Parental Coping Socialization is Associated with Healthy and Anxious Early-Adolescents' Neural and Real-World Response to Threat

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Developmental Science (In Press)

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There are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

Acknowledgements: Research funding was provided by P50 MH080215 (Ryan).

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Research Highlights:

- Youth typically learn how to cope with their anxiety through their parents' coping socialization behaviors. However, the neural mechanisms through which this occurs are unknown.
- Results show that engagement coping socialization during anxiety-eliciting, parent-child interactions are associated with increased anterior insula and perigenual cingulate activation to threat words in anxious early-adolescents.
- Conversely, findings show that coping socialization is associated with decreased anterior insula and pgACC activation in healthy early-adolescents.
- Greater coping socialization was indirectly associated with less use of disengaged coping (i.e., avoidance and distraction) in daily life through neural activation for anxious early-adolescents only.

Abstract

The ways parents socialize their adolescents to cope with anxiety (i.e. coping socialization) may be instrumental in the development of threat processing and coping responses. Coping socialization may be important for anxious adolescents, as they show altered neural threat processing and over-reliance on disengaged coping (e.g., avoidance and distraction), which can maintain anxiety. We investigated whether coping socialization was associated with anxious and healthy adolescents' neural response to threat, and whether neural activation was associated with disengaged coping. Healthy and clinically anxious early-adolescents (*N*=120; *M*=11.46 years; 71 girls) and a parent engaged in interactions designed to elicit adolescents' anxiety and parents' response to adolescents' anxiety. Parents' use of reframing and problem-solving statements was coded to measure coping socialization. In a subsequent visit, we assessed adolescents' neural response to threat words during a neuroimaging task. Adolescents' disengaged coping was measured using ecological momentary assessment. Greater coping socialization was associated with lower anterior insula and perigenual cingulate activation in healthy adolescents and higher activation in anxious adolescents. Coping socialization was indirectly associated with less disengaged coping for anxious adolescents through neural activation. Findings suggest that associations between coping socialization and early adolescents' neural response to threat differ depending on clinical status and have implications for anxious adolescents' coping.

Keywords: adolescent anxiety; threat processing; parenting; socialization; neuroimaging; coping

Parental Coping Socialization is Associated with Healthy and Anxious Early Adolescents' Neural and Real-World Response to Threat

Adolescents are at heightened risk for clinical levels of anxiety (Merikangas et al., 2010). This risk is believed to be putatively associated with growing awareness and fear of abstract forms of threat, including death, danger, social and academic evaluation (Beesdo, Knappe, & Pine, 2009; Weems & Costa, 2005). Anxiety is characterized by excessive vigilance towards threat, heightened physiological arousal, exaggerated negative emotionality, and maladaptive over-reliance on disengagement coping strategies, such as avoidance, in response to anxiety-provoking situations (Compas, Connor-Smith, Saltzman, Thomsen, & Wadsworth, 2001; LeDoux & Pine, 2016; Strawn, Dominick, et al., 2014; Suveg & Zeman, 2004; Zeman, Cassano, Perry-Parrish, & Stegall, 2006). These characteristics are thought to represent alterations in emotion processing and underlying neural systems. For children and adolescents, parental factors have been found to contribute to the development of negative emotion processing and coping abilities when measured behaviorally (Morris, Silk, Steinberg, Myers, & Robinson, 2007). Therefore, it is theorized that parental factors play a role in shaping the development of the neural circuitry underlying children's emotion processing and regulation (Kopala-Sibley et al., 2018).

Initial studies have shown support for the role of parenting on the neural substrates of emotional reactivity and regulation, particularly in younger children. For example, behavioral research has shown that the presence of mothers during fear conditioning has been shown to buffer children's conditioned startle responses (van Rooij et al., 2017). Affective neuroscience studies have also shown that viewing pictures of mothers (versus strangers) displayed during a neuroimaging task support the regulatory effects of amygdala reactivity by the prefrontal cortex

(Gee et al., 2014). However, these buffering effects were found only in children, and were absent in adolescents. Therefore, there may be different parenting factors that scaffold emotion processing and regulation abilities in older youth.

The potential for continued influence of parental factors on brain functioning in adolescence may be a result of the extended maturation process of the human brain, both functionally and structurally, which spans from infancy through late-adolescence/early-adulthood (Kopala-Sibley et al., 2018; Luna, Padmanabhan, & O'Hearn, 2010). Late childhood through early-adolescence is a major period of neural maturation in the frontal cortex, which occurs in the forms of myelination and synaptic pruning (see review by Andersen, 2003). This is a period of dramatic neuronal reorganization, such that there is a nearly 40% decrease in synaptic density by age 15. This maturation period coincides with increases in various cognitive abilities, such as abstract reasoning, emotion regulation, cognitive control, and support processes necessary for environmental adaptation (Andersen, 2003). Periods of major neural re-organization are known to be particularly sensitive to the influences of environmental factors, and it has been posited that such input from the environment helps to guide neural maturation processes that will be supportive of adaptive response and behavior (Andersen, 2003). Therefore, the information that youth learn from their parents during the early-adolescent period may play an important role in supporting ongoing maturation processes of brain function that subserve emotion processing and regulation. Given that many new challenges arise in early-adolescence, it is a particularly important period during which youth must learn how to adaptively cope with feelings of negative affect, such as threat. To this end, the current study seeks to examine how parental factors specific to socializing adaptive coping behaviors in youth may be associated with the functioning of neural regions that support threat processing in early-adolescents.

Though limited, affective neuroscience research has begun to show that parenting factors are related to adolescent neural processing of negatively valenced stimuli in regions implicated in processing threat stimuli. For example, healthy adolescents who reported having warmer parents exhibited less amygdala reactivity in response to negative emotional faces (versus neutral faces), possibly indicating less hypervigilance to and appraisal of threat in response to negative stimuli (Romund et al., 2016). Also, 7-year-old children who were behaviorally inhibited as toddlers exhibited lower ventrolateral prefrontal cortex (VLPFC) activation to peer rejection during adolescence, if they had harsh authoritarian parents (Guyer et al., 2015). Such findings indicate that negative or harsh parenting styles could be associated with reduced recruitment of prefrontal cortical regions that support regulatory processes in the context of processing threatening information. Together, these findings suggest that parental factors are important when trying to understand individual differences in the functioning of neural systems implicated in threat processing. While these research advances are important, to-date no studies have shown how the links between parental influences and neural function implicated in the processing of threat impact adolescents' day-to-day behavior.

The two previously mentioned studies focused on broad parenting factors including affect (i.e., warmth) and style (i.e., authoritative and authoritarian). However, the literature has shown that youth learn to utilize more adaptive response strategies to cope with negative emotion when their parents exhibit active, engagement-oriented coping socialization practices (Abaied & Rudolph, 2010; Morris et al., 2011; Morris et al., 2007; Zeman et al., 2006). These more specific parenting behaviors, including reframing, problem-solving, and encouragement to face fearful situations, are posited to model and support adaptive coping strategy use in children (i.e. parental coping socialization). Engagement-oriented coping socialization behaviors may be especially

important for adolescents with anxiety, as these youth tend to rely on disengaged coping strategies, such as avoiding, distracting, or escaping from benign situations, which they incorrectly judge as threatening and thereby anxiety producing (Barrett, Rapee, Dadds, & Ryan, 1996; Chorpita, Albano, & Barlow, 1996; Suveg & Zeman, 2004; Weems, Costa, Watts, Taylor, & Cannon, 2007). Therefore, in the current study we investigated the effects of these more specific parental coping socialization behaviors on neural activity in regions implicated in threat processing in healthy and anxious early-adolescents. We also explored whether these associations are related to adolescents' reported use of disengaged coping strategies on a daily basis in the real world. Findings from the current study could contribute to deepening our understanding of how adaptive coping responses to anxiety-provoking situations are socialized in adolescents. Despite the use of a cross-sectional design, this novel study could identify potential neural mechanisms that may explain the link between parental socialization behaviors and adolescent real-world coping behaviors.

