values from version 5.1 and version 6.0. There were strong correlations between the two versions of cortical thickness ($rs = .849–1.000$, $ps < .001$) and cortical surface area ($rs = .907–1.000$, $ps < .001$). We also replaced values obtained from version 6.0 with values from version 5.1 in analyses that yielded significant effects (i.e., IFG and insula). Results from these analyses were similar and suggest that differences in FreeSurfer versions did not drive the observed effects. We retained the values extracted using version 5.1 as they underwent a more rigorous quality control procedure that was similar across the two studies.

2.3 | Statistical analysis

2.3.1 | ROI analyses

Multiple regression analyses explored the association between emotion regulation and the thickness and surface area of each ROI independently (i.e., amygdala, insula, mOFC, ACC, and IFG). Similar analyses were conducted between emotional reactivity and thickness, and surface area. All analyses controlled for age to account for differences in head size and sex, due to prior literature indicating sex differences in emotion regulation strategies. Volumetric analyses controlled for intracranial volume (ICV). In studies of cortical volume, ICV is often used as a covariate to account for differences in head size since ICV directly relates to cortical volume, cortical thickness, and cortical surface area. However, we chose not to add ICV as a covariate for the cortical thickness or surface area associations because cortical structure does not maintain geometric similarity with scaling (Im et al., 2008). As noted by Migliorini and colleagues (2015), there is a lower-than-expected increase of cortical volume and thickness as ICV increases and a higher-than-expected increase in cortical surface area. Therefore, a linear normalization for ICV can introduce confounding by either overscaling or underscaling cortical measurements. For any ROIs showing significant effects, follow-up analyses were conducted for each hemisphere separately. Given the lack of prior literature within this age group and our small sample size, we did not initially correct for multiple comparisons in the ROI analysis, rather we chose a conservative number of ROIs (i.e., 4). However, to account for multiple comparisons, the Holm–Bonferroni method was used to adjust the *p*-values of significant associations post hoc (Holm, 1979).

2.3.2 | Exploratory whole-brain analysis

Following the ROI analyses, we employed a whole-brain vertex-by-vertex analysis to assess previously unexamined regions. In FreeSurfer's QDEC application, linear regressions examining associations between cortical thickness, surface area, emotion regulation, and emotional reactivity were conducted, controlling for age and sex. Smoothing was applied with a 10-mm full width at half maximum (FWHM) Gaussian kernel. Additionally, Monte Carlo simulations were utilized to correct for multiple comparisons and estimate appropriate cluster sizes (Hagler et al., 2006). All analyses utilized a minimum threshold of $p < .05$.

3 | RESULTS

3.1 | Preliminary analyses

Table 3 contains bivariate Pearson correlations between all dependent measures. As expected, participant age was significantly associated with emotion regulation, emotional reactivity measures, the area of the IFG and ACC, and the volume of the amygdala. Therefore, age was included as a covariate in all analyses. Child sex was significantly related to thickness of the insula, the area of the IFG, insula, and mOFC, and the volume of the amygdala. Sex was included as a covariate in all analyses. The relation between family income and our variables of interest (emotion regulation, emotional reactivity, IFG, insula, mOFC, ACC, and amygdala) was assessed through one-way analysis of variance. No significant associations were found. Therefore, socioeconomic status (SES) was ruled out as a potential confound in our sample. All assumptions for linear regression were met.

