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The Relation between Parent Depressive Symptoms and Neural Correlates of Attentional Control in Offspring: A Preliminary Study

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Abstract

Offspring of depressed parents are at significantly elevated risk for depressive disorders themselves; however, the specific mechanisms associated with this risk are not well known. Given the well-established link between parental depression and biased attention for negative stimuli in children, this preliminary study sought to examine the neural correlates of directing attention *toward* and *away* from emotional faces in children and adolescents in association with parent's current depressive symptoms. Forty-two youth (age 7–18 years) completed a task with conditions that manipulated whether participants were instructed to match emotional faces (explicit emotion processing) or match shapes in the context of emotional face distractors (implicit emotion processing) during functional magnetic resonance imaging. Results revealed a positive correlation between parent depressive symptoms and youths' recruitment of the dorsal anterior cingulate

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Contributors

K. L. Burkhouse, A. Kujawa, K. P. Fitzgerald, C. S. Monk, and K. L. Phan developed the initial study concept and design. K.L. Burkhouse, A. Kujawa, and K. L. Phan developed and analyzed study hypotheses. K.L. Burkhouse had primary responsibility for drafting the paper. All authors revised the manuscript and approved the final version for submission.

Conflict of Interest

The authors report no financial interests or potential conflicts of interests.

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cortex (ACC) during attempts to direct attention away (implicit processing) from negative faces. These findings were maintained after accounting for current anxiety and depressive symptoms among youth suggesting that the findings were not fully attributable to offspring's psychopathology. These preliminary findings suggest that altered dorsal ACC function may contribute to the biased attention for negative information typically demonstrated in youth at high risk for depression by disrupting efficient inhibition of negative stimuli.

Keywords

parental depression; emotion processing; vulnerability; anterior cingulate cortex; intergenerational transmission

1. Introduction

Having parents who have experienced depression is associated with a three- to fivefold increase in the risk to the offspring for developing a depressive episode by adolescence (Goodman et al. 2011; Weissman et al. 2016). Despite decades of research documenting this risk, the mechanisms associated with this vulnerability are not well understood. Consistent with cognitive models of depression (Clark and Beck, 1999), several studies suggest that parental depression is associated with biased cognitive-emotional processing for negative stimuli in offspring. Specifically, youth of parents with a history of depression exhibit biased attention for negative stimuli, including an initial orienting of attention towards and later difficulty diverting attention away from negative stimuli (Joormann et al. 2007; Kujawa et al. 2011).

It has been suggested that information processing biases develop in response to early stress and adversity (e.g., Ingram, 2003), in association with early individual differences in emotional reactivity and regulation (e.g., Mauer and Borkenau, 2007), and as a result of genetic influences (e.g., Beevers et al. 2009; Gibb et al. 2009). It has also been theorized that experiences during childhood may contribute to the development of "experience-specific" information processing biases (Cicchetti et al. 2000; Pollak, 2003; Rose and Abramson, 2002). That is, offspring of parents with higher levels of depression may be more likely to experience an attention bias that is specific for negative stimuli due to consistent overexposure to negative affect displayed by the parent (Gibb et al. 2009). Despite several studies documenting this bias for negative stimuli at the behavioral level, few studies have attempted to uncover the neural correlates of this negative emotion processing bias in relation to parent's depression.

There is some evidence from a small number of studies that children and adolescents of parents with a history of depression exhibit altered activation in limbic and prefrontal regions when passively viewing negative information. For example, adolescents of depressed parents exhibit decreased dorsolateral prefrontal cortex (PFC) activation when viewing fearful faces, compared to youth of nondepressed parents (Mannie et al. 2011). Other cross-sectional (Monk et al. 2008) and longitudinal (Swartz et al. 2015) studies highlight overactivation of the amygdala in response to directly viewing fearful faces among youth of depressed versus nondepressed parents. Although these prior studies help to provide insight

on the neural mechanisms associated with explicit emotion processing in youth at high risk for depression, the neural correlates of *implicit* emotion processing (i.e., diverting attention away from negative stimuli) remain unknown. One possibility is that risk for depression is characterized by altered activation of brain regions implicated in cognitive control. The anterior cingulate cortex (ACC) has been extensively linked to individuals' ability to shift attention away from emotional information (Etkin et al. 2011). In particular, portions of the rostral and dorsal ACC have been shown to be related to conflict monitoring and ability to down-regulate responses to negative emotional distractors (e.g., Etkin et al. 2006, 2011). Notably, adults with current depression, compared to healthy controls, recruit greater rostral and dorsal ACC when attempting to filter out negative stimuli (Disner et al. 2011; Elliott et al. 2002; Eugène et al. 2010; Mitterschiffthaler et al. 2008). It has been suggested that individuals with depression require greater cognitive effort to successfully inhibit negative information and thus recruit regions involved in attentional control to a greater extent relative to healthy individuals (for a review, see Disner et al. 2011).