Brain activity in early-adolescents was assessed using a functional neuroimaging task that involves processing threat-related information and elicits activation in brain regions implicated in youth anxiety (Strawn, Dominick, et al., 2014). Through a region-of-interest (ROI) approach, we focused on the amygdala, anterior insula, and subgenual cingulate (sgACC), which are part of a neural network circuit involved in detecting and appraising negative, threat-related stimuli (Guyer et al., 2008; Phan, Wager, Taylor, & Liberzon, 2002; Singer, Critchley, & Preuschoff, 2009). We also examined brain regions implicated in automatic fear regulation, involuntary attentional and emotional control, and subjective emotions, including the perigenual cingulate (pgACC/BA24) and the ventrolateral prefrontal cortex (VLPFC/BA47) (Blackford & Pine, 2012; Etkin, Egner, & Kalisch, 2011; LeDoux & Pine, 2016; Posner, Rothbart, Sheese, & Tang, 2007;

Strawn, Wehry, DelBello, Rynn, & Strakowski, 2012). We specifically included the pgACC (BA24) region, as opposed to the dorsal ACC region (BA32), as the pgACC is known to have the most dense bi-directional connections to the amygdala and insula (Blackford & Pine, 2012; Posner et al., 2007) and is implicated in the regulation of threat processing, fear extinction, and the facilitation of adaptive responses (Etkin et al., 2011). PgACC activity is also found to distinguish emotionally valenced words from neutrally valenced abstract words (Vigliocco et al., 2014), relevant to the task used in the current study.

In order to capture the specificity of parental coping socialization behaviors, we asked participants to complete two anxiety-provoking, parent-adolescent interaction tasks and coded how often parents used engagement-oriented coping socialization behaviors (e.g. reframing, problem-solving). Although parents' engagement-oriented coping socialization behaviors fall under the umbrella of supportive responses to children's emotions, as used in previous coding categorization systems (Eisenberg, Cumberland, & Spinrad, 1998; Eisenberg, Fabes, Carlo, & Karbon, 1992), we utilized a modified coding system that allowed us to focus on parenting behaviors theorized in the emotion socialization literature to specifically help youth cope with anxiety (Ginsburg & Schlossberg, 2002; Wood, McLeod, Sigman, Hwang, & Chu, 2003). Although parenting is often thought of as having direct effects on youth, these effects can be bi-directional (i.e., child behaviors and characteristics driving parental behaviors). Specific to anxiety, parents of anxious youth perceive their children's high reactivity in response to negative events, and in turn, may view their children as more vulnerable or helpless (Ginsburg & Schlossberg, 2002). Consequently, parents may exhibit high distress and react with over-controlling and intrusive behaviors or encourage avoidance in the context of potential threat. Such behaviors have adverse effects on how youth cope with anxiety, including the

reinforcement of youth's sensitivity to perceived threat, avoidance of challenges, and the maintenance of anxiety symptoms (Dadds, Barrett, Rapee, & Ryan, 1996; Lewis-morrarty et al., 2012; Van Der Bruggen, Stams, & Bögels, 2008; Zalewski, Lengua, Wilson, Trancik, & Bazinet, 2011). Therefore, we explore whether there are differences in the socialization of coping strategies in parents of healthy adolescents and those of anxious adolescents. In addition, it has been suggested that youth who are highly reactive to environmental cues may be more affected by parenting than less reactive youth (for review see Kiff, Lengua, & Zalewski, 2011). If this is the case, then it might be that neural response to threat-related information in anxious adolescents, characterized by high emotional reactivity, might be more susceptible to the effects of parenting than healthy adolescents. To test this hypothesis, we assessed whether parenting differentially influenced neural response to threat in anxious versus healthy youth.

We also explored whether adolescent brain function, associated with parenting, would be related to adolescent-reported use of disengaged coping in real-world environments. This may be particularly relevant to assess in clinically anxious adolescents, given that higher internalizing symptoms are found in youth who disengage (e.g., avoid) from their challenges, compared to those who actively engage with challenges (Compas et al., 2001). Regions implicated in detecting and regulating threat responses have been associated with cognitive coping responses in healthy and anxious populations (see review by Hofmann, Ellard, & Siegle, 2012). For example, adolescents who reported themselves as high in the dimension of harm avoidance, using a temperament questionnaire, exhibited greater activation in the sgACC during an inhibition-related task (Yang et al., 2009). More specific to the use of avoidance behaviors, during an avoidance-approach fMRI task healthy, 9-to-14 year old youth showed increased activation in the amygdala and insula to threat-related (i.e., snake) avoidance cues (Schlund et

al., 2010). Youth who had more frequent behavioral responses per second to avoidance cues exhibited higher amygdala activation, but lower anterior insula, pgACC, and anterior cingulate cortex activation (Schlund et al., 2010). These results suggest that greater avoidance tendencies may be associated with increased activation in affective salience regions and lower engagement of midline-prefrontal regions.

More lateral and superior regions of the PFC have also been implicated in cognitive coping responses in youth. Specifically, during an fMRI paradigm using facial expression stimuli, adolescents with generalized anxiety disorder were shown to have an attentional bias away from angry faces (possibly reflecting avoidance) and also showed greater activation in the VLPFC in response to angry faces, compared to healthy youth (Monk et al., 2006). However, activation in the VLPFC has also been found in healthy youth when they are instructed to utilize more adaptive coping strategies, such as reappraisal (McRae et al., 2012). Furthermore, adolescents with and without histories of maltreatment have been shown to exhibit greater activation in the superior PFC, anterior cingulate, and the lateral inferior frontal gyrus/VLPFC when asked to regulate their negative emotional response to negative images (versus passive viewing) (McLaughlin, Peverill, Gold, Alves, & Sheridan, 2015). Therefore, it is still unclear if functional activation patterns in PFC regions, such as the VLPFC, can differentiate the use of various coping strategies or if activation in these regions are general to youth's attempts to down-regulate negative emotions, regardless of strategy. Overall, though, studies to date suggest that the function of affective salience and regulatory regions may play a role in coping among adolescents. However, no study has assessed how neural activation in these regions may be associated with coping strategies used in real-world situations.

The current study used ecological momentary assessment (EMA), an ecologically valid approach, to assess how often early-adolescents use disengaged-coping strategies when faced with negative events occurring in their daily life. EMA allows behavioral information to be captured as it occurs in adolescents' natural environments. Furthermore, EMA reduces the reliance on retrospective accounts, which are often biased due to recency effects, bias toward infrequent events or peak-level subjective experiences, and inconsistent reports of coping strategy use (Stone et al., 1998). Adolescent disengaged coping was operationalized to include avoidance and distraction strategies because both of these strategies are known to contribute to the maintenance of anxiety (Aupperle & Paulus, 2010; Wright, Banerjee, Hoek, Rieffe, & Novin, 2010). Distraction can serve both adaptive and maladaptive functions and has been found to load onto a secondary, engagement coping factor (Connor-Smith, Compas, Wadsworth, Harding Thomsen, & Saltzman, 2000), we decided to consider it a disengagement strategy because distraction involves directing attention away from stressors, rather than engaging in more active strategies that involve solving one's problems or reframing the situation in efforts to reduce anxiety or fear (Compas et al., 2001). Although avoidance and distraction strategies can be adaptive in some circumstances, a previous study conducted in the current sample found these strategies to be ineffective in the down-regulation of nervousness for both anxious and healthy early-adolescents (Tan et al., 2012). The use of both of these strategies was also associated with attentional avoidance and higher vigilance towards threat during an fMRI dot probe task in the current sample of anxious adolescents (Price et al., 2016).

In the present study, we tested several hypotheses about the relationships between parental coping socialization during parent-child interactions, early-adolescents' neural response to threat words, and disengaged coping in daily life. First, preliminary analyses assessed whether

parents of anxious youth would exhibit less frequent coping socialization behaviors during anxiety-provoking interaction tasks than parents of healthy youth. Second, we hypothesized that for both healthy and clinically anxious early-adolescents, greater parental coping socialization would be associated with lower activation in regions implicated in vigilance and arousal to threat, including the amygdala, anterior insula, and sgACC. Additionally, we hypothesized that greater parental coping socialization would be associated with higher activation in regions implicated in fear regulation and involuntary attentional and emotional control, including the pgACC and VLPFC. Third, we explored whether the associations between parenting and early-adolescent neural threat processing differed between anxious and non-anxious adolescents. Specifically, we hypothesized that the neural function of the aforementioned brain regions implicated in threat processing would be more strongly associated with parental socialization in adolescents with clinical anxiety compared to healthy adolescents. Finally, for brain regions that were shown to be associated with parental coping socialization, we explored whether coping socialization would have indirect effects on adolescents' use of disengaged coping (i.e., lower reliance on avoidant and distraction coping behaviors) in daily life through neural activation.