3.2 | ROI analyses

3.2.1 | Emotion regulation and cortical thickness

Relations between emotion regulation and cortical thickness can be found in Table 4. Emotion regulation was associated with greater cortical thickness in the insula ($B = 0.142$, $\beta = .228$, $p = .012$; Figure 1), such that individuals with thicker insula had greater emotion regulation, as reported by parents when controlling for the effects of age ($B = –0.023$, $\beta = –.474$, $p < .001$) and the effects of sex ($B = 0.031$, $\beta = .188$, $p < .037$). The model ($F(3,95) = 12.491$, $p < .001$) accounted for 28% of the variance in emotion regulation. These results remained significant when adjusting the *p*-values according to the Holm–Bonferroni method. Follow-up analyses investigating lateralization of these effects revealed that this effect was driven by the thickness of the insula in the right hemisphere specifically ($F(3,95) = 12.965$, $p < .001$), which accounted for 27% of the variance in emotion regulation. Emotion regulation was associated with increased cortical thickness in the right insula ($B = 0.121$, $\beta = .241$, $p = .007$) when controlling for the effects of age ($B = –0.023$, $\beta = –.469$, $p < .001$) and the effects of sex ($B = 0.028$, $\beta = .171$, $p = .052$). These results remained significant when adjusting the *p*-values according to the Holm–Bonferroni method. Emotion regulation was not associated with cortical thickness in the left insula. There was also a nonsignificant trend toward a positive association between emotion regulation and thickness of the IFG ($B = 0.031$, $\beta = .178$, $p = .054$). Emotion regulation was not associated with cortical thickness in any other ROIs (*p*s = .230–.556) (Table 4).

3.2.2 | Emotional reactivity and cortical thickness

Relations between emotional reactivity and cortical thickness can be found in Table 4. Emotional reactivity was associated with reduced cortical thickness in the IFG ($B = –0.014$, $\beta = –.211$, $p = .033$; Figure 1), such that individuals with thinner IFG had greater emotional reactivity, as reported by parents when controlling for the effects of age ($B = 0.008$, $\beta = .376$, $p < .001$) and the effects of sex ($B = –0.006$, $\beta = –.091$, $p < .345$). The model ($F(3,95) = 6.001$, $p < .001$) accounted for 16% of the variance in emotion reactivity. When adjusting the *p*-values according to the Holm–Bonferroni method, these associations did not remain significant. Follow-up analyses investigating lateralization of these effects revealed that this effect was driven by the thickness of

TABLE 3 Bivariate correlations between study measures.

	1	2	3	4	5	6	7	8
Cortical thickness associations, *N* = 96								
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**
1. Age	–	0	–.450**	.330**	.183	.085	–.155	–.068
2. Sex		–	.155	–.068	–.180	–.250*	–.260	–.154
3. Emotion regulation			–	–.798**	.086	.179	.075	.119
4. Emotional reactivity				–	–.134	–.138	–.065	.132
5. IFG					–	.603**	.407**	.493**
6. Insula						–	.501**	.404**
7. ACC							–	.604**
8. mOFC								–
Surface area associations, *N* = 96								
	1	**2**	**3**	**4**	**5**	**6**	**7**	**8**
1. Age	–	–.005	–.450**	.330**	.216*	.173	.353**	.186
2. Sex		–	–.051	–.022	–.269**	–.409**	–.176	–.280**
3. Emotion regulation			–	–.798**	–.052	–.143	–.011	–.020
4. Emotional reactivity				–	–.007	.073	.001	.043
5. IFG					–	.657**	.511**	.612**
6. Insula						–	.549**	.713**
7. ACC							–	.675**
8. mOFC								–
Volume associations, *N* = 96								
	1	**2**	**3**	**4**	**5**			
1. Age	–	–.033	–.450**	.330**	.379**			
2. Sex		–	.155	–.068	.265**			
3. Emotion regulation			–	–.798**	–.130			
4. Emotional reactivity				–	.134			
5. Amygdala					–			

Abbreviations: ACC, anterior cingulate cortex; IFG, inferior frontal gyrus; mOFC, medial orbitofrontal cortex.
*$p < .05$; **$p < .01$.

the IFG in the right hemisphere ($F(3,95) = 5.903$, $p < .001$), which accounted for all 16% of the variance in emotion reactivity. Emotional reactivity was associated with reduced cortical thickness in the right IFG ($B = -0.012$, $\beta = -.209$, $p = .038$) when controlling for the effects of age ($B = 0.008$, $\beta = .383$, $p < .001$) and the effects of sex ($B = -0.006$, $\beta = -.099$, $p = .310$). When adjusting the *p*-values according to the Holm–Bonferroni method, these associations did not remain significant. Emotional reactivity was not associated with left IFG. Emotional reactivity was not associated with any other ROIs (*ps* = .073–.670).