In this preliminary study, we sought to extend the existing research by examining the association between parental depressive symptoms and neural correlates of *both* explicit and implicit emotion processing in offspring. We focused on children and adolescents who had no past or current diagnosable depressive disorders to rule out possible “scar” effects of a previously experienced episode. We included a sample with and without anxiety disorders in order to encompass a range of ‘at-risk’ youths given that children with a history of anxiety are at elevated risk for developing depression (e.g., Orvaschel et al. 1995; Silk et al. 2012). Youth completed the Emotional Faces Shifting Attention Task (EFSAT; Klumpp et al. 2012, 2013, 2014; Swartz et al. 2014) in which images comprising a trio of geometric shapes (circles, rectangles, triangles) were presented alongside a trio of emotional facial expressions (angry, fearful, happy), and the participant was prompted to match either faces or shapes. The task provides a measure of neural correlates of both explicit and implicit emotional processing, as the instruction to “Match Faces” requires individuals to direct attention *toward* emotional faces, whereas “Match Shapes” requires participants to direct attention *away* from emotional faces, respectively. In a previous study, we showed that healthy and anxious youth recruit limbic regions, such as the amygdala, when matching faces, and the ACC when matching shapes (i.e., directing attention away from emotional faces) (Swartz et al. 2014).

Consistent with previous studies (Monk et al. 2008; Swartz et al. 2015), we predicted that activation of the amygdala when directing attention toward negative stimuli among youth would be positively associated with parental depressive symptoms. During implicit emotion processing (i.e., directing attention away from negative emotion stimuli), consistent with adult depression studies (for a review, see Disner et al. 2011), we predicted that parent depressive symptoms would be positively associated with activation of the dorsal and rostral ACC among youth. We predicted that these effects would be maintained even after accounting for variance due to current anxiety and depressive symptoms in children and adolescents, indicating that the findings may be specific to having a parent with high depressive symptoms versus youth's own internalizing psychopathology. Finally, exploratory analyses were conducted to determine whether the relation between parent depressive symptoms and neural correlates of explicit and implicit emotion processing in

youth would be moderated by child anxiety status. We also examined whether youth's age would moderate the findings given previous documented effects of age on neural responses during explicit emotion processing (i.e., MacNamara et al. 2015; Swartz et al. 2015).

2. Methods

2.1 Participants

Participants were youth (Age Range 07–18; $M = 13.81$, $SD = 02.82$) participating in an anxiety treatment study at the University of Michigan (UM) and the University of Illinois at Chicago (UIC). Participant diagnoses were obtained through administration of the Kiddie-Schedule of Affective Disorders and Schizophrenia for School-Age Children (K-SADS-PL) (Kaufman et al. 1997) clinical interview administered by Master's or Doctoral level clinicians (see Kujawa et al. 2015 for more details). Exclusion criteria for the current study included history of depressive disorder, bipolar disorder, schizophrenia, intellectual disability, pervasive development disorders, current substance use disorders, or suicidal ideation. Fifty-four youth completed the Emotional Faces Shifting Attention Task. Of these fifty-four youth, twelve subjects were excluded from the analysis due to excessive motion (i.e., having any TR to TR movement exceeding 02mm, or 03 consecutive TRs exceeding the same in any direction - pitch, roll, or yaw). Thus, the final included sample consisted of forty-two youth. Of these individuals, twenty-four had a current anxiety disorder (primary diagnosis of generalized anxiety disorder, $n = 16$; primary diagnosis of social anxiety disorder, $n = 06$; primary diagnosis of separation anxiety disorder, $n = 02$) and eighteen had no history of any DSM-IV Axis-I disorder. Participants were not taking psychotropic medications (for at least four weeks) at the time of study entry and fMRI scanning. The youth in the current sample were 54.8% female, 73.8% Caucasian, 11.9% African American, 09.5% Asian, and 04.8% multiracial.