Methods

Participants

One hundred twenty early-adolescents (84.2% Caucasian), ages 9-14 years old (M=11.46, SD=1.52; 71 girls), including 87 with clinical anxiety, and their primary caregiver (114 mothers, 5 fathers, 1 grandmother; hereafter referred to as parents for brevity) were recruited for a child anxiety treatment study through local media advertisements, school counselors, mental health and pediatrician referrals, and other research studies (see Silk et al., 2018). We operationalized early adolescence in this study as beginning at age 9, as this age has been found to be around the

typical onset of the early stages of pubertal maturation (Herman-Giddens, 2006). Anxious youth were required to meet DSM-IV (American Psychiatric Association, 1994) criteria for current generalized anxiety, separation anxiety, and/or social anxiety disorders. Approximately 27% of anxious youth were diagnosed with multiple anxiety disorders and 14.3% had comorbid disorders (see Table 1 for complete details). For all participants, exclusion criteria included IQ below 70, assessed by the Wechsler Abbreviated Scale of Intelligence (Psychological Corporation, 1999), or risk for harm to self or others. Participants were also excluded if they reported any MRI contraindication. Exclusion criteria for anxious participants further included current use of psychotropic medications, current primary diagnosis of major depressive disorder, obsessive-compulsive disorder, post-traumatic stress disorder, conduct disorder, substance abuse or dependence, or attention deficit hyperactivity disorder (combined type or hyperactive-impulsive type), or a lifetime diagnosis of autism spectrum disorder, bipolar disorder, psychotic depression, schizophrenia, or schizoaffective disorder. The control group could not have a current or lifetime DSM-IV diagnosis (other than enuresis) or have a parent with a current or lifetime DSM-IV anxiety or mood disorder diagnosis. See Table 1 for participant demographics.

Procedure

Parents completed pre-screening phone interviews. During their first laboratory visit, parents and youth were briefed on the study protocol. Written informed consent from parents and assent from youth were obtained. Study procedures were approved by the University Institutional Review Board. Next, participants completed structured diagnostic interviews, questionnaires, and parent-adolescent observation tasks. Following visit 1, adolescents completed a 5-day ecological momentary assessment (EMA) protocol on study-provided mobile phones. Approximately three weeks later (M_{days}=23.61, *SD*=12.42), adolescents completed a functional

magnetic resonance imaging (fMRI) assessment at a brain imaging center. Out of 183 participating adolescents, 153 completed the fMRI scan. Of those, 33 were excluded from analyses due to: cyst found during scan (n=1); excessive motion (see preprocessing section; n=28); or missing behavioral responses on more than one-third of task trials (n=4). Participants who did not complete the scan or had unusable fMRI data were younger in age (M_{age}=10.30, *SD*=1.21) than included participants (*t*=5.27, *p*<.001), but did not differ in gender, race, or anxiety severity scores (*p*>.05).

Measures

Kiddie-Schedule for Affective Disorders and Schizophrenia-Present and Lifetime Version (KSADS-PL). Parents and youth were interviewed separately to determine adolescents' mental health history. Semi-structured KSADS-PL (Kaufman et al., 1997) interviews were completed by trained BA- and MA-level independent evaluators. Data from both informants was integrated for diagnoses. Inter-rater reliability using 16% of interviews was high (κ=.97) (Silk et al., 2018). A DSM-IV (American Psychiatric Association, 1994) final diagnosis was provided by a child psychiatrist during consensus case conferences.

Parent-adolescent interaction tasks. Parents and early-adolescents completed two interaction tasks, including a five-minute discussion in which the dyad discussed a recent time when the adolescent was worried (adapted from Suveg, Zeman, Flannery-Schroeder, & Cassano, 2005; Whaley, Pinto, & Sigman, 1999) and a five-minute speech task. In the speech task, the adolescent was told that they would be giving a video-taped, 1 min 30 sec speech about a topic they chose out of several challenging options. Youth were informed that their performance would be assessed and compared to others. Parents were asked to help their adolescent prepare. Adolescents were also given the option to complete a second speech. Parents and adolescents

were left alone to discuss whether or not to complete the second speech, during which we aimed to capture parents' behaviors used to encourage or discourage their adolescent's participation (Silk et al., 2013).

Parent and early-adolescent behaviors during both tasks were videotaped and coded using a modified version of the Living in Family Environments Coding System (LIFE; Hops, 2007). The LIFE system is an event-based, micro-social coding system that captures verbal content and nonverbal or paraverbal indices of affect. These content and affect codes are combined rationally into constructs, which are used for analysis. In the present study, we used a "Coping Statement" construct which included new content codes capturing: 1) parental encouragement to problem-solve and approach challenges; and 2) cognitive reframing, as long as they were said without aversive (aggressive/contemptuous) or anxious affects. For example, statements in which parents encouraged their adolescent to try the feared activity (i.e. speech task) included: "I think you should do it, too," or "the speech only takes a couple minute". An example of a statement in which the parent helped to reframe the situation or feared task, in order to help their adolescent cope with their anxiety, included: "the best way to overcome being uncomfortable at doing something is to do it and to do it often." Trained research staff who were not aware of diagnostic group assignment coded the interactions. Reliability assessed on 20% of interactions was good (κ=.72). Rate per minute of coping statements for both tasks was averaged to create a single coping statement variable.

EMA. Adolescents were given cellphones at visit 1 to complete 14 calls over 5 days. Trained interviewers administered ~5 minute phone interviews at random intervals, during pre-determined blocks, to assess adolescents' current emotional state, most positive and negative events occurring within the past hour, and coping strategy used in response to negative events

(details in Tan et al., 2012). Youth were called twice between the hours of 4 p.m. and 9:30 p.m. on weekdays (Thursday, Friday and Monday) and four times between the times of 11 a.m. and 9:30 p.m. on Saturday and Sunday, totaling 14 calls or sampling events. The current study focuses on "yes/no" endorsements of avoidant coping in response to negative events. The avoidant coping construct was based on two coping strategy questions: "Did you try not to think about it or try to forget all about it [the problem/negative event]?" (avoidance/suppression); and "Did you keep your mind off of the problem by doing something else?" (distraction). Adolescents rated their distress (angry, nervous, sad, and/or upset) levels on a scale of 1 through 5 (1=very slightly or not at all, 2=a little, 3=moderately, 4=quite a bit, 5=extremely). An emotion rated as a 1 or 2 would not necessarily be strong enough to require emotion regulation strategies. For this reason, we calculated the proportion of calls in which avoidance/suppression or distraction were endorsed in response to negative events that caused a distress level of 3 or above, similar to previous work on emotion regulation (Price, et al., 2016). The mean number of calls included was 7.95 (SD=3.89).

fMRI Task and Acquisition. Adolescents were familiarized with the scanner sounds and trained to minimize movement during an MRI simulation. Participants completed a structural scan followed by functional tasks, including the word valence identification (VID) task (adapted from Silk et al., 2007). Tasks were completed in random order, varying for each participant. During the slow-event related VID task, youth identified the valence of words (n=51) that were chosen from a word corpus normed for youth (Neshat-Doost, Moradi, Taghavi, Yule, & Dalgleish, 1999; Neshat-Doost, Moradi, Taghavi, Yule, & Dalgleish, 2000). Word types included physical threat (n=15), social threat (n=15), and neutral (n=15). A small number of positive words (n=6) were also included to add variation, but were not intended for analysis.

Participants were presented with each word, one time each, and were asked to indicate the valence of the word (i.e. positive, neutral, or negative) using a Psychology Software Tools™ glove. Words were presented using E-Prime software (Psychology Software Tools, Pittsburgh, PA) in black on a grey background, and valence identification options were displayed on screen throughout the task (e.g., "+N−" representing "Positive" on the index finger, "Neutral" on the middle finger and "Negative" on the ring finger). Trials began with a 900ms fixation cross, followed by a 1500ms word presentation, and ended with the presentation of a mask (a row of Xs) for a 9190ms inter-trial interval. Including such a mask allowed sufficient time to for elaborative processing following word presentation and allowed time for the hemodynamic response function to return to baseline (see Silk, Lee, Kerestes, et al., 2017).

The present study focused on physical threat words, such as "attacked," "fire," and "kidnapped," as threat to human safety and well-being are evolutionarily salient. Although threat words present no actual threat to participants, they have been found to activate cognitive and emotional processes associated with fear and anxiety—particularly among anxious populations (MacLeod, Mathews, & Tata, 1986). We did not compare neural activation during physical threat word trials to neutral word trials because neutral information is often found to trigger activation associated with ambiguity (Kober et al., 2008; Pfeifer et al., 2011), especially in youth (Silk et al., 2009; Thomas, Drevets, Dahl, & et al., 2001), making it difficult to interpret this contrast.

