THE CONTRIBUTION OF CHILDHOOD NEGATIVE EMOTIONALITY AND COGNITIVE CONTROL TO ANXIETY-LINKED NEURAL DYSREGULATION OF EMOTION IN ADOLESCENCE

BY

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THESIS

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Abstract

Adolescence has been identified as a period of heightened emotional reactivity, which is reflected in greater activation in emotion-processing regions of the brain in adolescents relative to children and adults. While elevated emotional reactivity and poor emotion regulation are thought to contribute to the rise in rates of internalizing psychopathology, including anxiety, during adolescence, little research has examined factors predicting individual differences in the neural regulation of emotion that can explain why only a subset of adolescents develop anxiety. The present study examined the contribution of childhood negative emotionality (NE) and cognitive control (CC) to neural processing of emotion in adolescence. A sample of 44 girls (*M* age = 15.5; *SD* = .35) were selected from a longitudinal study that included self, parent, and teacher report of NE and CC between 2nd and 7th grades. Following 9th grade, girls completed an emotion regulation functional magnetic resonance imaging (fMRI) task. We assessed neural dysregulation of emotion by examining functional connectivity between the amygdala and right ventrolateral prefrontal cortex (rVLPFC) during emotion regulation and found that NE predicted a less mature pattern of positive amygdala-rVLPFC connectivity while CC predicted a more mature pattern of negative amygdala-rVLPFC connectivity. Additionally, we found an interaction between NE and CC, such that NE predicted emotion dysregulation only at low levels of CC. Neural dysregulation of emotion was associated with anxiety symptoms across the following nine months. These findings identify important individual differences in the development of emotion dysregulation that contribute to risk for anxiety in adolescence.

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Introduction

Adolescence is a period of rapid emotional and cognitive development during which youth experience increasing autonomy and exposure to novel social and emotional stressors (Spear, 2000). Failure to develop effective emotion regulation strategies puts youth at risk for emotional difficulties, such as anxiety, that increase in prevalence and stability across adolescence (Pine et al., 1998). Contemporary theory and research on adolescent development implicate this stage as a time of increased neural and behavioral sensitivity to emotion (Casey, Jones, & Hare, 2008; Ernst, Pine, & Hardin, 2006; Hare et al., 2008; Silk et al., 2009). However, most youth develop effective ways of regulating emotions, while only a subset goes on to develop anxiety. It is therefore important to consider pre-existing individual differences that may lead to variability in neural signatures of emotion regulation during adolescence. Addressing this gap, the present study explored the contribution of two childhood traits, negative emotionality and cognitive control, to neural regulation of emotion in adolescence. In particular, to overcome limitations created by the independent investigation of individual brain regions (Pfeifer & Allen, 2016), functional connectivity analysis was used to examine coordination across regions involved in emotional reactivity (i.e., amygdala) and top-down control of emotion (i.e., prefrontal cortex). Moreover, to enhance understanding of the psychological implications of neural processing of emotion in adolescence, this study also examined the association between functional connectivity during emotion regulation and subsequent anxiety symptoms.

Emotion Processing in Adolescence

Several lines of research support the idea that adolescence is a period of heightened emotional sensitivity. Adolescents, especially mid-adolescents, report more negative emotions and greater day-to-day variability in emotional states than younger children, later adolescents, or

adults (Larson, Moneta, Richards, & Wilson, 2002; Silk et al., 2009; for reviews, see Ernst et al., 2006; Somerville, Jones, & Casey, 2010), and show greater physiological reactivity to emotion words than do children (Silk et al., 2009). Despite this adolescent-specific peak (Casey, 2015) in emotional reactivity, age-related increases in emotion regulation abilities allow adolescents to begin adapting to their changing environment and developing strategies to effectively regulate their emotions. For example, research reveals age-related increases in the ability to successfully down-regulate emotional reactivity to negatively valenced pictures (Silvers et al., 2012). Unfortunately, the growth of regulatory skills occurs at a slower rate than the peak in emotional reactivity, resulting in more difficulty regulating emotions during mid-adolescence than during childhood or adulthood (McRae et al., 2012).

Neural Correlates of Emotion Processing in Adolescence

One proposed explanation for the maturational gap between emotional reactivity and emotion regulation during mid-adolescence is that the neural systems involved in emotional reactivity develop earlier than the systems involved in cognitive control (Casey et al., 2008; Ernst et al., 2006; Nelson, Jarcho, & Guyer, 2016; Steinberg, 2008). Some evidence supports heightened amygdala reactivity to emotion in adolescents relative to children and adults (Guyer et al., 2008; Hare et al., 2008), but evidence regarding the development of regulatory regions is less consistent. Many studies have found that compared to activity in emotion-processing regions, activity in the prefrontal cortex (PFC) reflects slower maturation, with adults showing greater PFC activation than adolescents during emotion regulation tasks (Casey, 2015; Monk et al., 2003; Shulman et al., 2016). However, others have found that adolescents show greater PFC activation than children or adults during emotion regulation tasks (Crone & Dahl, 2012; Pfeifer & Allen, 2012).

This inconsistency highlights the need to move beyond studying differences in activation within specific regions to consider patterns of connectivity between regions, such as the amygdala and PFC, that interact to support effective emotion regulation. Although few studies compare connectivity patterns at different developmental stages, some research reveals that children show more positive functional connectivity between the amygdala and PFC during emotion regulation, whereas adolescents and adults show more negative amygdala-PFC functional connectivity (Gee et al., 2013; Gee et al., 2014; Guyer et al., 2008; Monk et al., 2008). This pattern is thought to reflect improved top-down regulation of amygdala hyperactivity, as evidenced by the finding that more negative amygdala-PFC functional connectivity in adolescence predicts faster amygdala habituation to fearful faces (Hare et al., 2008). Maintaining positive functional coupling throughout adolescence may thus reflect a more immature pattern of connectivity and a failure to effectively down-regulate emotional reactivity.