2.2 Procedure

Procedures were approved by the Institutional Review Boards at both UM and UIC. Parents and their offspring completed symptom measures and the K-SADS-PL. Youth also completed the fMRI scan.

2.3 Measures

Parental Depressive Symptoms—Parent's symptoms of depression were assessed using the Beck Depression Inventory (BDI)-II (Beck et al. 1996). The BDI-II is a 21-item questionnaire that assesses the severity of depressive symptoms in the past two weeks. For the majority of the sample (95%) depressive symptom measures were obtained from mothers. Mother-ratings were unavailable for a small proportion of the sample ($n = 02$) and depressive symptoms on fathers were collected. In the current sample, the range of parent BDI scores was 0–32 ($M = 08.02$, $SD = 08.86$). Twenty-nine percent of parents in the sample reported depressive symptoms above the clinical threshold as defined by a BDI score greater than 12 (Dozois et al. 1998).

Youth's Anxious and Depressive Symptoms—Youth's symptoms of anxiety and depression were assessed using the Multidimensional Anxiety Scale for Children (MASC)

(March et al. 1997) and the Children's Depression Inventory (CDI) (Kovacs, 1981), respectively.

Emotional faces shifting attention task (EFSAT)—(Klumpp et al. 2013, 2015; Swartz et al. 2014). A trial of the EFSAT consists of three faces in a triangular configuration, and three shapes in an upside-down triangular configuration (Figure 1). During the faces condition, participants were instructed to identify which face on the bottom row matched the emotion of the target face on the top. During the shapes condition, participants were instructed to match shapes on the top row with the target shape on the bottom. Participants completed two runs, for a total of 18 faces blocks and 18 shapes blocks with six blocks of each condition: match angry faces, fear faces, and happy faces, and match shapes with angry faces, fear faces, and happy faces as distractors. Each block began with a 4s cue to either 'match faces' or 'match shapes' followed by the four sequential matching trials, each lasting four seconds.

fMRI data acquisition and processing—MRI data were collected on 3 Tesla GE scanners with 8-channel head coils at both sites. At UM, functional data were collected with a gradient-echo reverse spiral acquisition with the following parameters: repetition time (TR) = two seconds, echo time (TE) = 30 ms, flip angle = 90°, = field of view (FOV) = 22 × 22 cm, acquisition matrix 64 × 64, 03-mm slice thickness, and 43 axial slices. At UIC, functional data were acquired using gradient-echo echo-planar imaging (EPI) sequence with the following parameters: TR = two seconds, TE = minFull [~25 ms], flip angle = 90°, FOV = 22 × 22 cm, acquisition matrix 64 × 64, 03-mm slice thickness, and 44 axial slices.

Functional images were preprocessed in SPM8 (Wellcome Trust Centre for Neuroimaging, <http://www.fil.ion.ucl.ac.uk/spm/>) for slice timing correction, image normalization, resampling at a 02 × 02 × 02 mm³ voxel size, and 08-mm Gaussian smoothing kernel. First-level within-subject analysis was performed with a general linear model (GLM) with 6 regressors of interest: Match Shape_[Angry], Match Shape_[Fear], Match Shape_[Happy], Match Face_[Angry], Match Face_[Fear], Match Face_[Happy]. Additional nuisance regressors for six motion parameters were included to correct for motion artifacts. For each participant, contrast images of brain activity were generated for second-level analysis. We collapsed across angry and fear facial expressions to evaluate activation to implicit and explicit threat processing and activation to non-threatening (i.e., happy) implicit and explicit processing.

2.4 Data Analysis

Consistent with previous studies in our lab using the EFSAT (Klumpp et al. 2013, 2015; Swartz et al. 2014), one-sample *t*-tests with parent depressive symptoms (i.e., BDI) as a covariate of interest were performed in SPM8 separately for Match Shape_[Threat] > Match Face_[Threat] and Match Shape_[Happy] > Match Face_[Happy]. Site/scanner (UM or UIC) and youth's current anxiety disorder status (absent or present) were included as covariates in second-level analyses. Based on *a priori* regions of interest from our hypotheses, we focused on the bilateral amygdala and ACC (encompassing the dorsal ACC intersected with the medial frontal region to include the rostral portion of the ACC) as defined by atlas-based anatomical ROIs with Automated Anatomical Labeling (AAL) toolbox within SPM.