3.2.3 | Emotional functioning and surface area

Neither emotion regulation nor emotional reactivity was associated with surface area in any ROIs ($p > .083$ and $p > .222$, respectively; Table 4).

3.2.4 | Emotional functioning and amygdala volume

Neither emotion regulation nor emotional reactivity was associated with amygdala volume ($p > .986$ and $p > .774$, respectively; Table 4).

3.3 | Whole-brain analyses

An exploratory vertex-by-vertex analysis revealed significant positive associations between emotion regulation and both left superior temporal thickness and right inferior temporal thickness (Figure 2), as well as negative associations between emotional reactivity and left superior temporal thickness (Figure 3). There were no associations between emotional regulation or reactivity and cortical surface area.

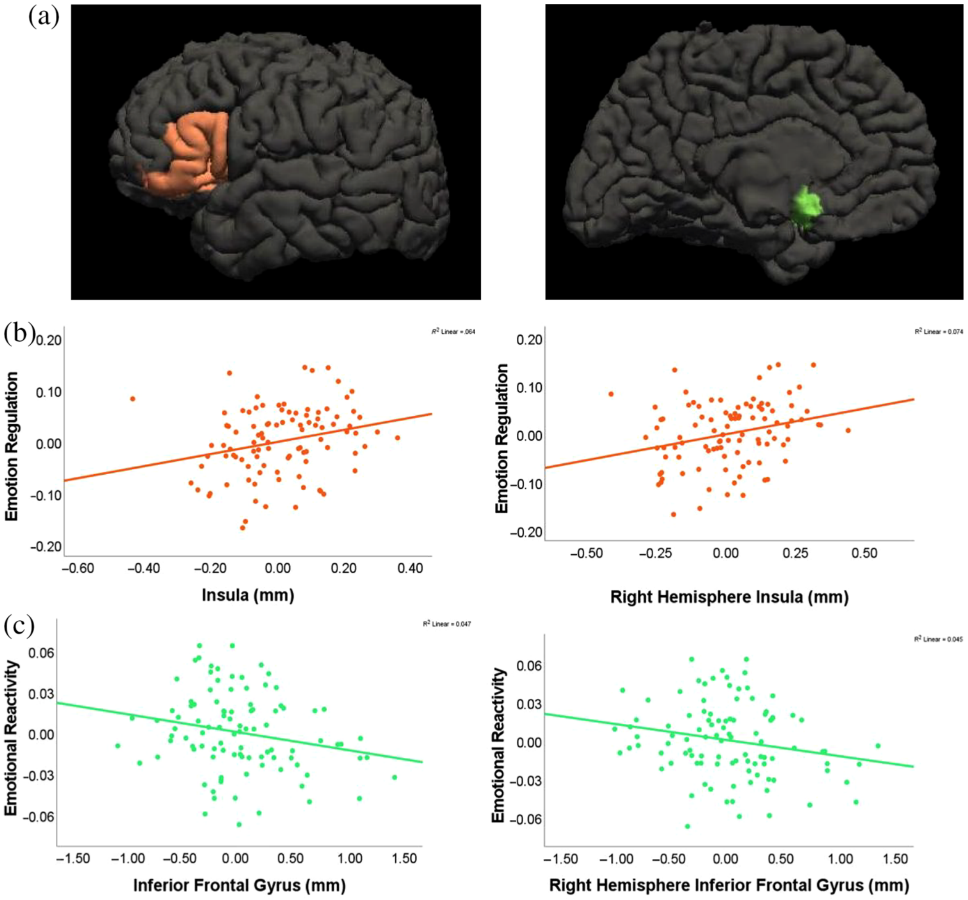

FIGURE 1 Associations of emotion regulation and emotional reactivity with regions of interest (ROIs). Panel A shows the location of the inferior frontal gyrus (IFG) (orange) and insula (green). Panel B shows significant associations with emotion regulation. Panel C shows significant associations with emotional reactivity.