Individual Differences in Neural Processing of Emotion

Despite this heightened reactivity to emotion in adolescents, many develop effective emotion regulation strategies, whereas a smaller proportion develop maladaptive strategies that put them at risk for adverse emotional outcomes. An important next step for research on adolescent neural development is to identify earlier individual differences that promote or prevent the development of adaptive emotion regulation strategies during adolescence (Pfeiffer & Allen, 2012; Somerville et al., 2010). To address this critical question, the present study examined whether negative emotionality and cognitive control, two temperamental traits measured in childhood, predict individual differences in emotion regulation in adolescents.

Negative emotionality as a predictor of emotion regulation. Negative emotionality (NE) is a dimension of temperament that may predict less effective emotion regulation in

adolescence. NE is characterized by susceptibility to experiencing negative emotions, difficulty being soothed once aroused, and heightened sensitivity to negative social cues (Compas, Conner-Smith, & Jaser, 2004; Rothbart, Adahi, Hershey, & Fisher, 2001). Trait differences in NE are associated with ruminative responses to negative emotions (Verstraeten, Vasey, Raes, & Bijttebier, 2009) and maladaptive emotion regulation strategies in children (Santucci et al., 2008) and adults (Gross & John, 2003). Little research has examined the impact of NE on neural systems involved in emotion regulation, although the related temperamental dimension of childhood behavioral inhibition (characterized by a tendency to show fear or reticence in novel social situations) predicts greater amygdala activation to emotional faces in early adolescence (Perez-Edgar et al., 2007). Additionally, NE is associated with greater amygdala activation during emotion maintenance in adults (Schaefer et al., 2002). High trait NE may sensitize individuals to emotionally salient information, increasing their reactivity to emotion cues and making it more difficult to effectively regulate this response. Accordingly, we hypothesized that childhood NE would predict a less mature pattern of amygdala-PFC functional coupling (i.e., positive functional connectivity) during emotion regulation in adolescence.

Cognitive control as a predictor of emotion regulation. Cognitive control (CC) develops across childhood and adolescence and plays an important role in emotion regulation. CC is characterized by the ability to maintain directed attention toward goals and to disengage from task-irrelevant information (Miller & Cohen, 2001). While CC increases into adulthood, there are stable individual differences in CC that distinguish individuals throughout development (Ochsner & Gross, 2005; Zhou, Chen, & Main, 2012). Neuroscience research supports the role of trait CC in emotion regulation. One longitudinal study found that self-regulation measured during childhood predicted greater PFC activation during an emotion regulation task completed

in adulthood (Casey et al., 2011), suggesting that individuals with higher CC may more effectively recruit regions of the PFC to down-regulate activity in emotion-processing regions of the brain. Therefore, we hypothesized that childhood CC would predict a more mature pattern of amygdala-PFC functional coupling (i.e., negative functional connectivity) during emotion regulation in adolescence.

Interactive contribution of negative emotionality and cognitive control to emotion regulation. Beyond the independent effects of NE and CC, there may be important interactive effects of these traits on emotion regulation. While heightened emotional sensitivity (i.e., high NE) may predict less effective emotion regulation, this effect may be weaker in youth who have better self-regulation abilities (i.e., high CC). Past research suggests that CC may moderate the effect of NE on coping styles (Lengua & Long, 2002), responses to parent-child stress (Yap et al., 2011), and rumination (Verstraeten et al., 2009). Yet research has not yet addressed the interactive effects of NE and CC on neural regulation of emotion. Past models of adolescent neural development have suggested a pattern of heightened reactivity in regions involved in emotional reactivity relative to regions involved in cognitive control (Casey et al., 2008; Ernst et al., 2006; Steinberg, 2008). However, these studies have not examined how this general pattern is impacted by trait differences in reactivity and regulation. Accordingly, it is important to understand how individual differences in these traits interact to predict functional coupling of limbic and frontal regions during emotion regulation. It is hypothesized that NE will predict more positive (i.e., maladaptive) functional coupling at low, but not high, levels of CC, suggesting that CC may serve as a buffer against the negative effects of NE on emotion regulation during adolescence.

Neural Processing of Emotion and Anxiety

In addition to identifying the childhood antecedents of individual differences in adolescent neural regulation of emotions, it is important to understand the psychological implications of these patterns for adolescent adjustment. Previous research suggests that age-related changes in neural responses to emotion may contribute to risk for anxiety in adolescence (Casey et al., 2008; Ernst et al., 2006). By mid-adolescence, most youth show a pattern of negative amygdala-PFC connectivity during emotion regulation (Gee et al., 2014). Maintaining a less mature pattern of amygdala-PFC coupling into mid-adolescence may be maladaptive, preventing adolescents from effectively regulating reactivity to emotional stimuli. As a result, adolescents may experience more emotional distress such as anxiety. Indeed, previous research has found that adolescents who show a less mature pattern of amygdala-PFC connectivity during emotion processing (Monk et al., 2008) and social evaluation (Guyer et al., 2008) are more likely to experience anxiety. Expanding on prior research, we used a longitudinal design to examine whether a less mature pattern of amygdala-PFC coupling (i.e., positive functional connectivity) would predict more future anxiety symptoms in adolescents.