Activations were deemed as significant using small volume correction (SVC) at $p < 0.001$ and family wise error (FWE) correction at $p < 0.025$. For completeness, we also examine activation clusters outside *a priori* ROIs at the whole-brain level using Family Wise Error (FWE) correction at $p < 0.025$. To interpret significant effects and for additional post-hoc analyses, activation (beta weights) from a 10-mm diameter sphere around the peak voxel was extracted from individual activity maps using MarsBar (Brett et al. 2002) and exported into SPSS. In SPSS, exploratory multiple regression analyses were computed to examine demographic and clinical variables as moderators of associations between *a priori* brain regions and parental depressive symptoms.

3. Results

3.1 Participant Characteristics

As shown in Table 1, participant demographic and clinical variables did not significantly differ across sites ($ps > 0.05$). We also examined whether parent depressive symptoms were correlated with child age, sex, or anxiety group status. Parent depressive symptoms were unrelated to youth's age or sex ($ps > 0.39$). However, youth with an anxiety disorder had parents with greater depressive symptoms ($M = 12.20$, $SD = 09.68$), relative to youth without an anxiety disorder ($M = 03.67$, $SD = 04.72$; $t(01,40) = 03.63$, $p < 0.001$).

3.2 Behavioral Performance

To examine the influence of emotion (angry, fear, happy) and condition (shapes versus faces) on accuracy and RT, a mixed-design ANCOVA was conducted with parent BDI and children's anxiety disorder status serving as the covariates. Results revealed a main effect of condition, $F(01,41) = 12.67$, $p < 0.001$, with accuracy being higher for matching shapes ($M = 83.42$, $SE = 02.21$) compared to matching faces ($M = 78.21$, $SE = 02.29$) across participants. There was also a main effect of condition for RT, $F(01, 41) = 21.76$, $p < 0.001$, with slower responses when matching faces ($M = 1687.02$, $SE = 97.21$) compared to matching shapes ($M = 1301.02$, $SE = 93.12$). Results also revealed a main effect of anxiety group for RT, $F(01,41) = 03.67$, $p = 0.04$, due to the anxiety disorder group being slower to respond overall. None of the other main effects or interactions with parent depressive symptoms were significant for accuracy or RT (lowest $p = 0.17$).

3.3 fMRI Results

Results of the ROI one-sample t -test revealed that greater parental depression was associated with increased activation of the cingulate cortex (encompassing the dorsal ACC) [(02 28 26), t -value = 04.45, volume = 5000 mm³, F_{WE} corrected $p < 0.01$; brodmann area 32] during the Match Shape [Threat] > Match Face[Threat] contrast (Figure 2). No other regions outside the ACC were positively or negatively associated with parental depressive symptoms for the Match Shape [Threat] > Match Face[Threat] contrasts within ROIs or for the whole-brain analysis when utilizing FWE correction. There were also no significant regions within the ROI or for the whole-brain analysis that were positively or negatively associated with change in parent's depressive symptoms for the Match Shape[Happy] > Match Face[Happy] contrast.¹

Next, because the Match Shape [Threat] > Match Face[Threat] contrast can be interpreted as increased activation when matching shapes (implicit threat emotion processing) or decreased activation when matching faces (explicit threat emotion processing), we extracted beta weights from a spherical ROI around the peak voxel for the dorsal ACC (*a priori* region) for Match Shape[Threat] > Implicit Baseline and Match Face[Threat] > Implicit Baseline contrasts. Partial correlations in SPSS between brain activation and parent BDI symptoms were then calculated controlling for study site and youth's anxiety status. Dorsal ACC activation during implicit threat emotion processing was positively correlated with BDI symptoms ($r = 0.42$, $p < 0.01$; Figure 3b); however, the Match Face[Threat] > Implicit Baseline contrast was not related to parent depressive symptoms ($r = 0.06$, $p = 0.73$; Figure 3c).

Next, additional steps were taken to determine the robustness of the relation between parent symptoms of depression and youth's dorsal ACC activation during the Match Shape [Threat] > Match Face[Threat] contrast. We first examined if the effect of youth's dorsal ACC activation during implicit threat processing was unique to parent depressive symptoms or if it was also significantly related to youth's own symptoms of anxiety and depression. None of the correlations with youth's symptoms reached significance (lowest $p = 0.12$). Moreover, the relation between dorsal ACC activation in children and adolescents during implicit threat processing and parent BDI symptoms remained significant even after adjusting for youth's current anxiety and depressive symptoms ($ps < 0.04$). This effect also remained significant after adjusting for children's behavioral performance while matching shapes in the context of threatening face distractors ($ps < 0.03$), and the relation between youth's behavioral performance and dorsal ACC activation during implicit threat processing was unrelated ($ps > 0.10$). Finally, the relation between youths' dorsal ACC activation during implicit threat processing and parent depressive symptoms also remained significant when excluding fathers and focusing solely on mother's depressive symptoms ($p < 0.05$).