Imaging Acquisition. Data were collected on a 3T Siemens Trio scanner across three runs/sessions. Stimuli were projected onto a rear projection screen and viewed through a mirror. E-Prime was used to present the task and collect behavioral responses. Responses were made with a 5-button Psychology Software Tools glove. Thirty-two, 3.2mm slices were acquired per

volume using a posterior-to-anterior a T2* weighted echo planar imaging pulse sequence (TR=1670 ms, TE=29 ms, FOV=205x205 mm, matrix size= 64 x 64, voxel size=3.2 x 3.2 x 3.2 mm^3, flip angle=75º, slice thickness=3.2 mm). 357 EPI volumes were acquired across the task (7 per 11.69s trial). 176 high-resolution T1-weighted MPRAGE images were also collected (TR=2100 ms, TE=3.31 ms, FOV=265x208, matrix size=256x208, voxel size=1.0 x 1.0 x 1.0 mm^3, flip angle=8º, slice thickness=1 mm).

Preprocessing and ROI data analysis. Analyses were conducted using NeuroImaging Software (Fissell et al., 2003), Analysis of Functional Neuroimaging (AFNI; Cox, 1996), and custom Matlab routines. Functional volumes were corrected for slice-timing and spatially realigned to correct for motion. Functional imaging data were slice-time corrected using *3dTshift* and motion-corrected using *3dVolReg* based on the first image (a reference image) implemented in AFNI. Linear trends over the run were removed using *niscorrect* from NeuroImaging Software. This procedure also reduces the impact of within-subject outliers by winsorizing or clipping outliers over 1.5 interquartile range (IQR) from the 25th or 75th percentiles to the nearest value. Data were temporally smoothed using a 7-point Gaussian filter (*nisfilter*). Images were co-registered to the MNI Colin27 template using the Automated Image Registration (AIR3.08) package's default 2^{nd} order model (a 30-parameter nonlinear automated warping algorithm) (Woods, Grafton, Watson, Sicotte, & Mazziotta, 1998; Woods, Mazziotta, & Cherry, 1993) and spatially smoothed using a 6mm FWHM Gaussian filter. Participants were excluded from analysis if >30% of scans showed incremental movement >1 mm or incremental rotation >1°, or if >30% of scans showed absolute movement from baseline >5 mm or absolute rotation >5°. We chose to use more liberal motion criteria based on previous papers in anxious youth who tend to have greater movement (Price et al., 2014). Results of additional analyses with

more stringent motion criteria are reported in Table 4. Raw BOLD signals were converted to percent change from the median of the three runs for each voxel, allowing us to scale the data to a similar baseline across three runs (Price, Paul, Schneider, & Siegle, 2013). Given that BOLD hemodynamic responses can vary based on task and/or brain regions (Handwerker, Ollinger, & D'Esposito, 2004), we did not apply the convolution of the hemodynamic response function because the long duration of each trial enabled slow event-related model free analysis (as in Price et al., 2014; Silk, Lee, Elliott, et al., 2017; Silk, Lee, Kerestes, et al., 2017).

ROIs were anatomically defined using AFNI's Talairach atlas and included the bilateral anterior insula (in the area Y>0), sgACC (BA25), pgACC (BA24; defined in area Y>21) in the rostral cingulate, and VLPFC (BA47). The amygdala region was anatomically defined by hand tracing on the MNI Colin 27 brain (x, y, z = ±23, −4, −17) (as in Siegle, Thompson, Carter, Steinhauer, & Thase, 2007). This region definition differs minimally from a Talairach Atlas based version, with the primary differences being imposing a constraint of 1mm boundaries from the medial and anterior boundaries of the subarachnoid space, ensuring the non-inclusion of peri-amygdaloid cortex, as well as exclusion of extended amygdala regions such as the bed nucleus of the stria terminalis. Adequate intra- and inter-rater reliability for this definition has been established in prior studies (Siegle, Steinhauer, Thase, Stenger, & Carter, 2002). See Figure 1 for ROI illustrations.

BOLD signal within each a priori, anatomically defined ROI was extracted. For each ROI, percent change values were averaged across all scans per physical threat word trial. Next, the percent change value during the pre-stimulus baseline (scan 1 of each trial) was subtracted from their respective trial average to create a physical threat > baseline contrast (as in Conner et al., 2012; Mandell, Siegle, Shutt, Feldmiller, & Thase, 2014; Price et al., 2013; Siegle et al.,

2007). The physical threat>baseline percent change was averaged across all physical threat word trials for each participant and used for final analyses in SPSS and Mplus 7.31 (Muthén & Muthén, 2011). Mean percent signal change values which fell beyond the 1.5 interquartile range from the 25th or 75th percentiles for each ROI were considered between-subject outliers. These outliers were rescaled to the outlier cutoff value to reduce effects of extreme values (Erceg-Hurn & Mirosevich, 2008). For exploratory purposes, whole-brain analyses were also completed showing: 1) main effects of task conditions; and 2) effects of parental coping socialization across the whole-brain. Results are presented in the supplement (see supplement section S1).

Although the condition of interest was physical threat > baseline, the specificity of significant associations with physical threat word processing were assessed. To do so, we ran two supplementary models. The first predicted physical threat > baseline ROI activations while accounting for ROI activations from the neutral > baseline contrast. This allowed us to ensure that any effects due to our variables of interest would be maintained above and beyond the shared effects between neutral word processing and threat word processing. In the second model, we re-ran the final model including neutral word > baseline mean activations for each ROI as additional outcome variables. We would expect that associations between parenting and neural response to neutral words (versus baseline) would emerge, in addition to any associations between parenting and neural response to threat words, if parenting was generally related to word processing, as opposed to threat-related processing specifically.

Analytical Plan

SPSS was used to complete preliminary analyses. Structural equation modeling (Mplus 7.31; Muthén & Muthén, 2011) was used for final analyses using robust full information maximum likelihood (RFIML) estimation in a random effects model. RFIML estimation, in

conjunction with the creation of a single indicator latent variable using the observed parental coping socialization data, allowed for the estimation of standard errors for missing data using log-likelihood values, determined by available information in the model. Therefore, we were able to complete analyses inclusive of the 14 participants (10 anxious, 4 controls) with missing observational data. Preliminary SPSS analyses showed that participants with missing observational data were mostly female (n=12), but did not differ in age, race, adolescent- or parent-reported anxiety severity scores (p>.05).

Parental coping statements, child age, and diagnostic group observed variables were centered. As mentioned above, a coping socialization latent factor was created, allowing the single indicator (parental coping statements observed variable) to fully load onto the latent factor, while constraining the indicator's residual variance to zero. This preserves the measurement of the observation data, while also enabling the utilization of the variance and covariance of this indicator variable to estimate model results for the entire sample, including those with missing data. Dependent variable data points (i.e., mean signal activation to threat) were found to have very small variances. Therefore, these were multiplied by 10 to ensure that deviations in variances and standard errors were not missed due to rounding estimations in MPlus. We first conducted exploratory analyses to assess for the effects of adolescent age in the model. In this model, interaction terms for coping socialization latent factor X group, coping socialization latent factor X age, and group X age were created. A 3-way interaction term was also created for coping socialization latent factor X age X group. Each ROI activation was regressed on child age, group, the coping socialization latent factor, and all interaction terms in a single SEM model.

If interaction terms including adolescent age were not significantly associated with neural response to physical threat in any ROI, they were not included in the final model for parsimony. Within the final model, we corrected for the number of ROIs assessed using the false discovery rate correction (FDR; Benjamini & Hochberg, 1995). Because our main hypotheses tested whether the relationship between parenting and neural response to threat differed based on adolescent clinical status, we used the significance tests for these coefficients to control for multiple comparisons. All parental coping socialization X group interaction-term significance statistics (i.e. p-values) from the SEM model were entered into an FDR correction calculator which calculated FDR-significance thresholds and FDR-adjusted p-values (a.k.a. q-values). If the FDR-adjusted p-value was less than its corresponding FDR-significance threshold, then the result was considered to pass the test for multiple comparisons. If significant interaction effects passed FDR correction (error rate $p<.05$), the interactions were probed using two individual, within-group (anxious, controls) models.