Gender Differences in Adolescent Emotion Processing

Adolescence also is a crucial period in the development of gender differences in emotional sensitivity. Compared to adolescent boys, girls are exposed to higher levels of social stressors (Hankin, Mermelstein, & Roesch, 2007; Rose & Rudolph, 2006) and experience more emotional reactivity and emotional distress following exposure to stressors (Charbonneau, Mezulis, & Hyde, 2009; Hampel & Petermann, 2006; Rudolph, Flynn, Abaied, Groot, & Thompson, 2009). Moreover, adolescent girls are more likely than adolescent boys to engage in maladaptive emotion regulation strategies such as rumination in response to social stressors (Jose

& Brown, 2008). There is also some evidence of gender differences in neural systems involved in emotion processing. Early adolescent girls show less amygdala habituation following repeated exposure to fearful faces than do boys (Thomas et al., 2001). Additionally, there is some evidence that age-related shifts in amygdala and PFC activity during emotion processing are more pronounced in girls than in boys (Killgore, Oki, & Yurgelun-Todd, 2001). Given girls' heightened exposure and sensitivity to emotion-inducing contexts and their increased use of certain maladaptive emotion regulation strategies (i.e., rumination) relative to boys during adolescence, the present study focused on neural processing of emotion specifically in adolescent girls.

Study Overview

This study used a longitudinal, multi-method design to examine childhood predictors of individual differences in the neural processing of emotion in adolescence. It builds upon past models of normative adolescent development by examining how individual differences in childhood NE and CC confer risk for, or resilience against, neural dysregulation of emotion in adolescence. NE and CC were examined annually when youth were in 2nd through 7th grade via parent-, teacher-, and child-report questionnaires. Neural processing of emotion was assessed in 9th grade using fMRI while adolescents completed an emotion regulation task. The comparison of interest in this task was the difference in brain connectivity when adolescents labeled facial expressions of negative emotions compared to passively viewing them. This task measures implicit emotion regulation thought to be engaged as a result of labeling emotions (Brooks et al., 2016; Gyurak, Gross, & Etkin, 2011; Lieberman et al., 2007). We hypothesized that (a) NE would predict a less mature pattern of amygdala-PFC connectivity; (b) CC would predict a more mature pattern of amygdala-PFC connectivity; and (c) the association between NE and less

mature patterns of amygdala-PFC connectivity would be strongest at low levels of CC (i.e., NE x CC interaction), highlighting the independent and interactive effects of childhood traits on emotion regulation in adolescence.

In addition to understanding how childhood traits influence individual differences in adolescent neural development, we were interested in exploring whether differences in the neural processing of emotion contribute to risk for anxiety. Building on past research suggesting a link between neural processing of emotion and concurrent anxiety symptoms (e.g., Monk et al., 2008), we examined the association between functional connectivity during emotion regulation and self-reported anxiety symptoms measured across the following nine months. We hypothesized that a less mature pattern of connectivity would predict more future anxiety symptoms, emphasizing the importance of linking individual differences in neural development to critical psychological outcomes.

Methods

Participants and Procedures

Participants were 44 adolescent girls (mean age = 15.5 years, *SD* = .35; 68.2% Caucasian, 27.3% African American, 2.3% Asian, and 2.3% Latina) recruited from a larger longitudinal study that began when youth were in second grade. Participants and their parents and teachers completed questionnaires annually between second and seventh grades. Youth completed questionnaires in small groups during elementary school and by classroom in middle school. Parents and teachers completed questionnaires independently and received monetary compensation. During the summer following 9th grade[1], a subset of youth from the longitudinal study was recruited to participate in a laboratory visit during which they completed computer tasks while undergoing functional magnetic resonance imaging (fMRI)[2]. Participants also completed questionnaires three, six, and nine months after the scan. Participants received monetary compensation for completion of the fMRI scan and follow-up questionnaires. Youth provided written assent and parents provided written consent. All procedures were approved by the university's Institutional Review Board.

Measures

Table 1 provides descriptive and psychometric information on the measures.

Negative emotionality. Trait negative emotionality (NE) was assessed using the Temperament in Middle Childhood Questionnaire (TMCQ; Simonds, Kieras, Rueda, & Rothbart, 2007; Simonds & Rothbart, 2004), which includes 25 items reflecting the tendency to show

[1]Two adolescents completed the laboratory visit during the summer following 10th grade due to prior ineligibility for the fMRI scan (i.e., metal braces).
[2]A total of 50 youth completed the laboratory visit. Six are excluded from analyses due to an error in the design of the emotion regulation task.

intense negative emotions, including anger (e.g., “Gets irritated when she has to stop doing something she is enjoying.”) and sadness (e.g., “Becomes sad when told to do something she doesn’t want to do.”) as well as low soothability (e.g., “Is very difficult to soothe when she has become upset.”). The NE subscale shows strong internal consistency and inter-rater reliability (Rothbart et al., 2001), including between parents and children (Lengua, 2003). Construct validity has been supported through associations with behavioral (Wilson, 2006) and clinical (Sugimura & Rudolph, 2012) outcomes. Parents reported on youths’ NE in second through fifth grade. Youth and teachers reported on youths’ NE in fifth through seventh grade. Composite scores of trait NE were created by standardizing ratings within informant and averaging across all informants (i.e., youth, parent, and teacher) and waves.