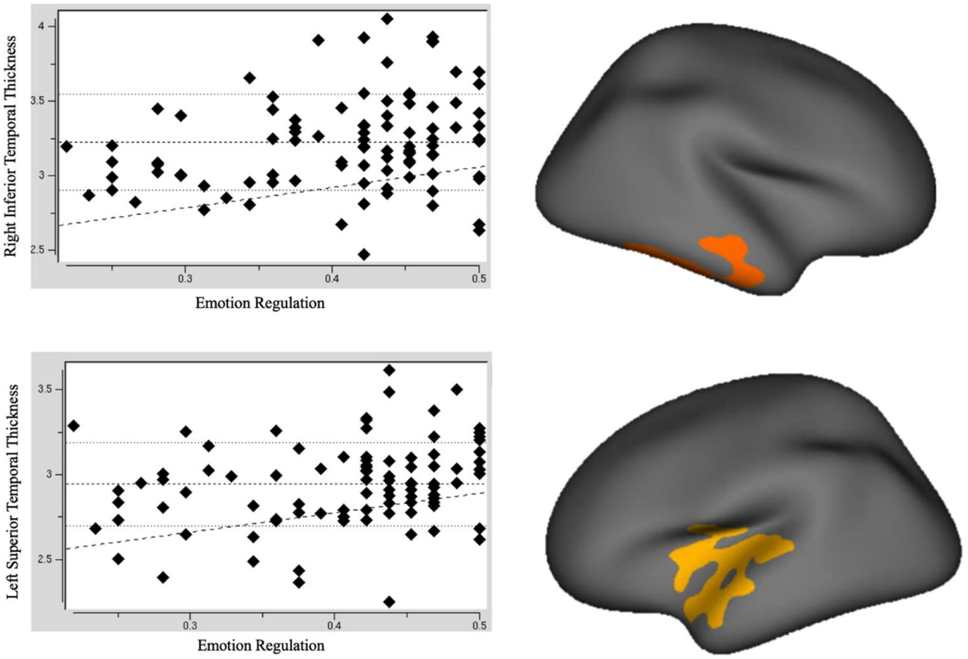

FIGURE 2 Results of the whole-brain vertex-by-vertex analysis with emotion regulation. The graphs show the associations of right inferior temporal thickness and left superior temporal thickness with emotion regulation.

TABLE 4 Associations between emotion regulation, emotional reactivity, and neural measures.

	Emotion regulation		Emotional reactivity	
	β	Adj. R^2	β	Adj. R^2
Demographic predictors				
Age	−0.462**	0.205	0.339**	0.106
Sex	0.161	0.016	−0.069	−0.006
Cortical thickness predictors				
IFG	0.178	0.24	−0.211*	0.133
lhIFG	–	–	−0.175	0.121
rhIFG	–	–	−0.209*	0.131
Insula	0.228*	0.26	−0.176	0.12
lhInsula	0.163	0.235	–	–
rhInsula	0.241*	0.268	–	–
ACC	0.056	0.212	0.043	0.092
mOFC	0.109	0.221	−0.116	0.103
Surface area predictors				
IFG	0.035	0.18	−0.095	0.088
Insula	0.189	−0.109	0.009	0.08
ACC	0.164	0.203	−0.141	0.098
mOFC	0.055	0.182	−0.027	0.81
Volume predictor				
Amygdala	−0.049	0.207	0.088	0.086

Note: Multiple regression analyses tested associations of emotion regulation and emotional reactivity measures with cortical thickness, surface area, and volume, adjusting for sex and age. Volumetric analyses also controlled for intracranial volume.

Abbreviations: ACC, anterior cingulate cortex; IFG, inferior frontal gyrus; mOFC, medial orbitofrontal cortex; rh, right hemisphere; lh, left hemisphere.