Post-hoc, within-group analyses used an SEM modelling approach to assess coping socialization, ROI activation, and adolescent disengaged coping associations for anxious and control groups, separately. As in the initial full model, a single indicator (coping socialization) latent factor was used. Adolescent age was entered as a predictor in the model. Five participants did not report a negative event with distress levels of 3 or more and two participants had missing data, leaving a total of 113 individuals with reports of at least one negative event with distress level of 3 or more. Given the use of the SEM analytical approach and its ability to handle missing data, we were able to include all participants in the analyses. Based on available EMA data, avoidance/suppression and distraction coping variables were significantly and positively correlated within both groups (anxious: n=83; $r=.360$, $p=.001$; control: n=30; $r=.672$, $p=.000$).

Avoidance/suppression and distraction coping variables from the EMA data were therefore used as indicator variables to create a single "disengaged coping" latent factor. The avoidance/suppression and distraction indicator variables were allowed to fully load onto the latent factor. Therefore, only variance that is shared with between the two strategies would load onto the latent factor. Residual variances of the indicator variables were allowed to freely vary. For each group, we regressed coping socialization on ROI variables found to have significant interaction effects in the initial full model. Models were run using standard maximum likelihood estimation and bias-corrected bootstrapping with 5000 samples. Utilizing a bootstrapping procedure allowed us to probe for indirect effects of parental coping socialization on adolescent coping through ROI activation and ensured the estimation of stable parameter estimates in models with lower sample sizes. Model fit for these post-hoc models were evaluated using standard fit indices and cutoff criteria [χ^2, $p>.05$; RMSEA<.05; CFI/TLI>.95; SRMR<.08). Unstandardized parameters and bias-corrected bootstrapped confidence intervals (CI, upper 2.5%, lower 2.5%) were used to determine significance of path estimates for these models.

Results

Preliminary Analyses

Descriptive statistics for all ROIs are reported in Table 2. All ROIs were significantly and positively correlated with each other. There were no significant bivariate correlations between gender, socioeconomic status (i.e. total household income), or race and ROI activation or coping socialization (*p*'s>.05), therefore these were left out of models for parsimony (Table 3 for correlations). Adolescent age was correlated with the parental coping socialization observed variable (*r*=-.248, *p*=.01). Based on results from the exploratory model, adolescent age was not shown to moderate the effects of parental coping socialization (Bs = -.048-.066, SEs =.043-.066,

p's >.05) or diagnostic group (Bs = -.157-.042, SEs = .075-.111, *p's* >.05) on neural response to threat in any ROI. Parental coping socialization X group X age interaction effects were also non-significant (Bs = -.030-.169, SEs =.096-.144, *p's* >.05). A significant main effect of child age was found on pgACC response to threat (B=.084, SE=.038, p=.027). Therefore, age was included as a covariate in all final models. We also re-ran the model to assess effects of pubertal status, in place of age, which yielded no main or interaction puberty-related effects on neural response to threat (*p's*>.05). Given that significant effects due to interactions with adolescent age or puberty were not found in the exploratory models, these were dropped from the final full model and within-group post-hoc models for parsimony. Parental coping socialization did not differ between groups (*t*=1.095, *p*=.276).

ROI Analyses

Full model (Table 4). Greater parental coping socialization was exhibited with younger adolescents (B=-.279, SE=.121, *p*=.021). No significant main effects of either parental coping socialization or diagnostic group on neural response to threat (relative to baseline) in any ROIs were found (*p*'s>.05). Adolescent age was significantly associated with response to threat in the pgACC (B=.079, SE=.037, *p*=.034). Controlling for multiple comparisons, significant coping socialization X group interaction effects were found in the bilateral anterior insula (L: *B*=-.432, SE=.159, $p_{\text{-FDR threshold}} \leq .007$; R: *B*=-.417 SE=.171), $p_{\text{-FDR threshold}} \leq .019$), and pgACC (*B*=-.429, SE=.169, $p_{\text{-FDR threshold}} \leq .013$; see Figure 2 for interaction illustration and participant data points). Results of the specificity analyses showed that significant interaction effects were maintained with regard to physical threat word processing when controlling for activation to neutral word processing. Furthermore, no significant effects of parental coping socialization X group were

found with regard to neural processing of neutral words when added as outcomes to the model (details in Supplement S2, Tables S2.1 and S2.2).

There was one participant with an outlier data point for parenting, therefore we re-ran the model treating the parenting data point for this participant as a missing data point. Excluding this data point from analyses did not yield significantly different results from the original models. Parenting coping socialization X group effects still remained in the bilateral anterior insula (L: B=-.428, SE=.167, p=.010; R: B=-.440, SE=.174, p=.012) and pgACC (B=-.457, SE=.174, p=.008). Therefore, the final results are based on fully available original parenting data.

Post-hoc within anxious group model (Figure 3a). Within the anxious group, the post-hoc model probing interaction effects evidenced excellent fit ($\chi^2_{(4)}$=2.65, *p*=.62; RMSEA=.00; CFI=1.00; TLI=1.00; SRMR=.022). No significant associations were found between adolescent age and coping socialization or adolescent coping (*p's*>.05). Controlling for adolescent age, coping socialization was positively associated with activation to threat stimuli in the anterior insula (L: β=.368, B=.255, (.078), *p*=.001; R: β=.303, B=.217 (.084), *p*=.010) and the pgACC (β=.292, B=.220 (.083), *p*=.008). Adolescent disengaged coping was also independently associated with ROI activations in the anterior insula (L: β=-.308, B=-1.316 (.631), *p*=.037; R: β=-.283, B=-1.258 (.633), *p*=.047) and the pgACC (β=-.364, B=-1.695 (.714), *p*=.018). Although, coping socialization was not significantly associated with adolescent coping (*r*=.105, B=.011 (.018), *p*=.532), given the independent effects found between ROI activations and both coping socialization and adolescent coping, indirect effects of coping socialization on adolescent disengaged coping through neural activation in the bilateral anterior insula and pgACC were tested. Unique contributions of indirect paths were tested for each ROI independently. Results of bootstrapping showed significant indirect effects through the left anterior insula (β=-117, B=-

.019 [CI: -.060, -.002]), the pgACC (β=-.106, B=-.017 [CI: -.056, -.001]), and a trend through the right anterior insula (β=-.085, B=-.014 [CI: -.047, .000]).

Given the high correlations found between these three regions (*r's*=.627-.787), we also tested whether significant indirect effects could be due to the shared variance among all three brain regions during threat word processing. This was tested by allowing the variances of neural activation from the bilateral anterior insula and the pgACC to freely load onto a single latent factor for neural threat processing. The neural threat processing latent factor was regressed onto parental coping socialization, controlling for adolescent age. The adolescent coping latent factor was next regressed onto the neural threat processing latent factor. Again, bias-corrected bootstrapping was conducted to test for indirect effects. This model evidenced excellent fit ($\chi^2_{(10)}$=10.84, *p*=.37; RMSEA=.03; CFI=1.00; TLI=.99; SRMR=.04). Results showed that coping socialization was significantly and positively associated with the neural threat processing latent factor (β=.351, B=.213 [CI: .073, .357]). The neural threat processing latent factor was also significantly and negatively associated with adolescent disengaged coping (β=-.438, B=-.129 [CI: -.292, -.021]). When accounting for neural threat processing, parental coping socialization was not significantly associated with adolescent coping (β=.268, B=.048 [CI: -.015, .128]). The model showed support for a significant indirect effect of parental coping socialization on adolescent disengaged coping through the neural threat processing latent factor (β=-.154, B=-.028 [CI: -.077, -.004]).

Post-hoc within control group model (Figure 3b). Within the control group, the post-hoc model examining interaction effects evidenced good fit ($\chi^2_{(4)}$=4.86, *p*=.30; RMSEA=.08; CFI=.99; TLI=.97; SRMR=.24). Adolescent age was not significantly correlated with adolescent coping (*p*>.05), but was negatively correlated with parental coping socialization (*r*=-.512, B=-

.651 (.261), *p*=.013). Controlling for adolescent age, coping socialization was significantly and negatively associated with activation to threat stimuli in the right anterior insula (β=-.608, B=-.488 (.158), *p*=.002) and pgACC (β=-.517, B=-.421 (.164), *p*=.010), but not in the left anterior insula (β=-.303, B=-.254 (.176), *p*=.150). No independent effects of adolescent disengaged coping were found on any ROI activations (*p's*>.05). Parental coping socialization was not significantly associated with adolescent coping (*r*=-.097, B=-.020 (.042), *p*=.627). Because there were no independent effects found between ROI activations and adolescent coping, indirect effects through neural activation in ROIs were not tested.