Cognitive control. Cognitive control (CC) was assessed using the effortful control scale from the TMCQ (Simonds et al., 2007; Simonds & Rothbart, 2004) and an overall score of executive function from the Brief Rating Inventory of Executive Function (BRIEF; Gioia, Isquith, Guy, & Kenworthy, 2000). The effortful control scale includes 15 items assessing attentional control (e.g., “When working on an activity, has a hard time keeping her mind on it.”) and inhibitory control (e.g., “Can stop herself from doing things too quickly.”). This scale shows strong internal consistency as well as test-retest and inter-rater reliability (Simonds et al., 2007). Construct validity is supported in community child samples (Kochanska, Murray, & Harlan, 2000; Muris, van der Penne, Sigmond, & Mayer, 2008). The BRIEF includes 86 items assessing different deficits in executive function. For this study, a subset of 40 items was selected to assess working memory (e.g., “Has trouble with chores or tasks that have more than one step.”), planning (e.g., “Does not plan ahead for school assignments.”), shifting (e.g., “Tries the same approach to a problem over and over, even when it does not work.”), and inhibition (e.g., “Has

trouble putting the brakes on her actions."). The BRIEF shows strong internal consistency and inter-rater reliability, including between parents and teachers (Agoston & Rudolph, 2016; Gioia & Isquith, 2004). Construct validity of the BRIEF has been supported in both clinical and community samples (Gioia, Isquith, Guy, & Kenworthy, 2000; Gioia et al., 2002). Parents reported on youths' effortful control in second through fifth grade. Youth and teachers reported on youths' effortful control in fifth through seventh grade. Teachers reported on youths' executive function in sixth and seventh grade. Because effortful control and executive function overlap conceptually and are correlated in child samples (Blair & Razza, 2007; Zhou et al., 2012), including our sample (rs = .51 - .87, ps < .001), composite scores of CC were created by reverse scoring the BRIEF and then standardizing ratings within informant and averaging across all measures, informants (i.e., youth, parent, and teacher), and waves. High scores on this composite measure reflect higher levels of cognitive control.

Anxiety symptoms. Youth completed the Revised Child Manifest Anxiety Scale (RCMAS; Reynolds & Richmond, 1985). The RCMAS includes 28 items assessing anxiety symptoms (e.g., "I often worry about something bad happening to me."). The RCMAS shows strong internal consistency (Reynolds & Richmond, 1978) and test-retest reliability (Wisniewski, Genshaft, Mulick, & Coury, 1987). Construct validity, including convergent and divergent validity (Reynolds, 1982), has been supported through comparisons of youth with and without anxiety disorders (Seligman, Ollendick, Langley, & Baldacci, 2004). Youth reported their anxiety symptoms at three, six, and nine months following the scan session. Completion rate across the follow-up period was 98%, 89%, and 93% at three, six, and nine months respectively. An overall follow-up anxiety score was calculated by averaging across the nine months following the scan session.

Emotion regulation fMRI Task. The fMRI scan included an emotion regulation task modified from Lieberman and colleagues (Lieberman et al., 2007). During one functional run, participants were presented with faces displaying negative emotions (anger, sadness, and fear). Participants completed two blocks during which they were instructed to observe the emotional faces (*observe*; Figure 1a), and two blocks during which they were instructed to match the emotional face to one of two emotion word labels presented below the image (*label*; Figure 1b). Each block consisted of six trials, each of which lasted six seconds. Block order was randomized across participants, and a 10-second rest period occurred between blocks. The race of models (half African American and half European American) and emotion types were randomized within the blocks; all photos were of women taken from the NimStim (Tottenham et al., 2009).

Data Acquisition and Analysis

fMRI data acquisition. The fMRI data were collected using a 3 Tesla Siemens Trio MRI scanner. The task included T2*-weighted echoplanar images (EPI) [slice thickness=3 mm; 38 slices; TR=2sec; TE=25msec; matrix=92x92; FOV=230 mm; voxel size 2.5x2.5x3mm^3]. Structural scans consisted of a T2*weighted, matched-bandwidth (MBW), high-resolution, anatomical scan (TR=4sec; TE=64msec; matrix=192x192; FOV=230; slice thickness=3mm; 38 slices) and a T1* magnetization-prepared rapid-acquisition gradient echo (MPRAGE; TR=1.9sec; TE=2.3msec; matrix=256x256; FOV=230; sagittal plane; slice thickness=1mm; 192 slices).

fMRI data analysis. The fMRI data were preprocessed using statistical parametric mapping (SPM8; Wellcome Department of Cognitive Neurology, Institute of Neurology, London, UK). Images were spatially realigned to correct for head movement. Functional data were coregistered to the structural MPRAGE, which was then segmented into cerebrospinal

fluid, gray matter, and white matter. Structural and functional images were then transformed into standardized stereotactic space as defined by the Montreal Neurological Institute. The normalized functional data were smoothed using an 8mm Guassian kernel, full-width-at-half-maximum, to increase signal-to-noise ratio.

For each participant's data, a general linear model (GLM) was created using regressors that corresponded to the two task conditions. High-pass temporal filtering with a cutoff of 128 seconds was applied to remove low-frequency drift in the data. Parameter estimates resulting from the GLM were then used to create linear contrasts. Because we were interested in emotion regulation, we focused on the contrast between the *label* and *observe* conditions (i.e. *label>observe*). We chose the *label>observe* contrast because it is thought that putting an emotion into words via labeling helps down-regulate emotional reactivity, whereas passively observing an emotion may elicit more emotional reactivity (Lieberman et al., 2007).