$^{*}p < .05$; $^{**}p < .001$.

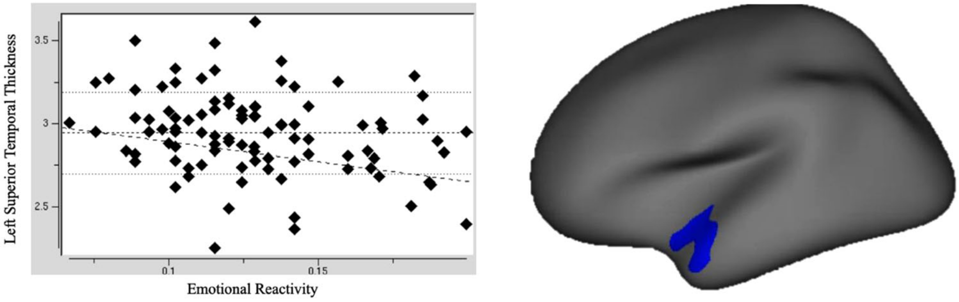

FIGURE 3 Results of whole-brain vertex-by-vertex analysis with emotional reactivity. The graph shows the association between left superior temporal thickness and emotional reactivity.

4 | DISCUSSION

Findings from this study revealed relations between emotion regulation, emotional reactivity, and components of cortical morphology. Specifically, children who employ more emotion regulation skills had thicker insula. Children who were more emotionally reactive had thinner IFG. However, there were no relations between emotion regulation or reactivity and amygdala volume or cortical surface area. An exploratory analysis also suggested associations between emotion regulation and reactivity in right temporal cortical regions. These results provide some support of the top-down model of processing proposed by the MCCE model in children. However, because relations were not found with all proposed regions, it may be the case that children utilize a different or perhaps broader neural network related to emotional functioning.

The MCCE model posits that, within adults, prefrontal and cingulate areas of the cortex are associated with emotion regulation and potentially downregulate areas responsible for generating emotion like the amygdala, ventral striatum, and vmPFC (Ochsner & Gross, 2005). Since brain structure and function are interdependent, findings from our structural ROI analyses potentially support the role of the insula and IFG in emotion regulation and emotional reactivity, respectively. However, this contrasts with the MCCE model, which posits that the insula is primarily responsible for emotion generation and is downregulated by regions like the IFG during emotion processing. It is important to note that further fMRI studies are needed to confirm these inferences. Furthermore, while the temporal lobe is not explicitly considered in the MCCE model, other work has suggested it may play a role in the evaluation of emotional significance of stimuli (Geva et al., 2011; Girbau, 2007; Lieberman et al., 2011; Morin, 2011; Shergill et al., 2003). Our exploratory whole-brain analysis supports this theory by suggesting associations between emotional functioning and the thickness of a more global network, including the temporal lobe rather than the specific prefrontal and cingulate areas implicated in the MCCE model.

One explanation for our findings being partially consistent with the MCCE model is that emotion regulation capacities could move from a global network consisting of both temporal and prefrontal regions and become more localized exclusively in the prefrontal cortex across development. Thus, these results could support a model of Interactive Specialization for emotion regulation development, specifically during early childhood. The model of Interactive Specialization argues that brain networks become more specialized across development and has been widely supported (Durston et al., 2006; Gaillard et al., 2003; Johnson, 2000, 2011). Importantly, fMRI work has suggested the involvement of temporal regions in emotion processing through adolescence and adulthood (Pozzi et al., 2021). Although emotion regulation may develop through the interactive specialization of neural regions, more work is needed to best understand the developmental trajectory.

An important consideration is that the association between the IFG and emotional reactivity did not survive corrections for multiple comparisons. However, given the lack of previous literature on this age group and our small sample size, it is possible that this is a false negative. Additional research with greater power is needed to further explore the role of the IFG in emotional reactivity in early childhood.