Discussion

When parents use coping socialization strategies that encourage youth to face challenges and help them to reframe perceived threats, positive adolescent adjustment is more likely, including lower internalizing symptoms and better treatment response among anxious adolescents (Morris et al., 2007; Silk et al., 2013). With the use of laboratory observations, findings from the current study indicate that engagement-oriented coping socialization behaviors are also associated with early-adolescents' neural activity in neural regions associated with threat processing, including the anterior insula and pgACC. Contrary to theory positing differences in how parents of anxious youth might respond to their children's affect in challenging situations, we found no evidence in the current sample that parents of anxious adolescents utilize less coping socialization behaviors, compared to parents of healthy adolescents, during anxiety-provoking interactions with their adolescents. However, we did find that the relationship between coping socialization and early-adolescent neural activity during threat processing differed between anxious and non-anxious youth. Furthermore, we found evidence suggesting that greater parental coping socialization was indirectly associated with lower reliance on disengaged coping

strategies in response to negative daily events through greater activation in the bilateral anterior insula and pgACC activation. Although parenting was associated with neural activation to threat in healthy youth, the indirect effects were specific only to the group of anxious youth. Overall, the results of this study provide novel evidence that specific engagement-oriented coping socialization behaviors and verbalizations made by parents to help scaffold adaptive coping in early-adolescence are associated with both neural activity to threat-related information and levels of adolescent disengaged coping in the real world.

In this sample, parents of anxious youth were observed to provide the same level of engagement-oriented coping socialization during interactions as parents of healthy adolescents. Therefore, we did not find support for the theory that parents of anxious youth may be less inclined to encourage their youth to reframe, problem-solve, and face fearful situations. However, our results suggest that youth who exhibit greater reactivity, including those with anxiety, may be more responsive to or reliant upon their parents' behaviors to help guide their own behavior, than less reactive youth. Anxious adolescents whose parents exhibited more coping socialization showed higher anterior insula and pgACC activation in response to threat stimuli. Interestingly, these neural patterns of activation were directly related to less adolescent disengaged coping. In addition, parental effects of coping socialization were indirectly associated with disengaged coping behavior in early-adolescents through both the unique and shared effects of activation in the anterior insula and pgACC. The anterior insula is a functionally complex brain region that has been implicated in a diverse range of cognitive control and emotional processes (Uddin, Kinnison, Pessoa, & Anderson, 2014). For example, the anterior insula has been associated with increased visceral response, and awareness and experience of emotion (Singer et al., 2009), while it has also been shown to play an important role in the integration of

information and assisting cognition by supporting flexibility of neural engagement of various brain networks, such as the executive network and the default-mode network (Uddin et al., 2014). The pgACC has also been associated with emotion regulation, including perception of social/physical pain and fear extinction, and is also densely connected with many brain areas, including the anterior insula (Etkin et al., 2011; Posner et al., 2007).

Our results could indicate that as early-adolescents with anxiety are exposed to greater scaffolding by parents' coping socialization behaviors, greater anterior insula and pgACC engagement might be reflecting *both* heightened and likely aversive emotional reaction in response to threat *and* greater recruitment of neural regions that support cognitive control processes in response to threat stimuli. A few studies have also found that anxious adolescents may rely more heavily on neural regions implicated in regulation during threat processing, compared to healthy youth (McClure, Monk, Nelson, & et al., 2007; Monk et al., 2008; Telzer et al., 2008). Our findings may similarly suggest that anxious adolescents who recruit both the anterior insula and pgACC tend to rely less on disengagement coping strategies in response to negative events. Interestingly, our results showed that there were indirect effects of parenting on early-adolescent coping through the shared variance among these regions during threat processing. This could indicate that, not only are there unique effects for each of these neural regions, but importantly there is a shared underlying process through which all three of these regions may similarly contribute to both process threat and lower anxious adolescents' reliance on disengaged coping. Furthermore, teaching youth to engage with threatening challenges is a major objective of CBT treatment for anxiety (Chu & Harrison, 2007). Silk and colleagues (2013) have shown that parental encouragement to approach fears leads to better CBT treatment outcomes in anxious adolescents. It is thus possible, given our results, that activation of the

anterior insula and/or the pgACC may be involved in this process. Indeed, higher pre- and post-treatment insula activation during threat processing in anxious adults and adolescents has also been related to better brief-CBT and mindfulness-based CBT response, respectively (Reinecke, Thilo, Filippini, Croft, & Harmer, 2014; Strawn, Cotton, et al., 2014). It therefore may be that similar cognitive processes targeted during CBT therapies are also supported by coping socialization that encourages engagement-oriented coping.

In contrast to the findings for anxious adolescents, we found that as parents of healthy youth exhibited more coping socialization, these early-adolescents showed lower anterior insula and pgACC reactivity to threat, though no associations between brain function and adolescent coping in daily life were found. Given the role of the anterior insula in both emotional and cognitive processes (Uddin et al., 2014), it may be that when healthy youth are exposed to greater levels of coping socialization, threat words are not perceived as salient and/or as threatening, decreasing the need for insula engagement. Furthermore, the pgACC has been associated with emotion regulation, including perception of social/physical pain and fear extinction, and it is also densely connected with limbic brain areas, including the anterior insula (Etkin et al., 2011; Posner et al., 2007). Thus, in the current study, lower pgACC activation in the healthy adolescents exposed to more coping socialization may reflect less need to recruit pgACC to extinguish threat processing. Alternatively, work in cognitive developmental neuroscience has supported that as neural processes mature, they become more focal (Luna et al., 2010). Consequently, an alternative interpretation could be that the reduced activation of the anterior insula and pgACC in healthy adolescents, whose parents exhibit more engagement-oriented coping socialization, reflects more efficient threat processing. These hypotheses would need to be tested further in future studies.

Despite the strengths of the current study, there were several limitations. The study was limited by the small size of our control sample, as anxious youth were oversampled due to the treatment study design. We were also not able to assess ethnic differences because the sample used in the study was primarily Caucasian. It is also important to note that the group of early-adolescents with anxiety in this sample had to meet strict inclusion criteria to be accepted into the larger child anxiety treatment study. Consequently, the youth included in this study had lower rates of comorbidity than is typically seen in anxiety studies. It will be important for future work to extend these investigations using adolescent samples who have higher rates of comorbid diagnoses.

Although the aim of the current study was to elucidate the ways in which parental coping socialization might impact the functioning of neural regions supporting threat processing in healthy and anxious early-adolescents, neither causation nor directionality could be inferred as this study was cross-sectional. Researchers might consider employing an experimental design in future studies, in which adolescents are presented with parental coping socialization statements that encourage both approach and avoidance of threat while in the scanner. This could possibly enable the investigation of more real-time, moment-to-moment differences in brain response to threat stimuli directly following specific parental coping socialization prompts. Importantly, we also acknowledge the important consideration of bi-directional parent-child effects. Previous research has shown that child characteristics, such as fearful and irritable temperament, can predict later parenting behavior (e.g., acceptance and use of discipline) (Lengua & Kovacs, 2005). Therefore, it is possible that parental behaviors may have been driven by child characteristics, such as reactivity in the anxious sample. For example, results in this study could be interpreted as suggesting that anxious youth who have greater neural reactivity to threat might

elicit more coping socialization behaviors from their parents. Future research using observational methods should examine whether anxious adolescents actively seek support from their parents or if parents initiate support without adolescent prompting. This could help to shed more light on the directionality of parent-adolescent behaviors.

In addition, our fMRI task included a relatively low number of trials per condition, possibly increasing the signal-to-noise ratio. The task also did not require adolescents to actively down-regulate negative affect through prescribed strategies, such as reappraisal. Future work might focus on how the effects of parental coping socialization could affect neural activation in prefrontal cortical regions implicated in voluntary emotion regulation and reappraisal processes, including the dorsolateral prefrontal cortex and the posterior parietal lobe (Buhle et al., 2014). Finally, we utilized relatively liberal motion correction criterion to retain the largest sample size and maximize the power to test the study hypotheses. It is important to note that in addition to absolute motion correction parameters, we did also exclude participants that showed incremental movement using a conservative threshold (>1 mm or >1°). Relatively lenient absolute motion correction criteria is somewhat commonly used in other studies examining neural activation in younger, clinical samples (for examples, see Forbes, Phillips, Silk, Ryan, & Dahl, 2011; Price et al., 2016), though more effective participant training and simulation procedures should be used in future studies examining neural activation in similar samples of early-to-mid-adolescents from clinical populations.