Finally, exploratory analyses in SPSS were computed to examine moderators (youth age, youth anxiety group). Hierarchical multiple regression analyses were computed predicting dorsal ACC activation during the Match Face[Threat] > Match Shape[Threat] contrast with youth age and parent BDI in Step 1, followed by the parent BDI by youth age interaction in Step 2. The BDI $\times$ youth age interaction was non-significant, $t(03,41) = 0.14$, $p = 0.99$. An identical regression was conducted using child anxiety status as the moderator instead of age. In this analysis, the BDI $\times$ child anxiety group interaction was also non-significant, $t(03,41) = 0.69$, $p = 0.50$.

4. Discussion

In this current preliminary study, we examined the neural correlates of directing attention *toward* (explicit emotion processing) and *away* (implicit emotion processing) from emotional stimuli in youth in relation to parent depressive symptoms. Consistent with our hypothesis, we found that parent depressive symptoms were positively correlated with youths' recruitment of the dorsal anterior cingulate cortex (ACC) during attempts to direct

¹We also examined whether age moderated the relation between parent depressive symptoms and brain activation for the Match Shape [Threat] > Match Face[Threat] and Match Shape [Happy] > Match Face[Happy] contrasts within ROIs or whole-brain analysis utilizing FWE correction. However, results revealed no significant regions.

attention away from negative (i.e., threatening) stimuli. This finding was not moderated by child's age or anxiety group status suggesting similar effects across the youth in the sample. Importantly, this finding was maintained after adjusting for youth's current depressive and anxious symptoms suggesting that this result was not due simply to the offspring's internalizing symptoms and may represent a potential vulnerability factor that places youth of parents with higher depressive symptoms at high risk for developing depression in the future. Notably, the dorsal ACC has been linked to cognitive control particularly in the context of conflict monitoring and during attempts to down-regulate negative emotional distractors (Etkin et al. 2011), and depression in adults has been characterized by overactivation of the rostral ACC when attempting to disengage from negative stimuli (Disner et al. 2011). The current study provides preliminary evidence that overactivation of the dorsal region of the ACC during attentional control may be one possible mechanism involved in the intergenerational transmission of depression.

Contrary to our hypothesis, we did not find evidence of enhanced amygdala activation during explicit processing of emotional stimuli in youth being related to parent depressive symptomatology. Notably, previous studies examining the relation between amygdala activation during explicit emotion processing and youth depression risk focused on parent's history of major depressive disorder (i.e., Mannie et al. 2011; Monk et al. 2008; Swartz et al. 2015), whereas the current study focused solely on current depressive symptoms. Therefore, these differences in measurement across studies may be one explanation for the different pattern of findings. On the other hand, the task used to probe explicit emotion processing in the current study was also different to that of previous studies. Specifically, the EFSAT task does not have a baseline (e.g., fixation) condition; therefore, explicit emotion processing was contrasted with implicit processing rather than a baseline condition, and may have been confounded by cognitive control.

As previously discussed, the extent to how negative information processing biases develop in offspring of parents with depression is not completely understood. The current study was preliminary in nature and focused on the relation between neural correlates of attentional control in offspring and parent current depressive symptoms, rather than history of depressive diagnoses. Therefore, we are unable to draw strong conclusions regarding the nature of how aberrant dorsal ACC activation during implicit threat processing develops among children of parents with high depressive symptoms. However, consistent with other risk factors involved in the intergenerational transmission of depression, deficits in attentional control among offspring likely involve both genetic and environmental (e.g., parenting) influences. For instance, these negative processing biases among offspring may be "experience-specific" (i.e., Cicchetti et al. 2000; Gibb et al. 2009; Pollak, 2003; Rose and Abramson, 2002) and develop as a result of consistent overexposure to the parent's negative affect or behaviors (i.e., exposure to higher levels of parental criticism, hostility). On the other hand, these biases may result from genetic or biological factors and may be passed down from the depressed parent to the offspring. Future larger, longitudinal studies are needed to examine how certain characteristics of parent's depression (i.e., single versus recurrent episodes, duration of episodes) shape these neural systems during attentional control.