To assess emotion regulation, we focused on connectivity between the amygdala and regulatory regions in the prefrontal cortex (PFC). We used psychophysiological interactions (PPI) to examine neural connectivity, using the bilateral amygdala as the seed region. The amygdala region of interest (ROI) was defined by combining the left and right anatomically-defined amygdala. The automated gPPI toolbox in SPM (gPPI; McLaren, Ries, Xu, & Johnson, 2012) was used (1) to extract the deconvolved times series from the bilateral amygdala ROI for each participant to create the physiological variables, (2) to convolve each trial type with the canonical HRF to create the psychological regressor, and (3) to multiply the time series from the psychological regressors with the physiological variable to create the PPI interaction. Given our *a priori* hypotheses, we restricted our PPI analyses to the right VLPFC (rVLPFC). We focused on the rVLPFC because past research has found this region to be more active during emotion

labeling than passive viewing, and activation in this region is positively correlated with reduced amygdala activation in the *label* condition (Lieberman et al., 2007). The rVLPFC was defined using the inferior frontal orbital gyrus in the AAL atlas in the WFU PickAtlas (Maldjian, Laurienti, & Burdette, 2004; Maldjian, Laurienti, Kraft, & Burdette, 2003; Tzourio-Mazoyer, et al., 2002). Parameter estimates of signal intensity were extracted from the PPI analysis and represent connectivity between the amygdala and rVLPFC. We used a linear regression model in SPSS with NE, CC, and their interaction as predictors of functional connectivity between the amygdala and rVLPFC during the *label>observe* contrast.

Results

Bivariate Correlations

Table 2 presents bivariate correlations among the variables. Negative emotionality and cognitive control were negatively correlated. As predicted, amygdala-rVLPFC functional connectivity during emotion regulation (*label >observe)* was positively correlated with negative emotionality (Figure 2a) and negatively correlated with cognitive control (Figure 2b).

Negative Emotionality x Cognitive Control Predicting Amygdala-VLPFC Functional Connectivity

A hierarchical linear regression analysis was conducted to examine the independent and interactive contribution of negative emotionality and cognitive control to amygdala-rVLPFC functional connectivity during the emotion regulation task (Table 3). Negative emotionality and cognitive control were standardized, and their interaction was calculated as the product of the standardized variables. The main effects of negative emotionality and cognitive control were entered in the first step of the regression, and their interaction was entered in the second step. The NE x CC interaction was decomposed using simple slopes analysis at low (-1 SD), moderate (mean), and high (+1 SD) levels of CC (Aiken & West, 1991).

The regression analysis revealed a significant main effect of negative emotionality and a nonsignificant main effect of cognitive control in Step 1 (when only the main effects were entered). As hypothesized, the interaction of negative emotionality and cognitive control significantly predicted functional connectivity during emotion regulation (Table 3). As shown in Figure 3, decomposition of the interaction revealed that negative emotionality significantly predicted more positive functional connectivity at low (b = 0.38, $t = 2.40$, $p = .02$) but not

moderate (b = .19, t = 1.04, p = .31) or high (b = -0.002, t = -0.008, p = .99) levels of cognitive control.

Functional Connectivity Predicting Anxiety

To explore behavioral correlates of functional connectivity during the emotion regulation task, we examined the association between amygdala-rVLPFC functional connectivity and subsequent anxiety (Table 2). As expected, positive functional connectivity was significantly associated with anxiety across the following nine months (Figure 4a).

Discussion

Past theory and research have identified adolescence as a period of sensitivity in emotion processing regions coupled with less effective down-regulation of this reactivity by regulatory regions (Casey et al., 2008; Crone & Dahl, 2012). However, not all adolescents exhibit neural dysregulation of emotion, highlighting the importance of identifying factors that predict which youth may show this pattern due to individual differences earlier in development (Pfeifer & Allen, 2012). To address the scarcity of research in this area, the present study examined how individual differences in two childhood temperamental traits, NE and CC, predict variability in functional connectivity between the amygdala and PFC during emotion regulation. In line with our hypotheses, we found that childhood NE predicted less mature amygdala-PFC connectivity during adolescence, but this effect was specific to adolescent girls with low levels of childhood CC. Additionally, an immature pattern of amygdala-PFC connectivity during emotion regulation predicted future anxiety symptoms.

Temperamental Traits and Neural Dysregulation

The observed link between temperament and neural regulation of emotion suggests a mechanism through which individual differences in childhood traits may confer risk for, or resilience against, emotional difficulties during adolescence. While adolescence has been implicated as a time of heightened emotional reactivity combined with compromised emotion regulation, the present study suggests that this disjuncture may be especially relevant for youth with high levels of NE. These adolescents may be especially sensitive to negative emotion cues, making it more difficult for them to down-regulate amygdala reactivity during an emotion regulation task. NE has previously been associated with less effective emotion regulation strategies in children (Santucci et al., 2008) and adults (Gross & John, 2003). The present study

adds to this line of research by identifying a neural pathway (immature amygdala-PFC connectivity) through which NE contributes to less effective emotion regulation.

Importantly, however, in this study we found that the link between NE and emotion dysregulation was specific to girls with compromised CC. This pattern builds on past research revealing that CC moderates the effect of NE on responses to stress (Lengua & Long, 2002), ruminative emotion regulation strategies (Verstraeten et al., 2009), and symptoms of emotional distress (Muris, Meesters, & Blijlevens, 2007). In our study, NE predicted a less mature pattern of amygdala-PFC connectivity only in adolescent girls with poor CC, suggesting that CC may mitigate against the effects of NE on neural dysregulation of emotions, allowing youth to more effectively recover from negative information and redirect their attention towards task-relevant goals.

Neural Dysregulation and Anxiety

This study also found that neural dysregulation of emotion predicts prospective anxiety symptoms during adolescence. Past studies have found that anxious adolescents exhibit more positive amygdala-PFC connectivity while viewing angry faces (Monk et al., 2008) and anticipating social feedback (Guyer et al., 2008). The present study builds upon this research, finding that more positive (i.e., less mature) amygdala-PFC connectivity during emotion regulation predicted higher levels of anxiety over the nine months following the scan. This research adds to existing knowledge by examining connectivity during emotion regulation, when PFC down-regulation of amygdala is especially salient, and by examining the prospective association between neural dysregulation of emotion and anxiety symptoms.