In sum, the findings from this study show that parenting behaviors that help youth learn to cope are related to patterns of neural activation associated with processing of threat-related information during early-adolescence. This suggests that parents have the potential to engage in specific strategies that may scaffold the adolescent brain to effectively process threat and cope

with future challenges. Findings specific to anxious youth indicate that the ways in which parents socialize engagement-oriented coping is related to lower adolescent reliance on potentially maladaptive disengaged coping strategies through the functioning of particular neural regions (i.e., anterior insula and pgACC) during threat processing. This suggests that incorporating parent-coaching modules that teach parents how to socialize engagement-oriented coping strategies in the home could potentially improve treatment outcomes for clinically anxious early-adolescents through shifts in the adolescents' threat processing (Ginsburg & Schlossberg, 2002). Researchers should consider investigating this through longitudinal family-based intervention designs that also incorporate neuroimaging at multiple timepoints. In addition, future studies may help to increase our understanding of the relative effectiveness of each parenting behavior in scaffolding adaptive coping in anxious youth by assessing the effects of coping socialization behaviors separately, rather than collectively as in the current study.

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Table 1. Adolescent participant demographic and clinical characteristics by group

	Anxious (*n*=87)	Control (*n*=33)	*t*-statistic/χ^2
Age [*M* (*SD*)]	11.36 (1.45)	11.74 (1.68)	1.24
Gender (% F)	60.9	54.5	.402
Race (%)			8.84*
White (non-Hispanic)	89.7	69.7	
Black	4.6	18.2	
Hispanic	1.1	6.1	
Biracial	4.4	6.0	
Family income ($k) [*M* (*SD*)]	89.40 (78.34)	73.22 (38.16)	-.84
SCARED [*M* (*SD*)]			
Adolescent report	38.54 (11.69)	9.93 (7.71)	-12.81***
Parent report	35.41 (12.38)	3.44 (3.05)	-14.41***
Anxiety Diagnosis (%)			120.00***
Generalized anxiety disorder	70.5	0	
Social anxiety disorder	27.3	0	
Separation anxiety disorder	20.5	0	
Panic disorder	2.2	0	
Specific phobia	9.1	0	
Comorbid Diagnosis (%)			
Major depressive disorder	1.1	0	
Tourette syndrome	1.1	0	
Attention deficit hyperactivity disorder [a]	3.4	0	
Oppositional defiant disorder	1.1	0	
Enuresis	1.1	0	
Other	2.2	0	
Negative events reported with $\geq$ 3 distress [M (SD)]	8.58 (3.71)[1]	6.33 (3.94)[2]	2.90**
Suppression/avoidance use [proportion of negative events, *M (SD)*]	.70 (.30)[1]	.60 (.39)[2]	.61
Distraction use [proportion of negative events, *M (SD)*]	.46 (.27)[1]	.42 (.33)[2]	1.34
Parental Coping Socialization [RPM; *M (SD)*]	1.20 (.81)[3]	1.01 (.74)[4]	1.095

*p<.05, **p<.005, ***p<.001; [a] Inattentive subtype; [1] n=85; [2] n=30; [3] n=77; [4] n=29; Note: SCARED=Screen for Child Anxiety Related Disorders, RPM=Rate/minute, ROI=Regions of interest, L=Left, R=Right; sgACC=Subgenual anterior cingulate, pgACC=Perigenual anterior cingulate, VLPFC=Ventrolateral prefrontal cortex

Table 2. Descriptive statistics for ROI BOLD activation by group

	Anxious (*n*=87)	Control (*n*=33)	*t*-statistic/χ^2
ROI Percent Change [*M* (*SD*)]			
Amygdala L	-.00027 (.073)	-.01849 (.075)	1.21
Amygdala R	.00055 (.075)	-.01356 (.067)	.949
Anterior Insula L	.01791 (.056)	.01084 (.065)	.586
Anterior Insula R	.02245 (.059)	.01910 (.063)	.274
sgACC	-.00609 (.077)	-.01979 (.072)	.889
pgACC	-.00625 (.061)	-.01344 (.064)	.566
VLPFC L	.00648 (.048)	.00303 (.055)	.337
VLPFC R	.01602 (.059)	.02271 (.060)	-.548

Table 3. Correlations of adolescent characteristics and neural ROIs across full sample (*N*=120)

		1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1.	Gender	1											
2.	Race	-.024	1										
3.	Household Income	-.196	-.084	1									
4.	Age	-.111	-.032	.041	1								
5.	L Amygdala	.121	-.014	.080	.051	1							
6.	R Amygdala	.092	.017	.114	.024	.849**	1						
7.	L Anterior Insula	-.029	.040	.114	.055	.668**	.563**	1					
8.	R Anterior Insula	.010	.074	.077	.075	.649**	.629**	.813	1				
9.	Subgenual Cingulate (BA25)	.085	.070	.010	.137	.680**	.633**	.522**	.512**	1			
10.	Perigenual Cingulate (BA24)	-.124	.046	.033	.198*	.526**	.481**	.700**	.700**	.560**	1		
11.	L VLPFC (BA47)	-.077	-.004	.046	.104	.700**	.631**	.752**	.742**	.643**	.716**	1	
12.	R VLPFC (BA47)	-.047	.013	-.059	.111	.649**	.662**	.634**	.754**	.631**	.644**	.864**	1

*p<.05, **p<.001; Note: L=left, R=right, BA=Brodmann area, VLPFC=ventrolateral prefrontal cortex

TABLE 4. Unstandardized effects of parental coping socialization on neural activity to physical threat words (relative to baseline), controlling for adolescent age (*N*=120)

	B	***SE***	***p-uncorr***	***p-FDR Adjusted*** (i.e. q-values)
Amygdala (L)				
Age	.030	.046	.523	
Parental coping socialization	.022	.097	.820	
Group	-.200	.151	.185	
Parental coping socialization x Group	-.142	.227	.531	.531
Amygdala (R)				
Age	.005	.046	.908	
Parental coping socialization	-.044	.093	.631	
Group	-.162	.149	.276	
Parental coping socialization x Group	-.191	.206	.354	.405
Anterior Insula (L)				
Age	.024	.036	.503	
Parental coping socialization	.109	.072	.131	
Group	-.093	.119	.439	
Parental coping socialization x Group	-.432[a,b]	.159	.007	.040
Anterior Insula (R)				
Age	.026	.037	.473	
Parental coping socialization	.066	.076	.385	
Group	-.062	.122	.614	
Parental coping socialization x Group	-.417[a,b]	.171	.015	.040
Subgenual Cingulate (BA25)				
Age	.064	.046	.165	
Parental coping socialization	.022	.097	.824	
Group	-.185	.153	.228	
Parental coping socialization x Group	-.394	.213	.064	.128
Perigenual Cingulate (BA24)				
Age	.079	.037	.034	
Parental coping socialization	.060	.075	.423	
Group	-.122	.124	.329	
Parental coping socialization x Group	-.429[a]	.169	.011	.040
Ventrolateral prefrontal cortex (L; BA47)				
Age	.036	.031	.249	
Parental coping socialization	.058	.065	.373	
Group	-.054	.102	.593	
Parental coping socialization x Group	-.222	.151	.140	.187
Ventrolateral prefrontal cortex (R; BA47)				
Age	.035	.037	.347	
Parental coping socialization	.016	.078	.833	
Group	.036	.122	.765	
Parental coping socialization x Group	-.292	.179	.104	.166

Note: L=left, R=right, BA=Brodmann area; [a] Standardized coefficients were comparable when imaging data was dropped if: >10% scans had absolute movement from baseline >5 mm/5°; [b] Standardized coefficients were comparable when imaging data was dropped if: >30% scans had absolute movement from baseline >2 mm/2°

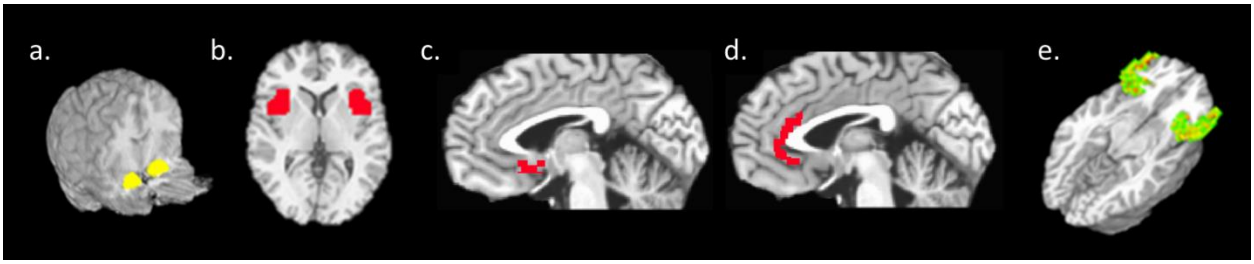

Figure 1. Masks of anatomically defined regions of interest. (a) bilateral amygdala, (b) bilateral anterior insula, (c) subgenual cingulate cortex (BA25), (d) perigenual cingulate cortex (BA24); (e) bilateral ventrolateral prefrontal cortex (BA47).