Despite associations between the cortical thickness of several ROIs and emotional functioning measures, there were no significant findings with surface area measures. These conflicting findings align with theories that argue that cortical thickness and surface area are two similar but distinct measures of cortical morphology (Raznahan et al., 2011; Wierenga et al., 2014). More work is needed to understand how these two distinct measures function and interact across development. Additionally, future work should investigate the relations of both cortical thickness and cortical surface area with emotional functioning across the life span.

In summary, the current study adds to growing evidence that both emotion regulation and emotional reactivity are related to several ROIs in the frontal cortex as well as a more global region in the temporal lobe. Furthermore, this investigation extends these findings by suggesting that individual differences in emotion regulation and emotional reactivity may relate to individual differences in brain structure during early to mid-childhood, an understudied population in neuroscientific research. Finally, this study provides important evidence to support a model of Interactive Specialization in the development of emotion regulation and helps to inform the developmental timeline of the MCCE model. However, it should be noted that emotion regulation is a complex process, and further work is needed to fully understand individual differences in the neural bases of emotional functioning. Further research may investigate the clinical implications of these differences in atypical samples or longitudinally.

This study has several limitations. First, while the ERC is a well-established instrument, it could have introduced bias to the study because it is a parent report measure (Cohen & Mendez, 2009; Izard et al., 2008). For example, parents may be more likely to rate their child's emotion regulation skills higher to appear more socially desirable. Further, the emotion regulation scale of the ERC indexes a broad range of factors, including empathy. While emotion regulation is known to be necessary for empathy (Maibom, 2019; Thompson et al., 2019), the ERC may be tapping a measure of empathy that is independent of emotion regulation. It may be beneficial for future studies to utilize a behavioral observation of emotion regulation instead of a parent report measure. Additionally, the lack of socioeconomic and ethnic diversity in this sample is a limitation. While SES did not relate to the measures used within our sample, this may be due to the lack of variability in family income and not due to a true null finding. Further, some research suggests that cultural differences impact components of emotion regulation (Ford & Mauss, 2015; Miller & Kinsbourne, 2012). Therefore, these findings may not generalize across socioeconomic classes, racial/ethnic groups, or broader cultural contexts. Future studies should attempt to replicate these findings both within and across culturally and socioeconomically diverse samples. Additionally, this study only included structural MRI data, which limits the ability to hypothesize about functional activity within the regions implicated in this research. We included a broad age range across early to mid-childhood, but acknowledge that associations between emotional functioning and brain structure may change across this range. Further, because of this study's cross-sectional design, these findings only speak to individual differences in cortical thickness within this age group; we cannot conclude about maturational changes. Future work, preferably longitudinal in nature, is needed to address questions regarding variations of associations within this age range.

This study provides evidence that the MCCE model may not fully explain the neural circuitry underlying emotion in children. Due to our sample size, we chose a conservative number of ROIs from the MCCE model to test in relation to emotion regulation and emotional reactivity. However, it should be noted that several neural networks have been proposed to play a role in emotional functioning in adults (Morawetz et al., 2020). Therefore, the neural circuitry in children is likely more extensive than the MCCE model suggests. Future studies should investigate the neural bases of emotion regulation in children more broadly to understand these relations fully.

5 | CONCLUSION

Emotional regulation is necessary for socioemotional success and later mental health outcomes. Therefore, it is essential to examine neurobiological correlates of emotion regulation and emotional reactivity across development. Results from the current study provide empirical support that in early to mid-childhood, individual differences in emotion regulation and reactivity map onto individual differences in brain structure. Associations were found with cortical thickness but not surface area, suggesting these are two related but separate measures of cortical morphology. Lastly, these findings potentially support the theory of interactive specialization in the development of emotion regulation by suggesting emotion regulation processes may be regulated by global temporal and prefrontal regions early in development before potentially localizing within the prefrontal cortex. However, more research is needed to better understand the link between emotion regulation and brain structure from a life span perspective.

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CONFLICT OF INTEREST STATEMENT

The authors declare no conflicts of interest.

DATA AVAILABILITY STATEMENT

Data are available upon reasonable request.

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