While adolescence has long been of interest as a period of increasing risk for the development of anxiety, these findings help explain why only certain adolescent girls develop

anxiety during this time (Pfeiffer & Allen, 2012). Most youth show a pattern of negative amygdala-PFC connectivity by mid-adolescence (Gee et al., 2014). Our results suggest that girls who retain a less mature pattern of positive connectivity may be especially susceptible to the development of anxiety. Ineffective regulation of amygdala activity may lead to hypervigilance for negative emotional cues and inability to direct attention away from emotionally distracting information, setting the stage for the development of sustained worry and eventual anxiety. In support of this proposed pathway, prior research has found that adolescents higher in trait anxiety show less amygdala habituation following repeated exposure to emotional faces (Hare et al., 2008).

Contributions and Limitations

This research highlights potential strategies for improving emotion regulation and preventing the emergence of anxiety in girls during adolescence. Specifically, interventions that increase CC may promote the development of more effective emotion regulation in adolescence and may be especially beneficial to girls with high levels of NE. In support of this idea, research on mindfulness, a therapeutic approach that increases CC (Chambers, Lo, & Allen, 2008; Slatger et al., 2007), has found that mindfulness improves emotion regulation (Hilt & Pollack, 2004), alters connections between brain regions involved in emotion reactivity and regulation (Lutz et al., 2014), and decreases symptoms of anxiety (Hofmann, Sawyer, Witt, & Oh, 2010; Miller, Fletcher, & Kabat-Zinn, 1995).

Despite the novel contributions of this study, it has noteworthy limitations. First, although previous research suggests emotion labeling is an effective implicit emotion regulation strategy (Brooks et al., 2016; Gyurak et al., 2011; Lieberman et al., 2007), we did not explicitly instruct participants to regulate their emotions during the task. As we cannot be certain that

labeling emotions engaged emotion regulation strategies to a similar extent in all of our participants, future studies examining patterns of functional connectivity during explicit emotion regulation tasks are warranted. Second, this study only examined neural regulation of emotion in adolescent girls. While girls tend to show heightened emotional reactivity during adolescence (Charbonneau et al., 2008), it is not clear the extent to which these findings generalize to boys. Third, while NE and CC were measured across childhood, neural processing of emotion was measured at a single point in time. It is likely that temperament and neural systems supporting emotion regulation have bidirectional effects on each other across child and adolescent development. Ideally, future research will examine the interdependence of temperament and individual differences in neural processing of emotion across development.

Conclusion

The present study examined whether individual differences in childhood temperament predict variability in neural regulation of emotion during adolescence. Findings revealed that NE predicted less mature amygdala-PFC functional connectivity during emotion regulation, an effect that held only in girls with poor cognitive control in childhood. Furthermore, less mature connectivity predicted higher levels of anxiety across the following nine months, identifying an important psychological impact of neural dysregulation of emotion and highlighting the importance of considering individual differences in emotion regulation during adolescence.

Tables

Table 1

Descriptive and Psychometric Information

	2nd grade		*3rd grade*		*4th grade*		*5th grade*		*6th grade*		*7th grade*		*Follow Up*	
Measure	*M (SD)*	*a*	*M (SD)*	*a*	*M (SD)*	*a*	*M (SD)*	*a*	*M (SD)*	*a*	*M (SD)*	*a*	*M (SD)*	*a*
Negative Emotionality														
Parent	2.48 (.51)	.91	2.53 (.45)	.91	2.33 (.51)	.92	2.28 (.59)	.92						
Child							2.62 (.88)	.92	2.53 (.86)	.92	2.51 (.90)	.93		
Teacher							2.06 (.79)	.96	1.84 (.75)	.96	1.86 (.64)	.95		
Effortful Control														
Parent	3.38 (.49)	.73	3.33 (.60)	.75	3.51 (.54)	.71	3.55 (.70)	.91						
Child							3.54 (.71)	.84	3.58 (.71)	.86	3.55 (.69)	.84		
Teacher							3.69 (.88)	.94	3.71 (.89)	.94	3.80 (.85)	.93		
Executive Function Deficits									1.34 (.41)	.98	1.28 (.35)	.97		
Anxiety Symptoms													13.44 (.27)	.95

Table 2

Intercorrelations among the Variables

Measure	1	2	3	4
1. Negative Emotionality	--			
2. Cognitive Control	-.84***	--		
3. Functional Connectivity	.48***	-.38*	--	
4. Follow Up Anxiety Symptoms	.66***	-.60***	.32*	--

Note. $^{*}p < .05$. $^{**}p < .01$. $^{***}p < .001$.

Table 3

Predicting Positive Functional Connectivity from Negative Emotionality, Cognitive Control, and their Interaction

Predictors	*ß*	*t*
Step 1		
Negative Emotionality	.55	2.22*
Cognitive Control	.09	.35
Step 2		
Negative Emotionality	.27	.99
Cognitive Control	-.13	-.50
Negative Emotionality x Cognitive Control	-.33	-2.20*

Note. Coefficients and *t*s represent statistics at each step of the regression equation.
*$p < .05$