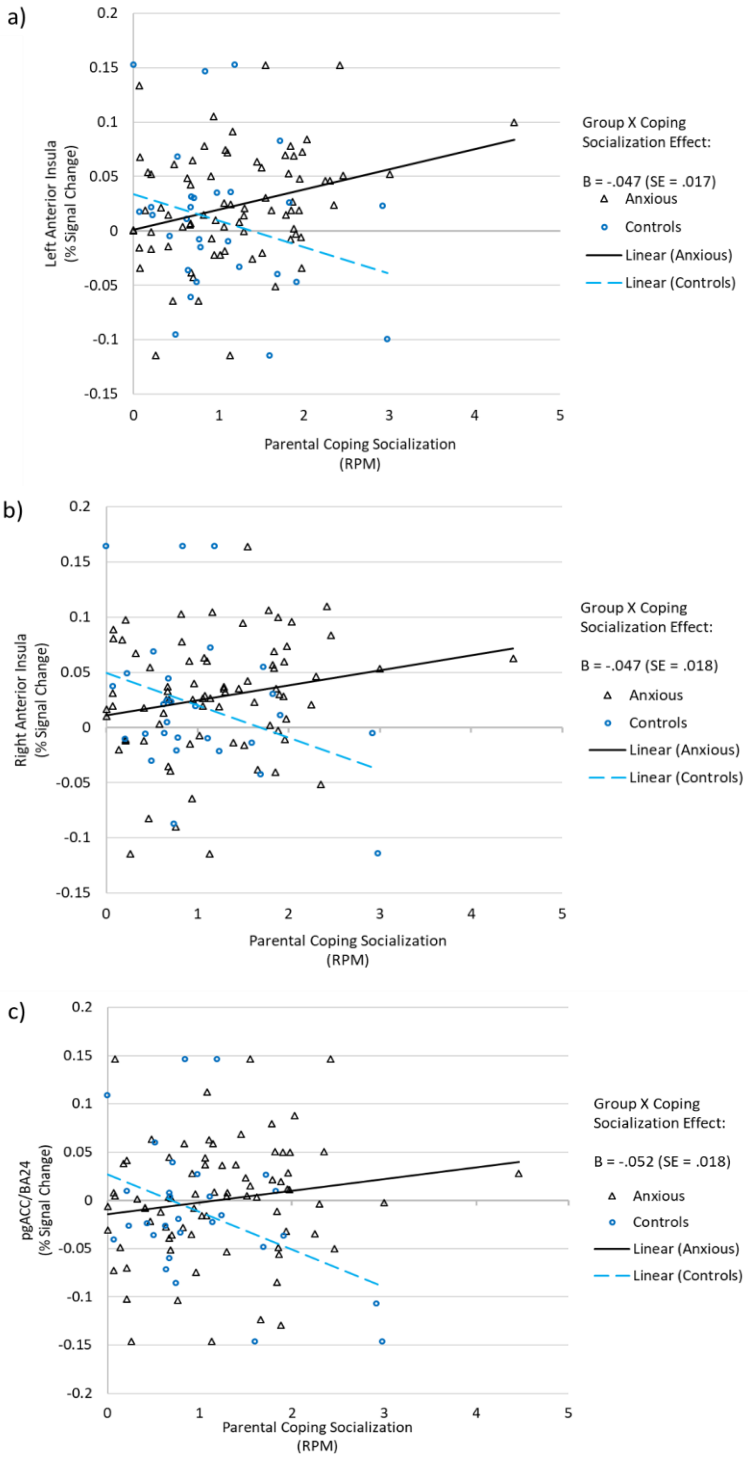

Figure 2. Significant parental coping socialization statement use X clinical group interaction effects on ROI activation for physical threat>baseline contrast are illustrated for participants with full data available (*n*=106). Panel: a) left anterior insula, b) right anterior insula, and c) pgACC (BA24). Note: Regression statistics shown for each interaction effect were estimated in the final SEM model which included the full sample (*n*=120); pgACC=perigenual cingulate.

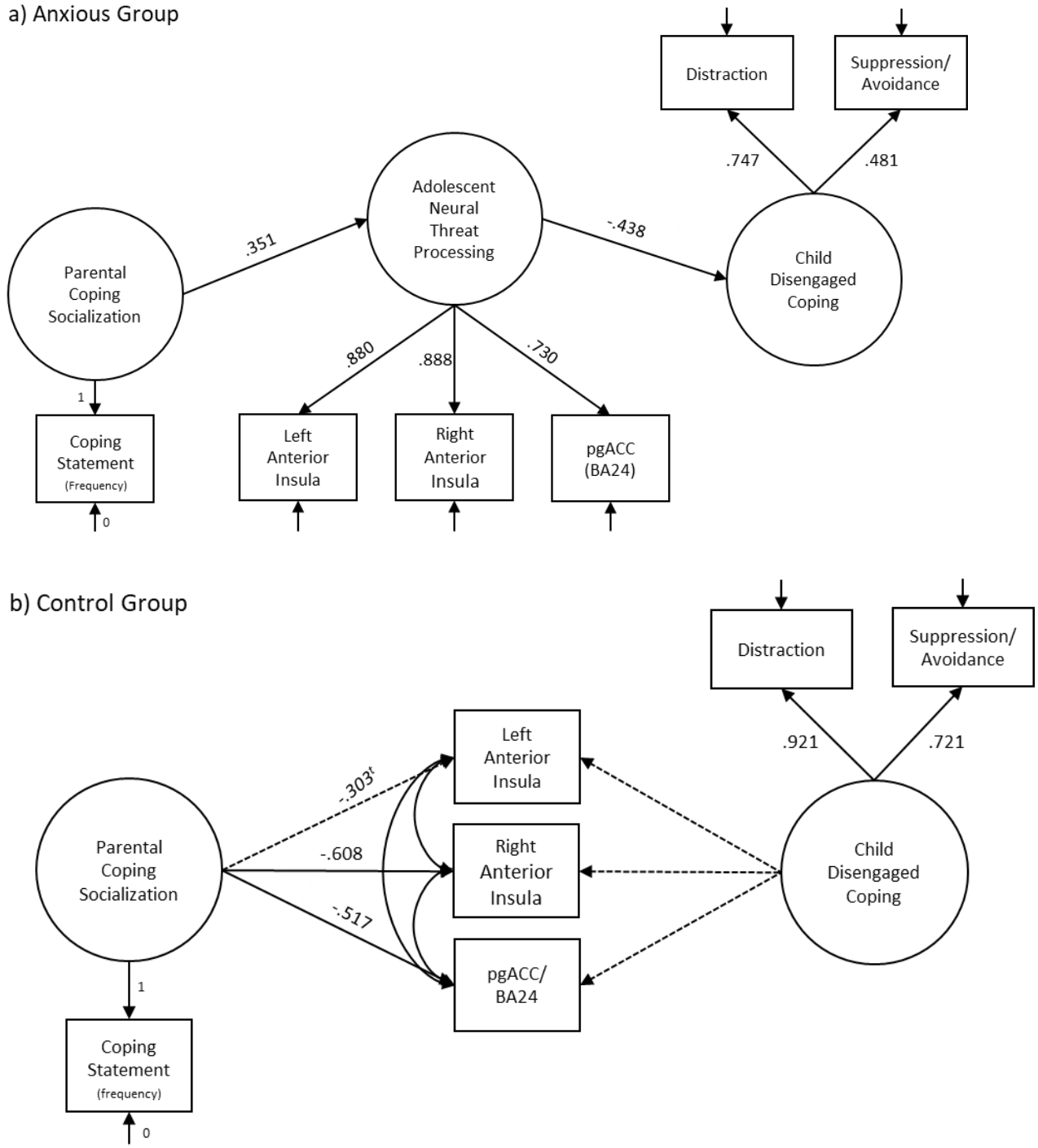

Figure 3. Post-hoc, within-group SEM models, including standardized beta coefficients, for: a) anxious adolescent group, b) healthy adolescent group. Note: $t \leq .10$; Solid lines=significant paths ($p_{FDR} \leq .05$), Dashed lines=non-significant paths; pgACC=perigenual cingulate cortex.