Figures

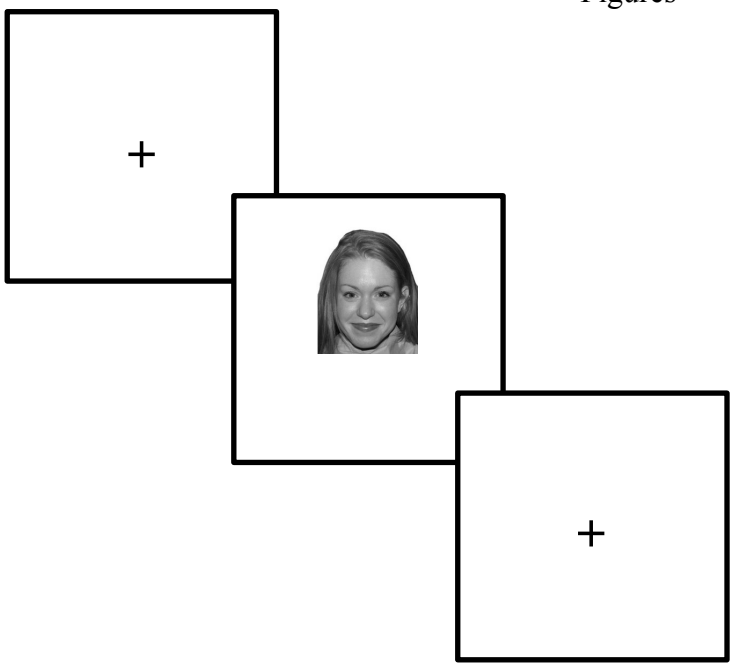

Figure 1a. Observe condition of the emotion regulation task.

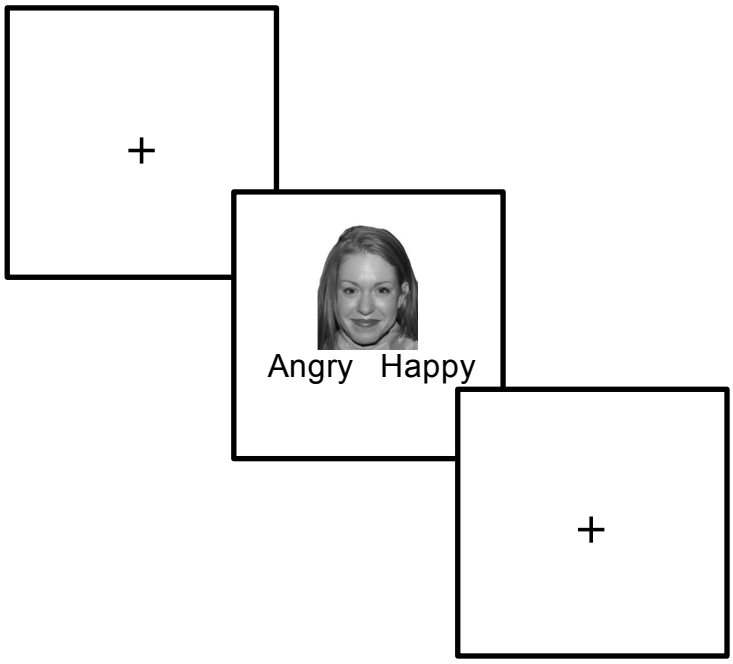

Figure 1b. Label condition of the emotion regulation task.

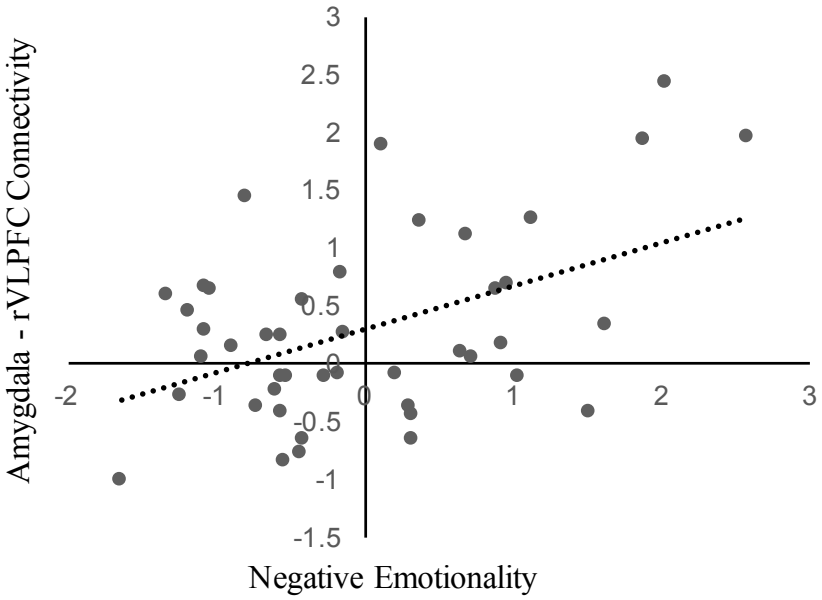

Figure 2a. Positive association between negative emotionality and functional connectivity.

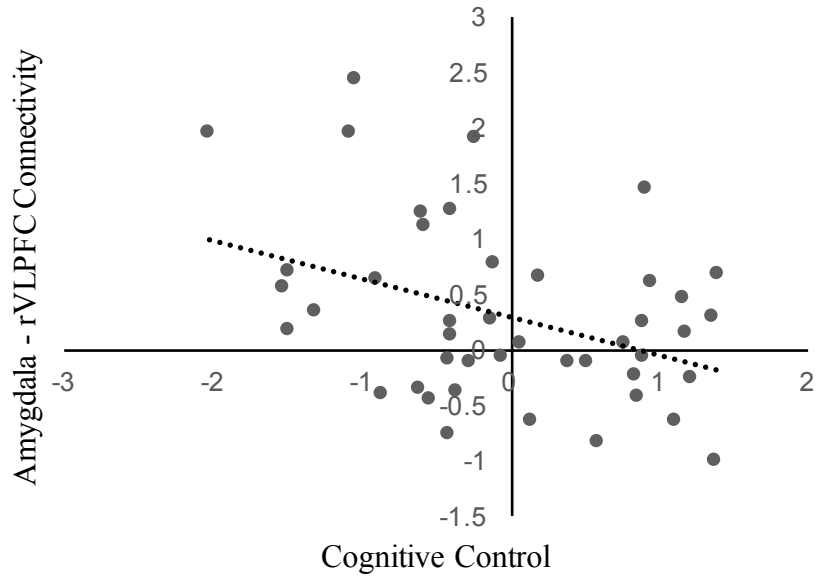

Figure 2b. Negative association between cognitive control and functional connectivity.

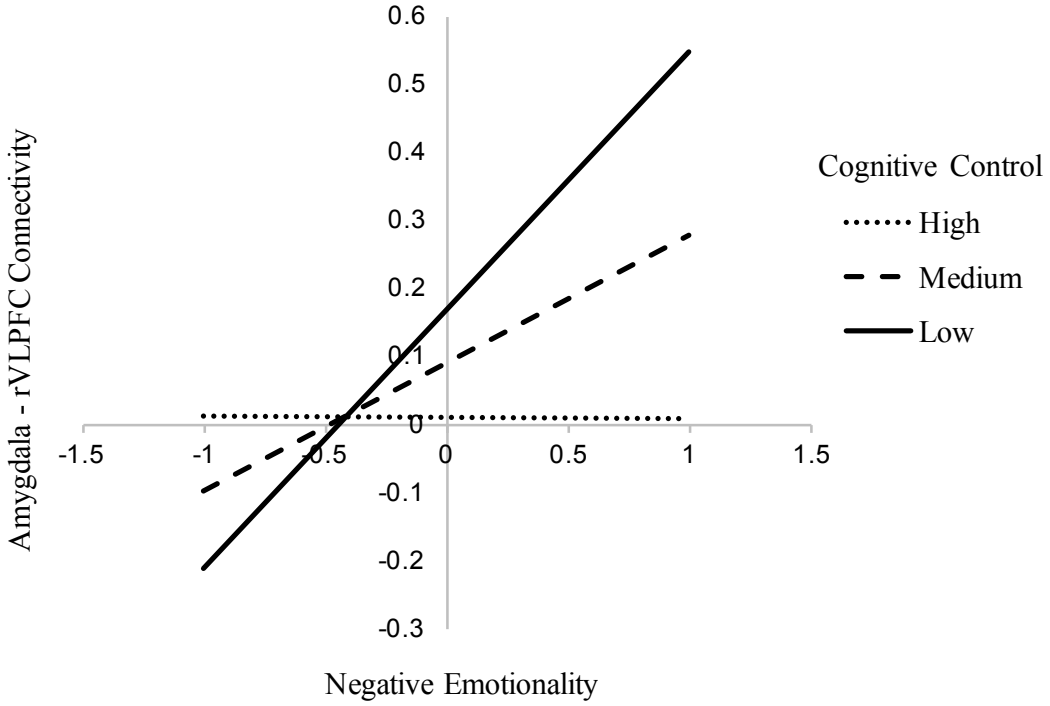

Figure 3. Association between negative emotionality and functional connectivity at low, moderate, and high levels of cognitive control.

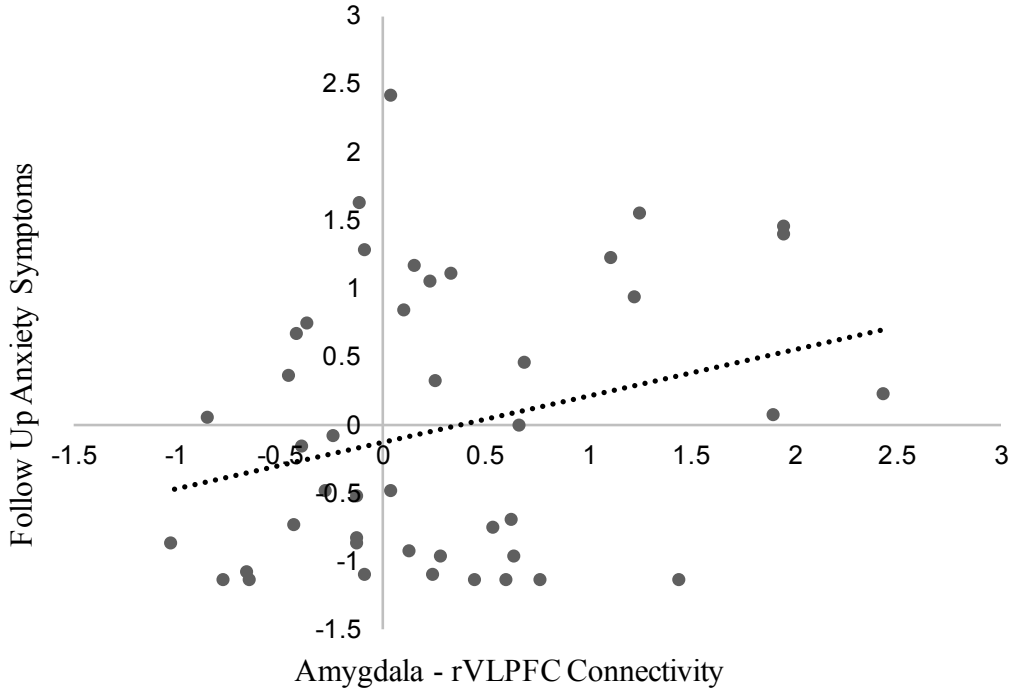

Figure 4. Association between functional connectivity and anxiety symptoms across follow-up.

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