There were limitations to the current study that provide important directions for future research. First, as previously mentioned, the current study did not collect data on history of depressive diagnoses among parents. It is possible that a portion of the parents in the current study with low current depressive symptoms may have experienced a previous episode of depression. This may have weakened our power to detect effects in other neural regions. Thus, future studies are needed to determine if the current preliminary findings replicate among youth of parents with a history of depression. Similarly, the current study did not have complete data on parental anxiety. Knowing that anxiety and depression are highly comorbid disorders (e.g., Brown et al., 2001), future studies are needed to determine whether the relationship between parental depressive symptoms and offspring dorsal ACC response during implicit threat processing is unique to parent depression, or is also related to elevated anxiety in the parents. Third, future, prospective designs are needed to determine if these neural abnormalities during implicit emotion processing in youth predict future onset of depression, thereby representing a putative vulnerability factor. Similar to this, many of the youth in the current sample had a current diagnosis of an anxiety disorder. Although the findings were maintained when adjusting for youth's current symptoms and diagnoses of anxiety and the cohort serves as a clinically representative group with a broad range of vulnerability, future studies are needed to determine if these findings replicate with a sample of children and adolescents free of any psychiatric history. Finally, we used a stringent whole-brain correction for multiple comparisons in a relatively small sample and thus the study is vulnerable to false negatives.

In summary, this is the first study to examine the neural correlates of implicit emotion processing in children and adolescents in relation to parent depressive symptoms. Consistent with adult depression studies (Disner et al. 2011; Elliott et al. 2002; Eugène et al. 2010; Mitterschiffthaler et al. 2008), the current findings indicate that greater symptoms of depression in parents correlate with increased recruitment of the dorsal ACC during attempts to direct attention away from negative stimuli in offspring. Altered dorsal ACC function may contribute to the biased attention for negative information typically demonstrated in youth at high risk for depression by disrupting efficient inhibition of negative stimuli. Future, prospective designs are needed to examine whether altered dorsal ACC activation during attentional control predicts future depression onset among youth at high risk for depression. The current preliminary findings assist in providing insight into the neural mechanisms involved in the intergenerational transmission of depression.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Highlights

- Parental depression is associated with numerous adverse outcomes in offspring; the mechanisms associated with this risk are not well known.
- We examined the neural correlates of explicit and implicit emotion processing in youth in relation to parent depression.
- Greater symptoms of depression in parents correlated with increased recruitment of the dorsal ACC during implicit processing of negative stimuli in offspring.
- Altered dorsal ACC function may contribute to the biased attention for negative information typically demonstrated in youth at high risk for depression by disrupting efficient inhibition of negative stimuli.

Figure 1. Schematic of exemplar ‘Match Faces’ and ‘Match Shapes’ blocks in the Emotional Faces Shifting Attention Task (EFSAT). Trials were presented in block format and participants were instructed at the beginning of each block to either match faces or match shapes for that block. The match faces condition requires attending to the emotional faces whereas the match shapes condition requires performing the shape-matching task in the context of emotional face distractors.

Figure 2.

Increased dorsal anterior cingulate cortex activation during Match Shape_[Threat] > Match Face_[Threat] contrast in youth is associated with elevated parental depressive symptoms (family-wise error corrected for multiple comparisons across small volumes of interest).

Figure 3.

Scatter plot depicting that elevated parental depressive symptoms is associated with increased dorsal anterior cingulate cortex activation (mean blood-oxygen-level-dependent parameter estimates) during a) Match Shape_[Threat] > Match Face_[Threat], b) Match Shape_[Threat] > Baseline (Implicit Threat Processing), c) Match Faces_[Threat] > Baseline (Explicit Threat Processing).

Participant characteristics by study site (University of Michigan [UM] or University of Illinois at Chicago [UIC]).

Table 1

	UM (n = 20)		UIC (n = 22)		t-value
	Mean	SD	Mean	SD	
Youth Age	14.00	03.29	13.64	02.38	0.68
CDI	07.53	07.46	10.14	10.18	0.36
MASC	46.40	20.06	45.42	21.42	0.88
Parent BDI	06.25	06.52	09.77	10.04	0.15
	N	%	N	%	χ ²
Youth Gender (Female)	13	65.0	10	45.5	1.62
Youth Race (Caucasian)	17	85.0	14	63.6	2.47
Youth Anxiety Diagnosis (Present)	11	55.0	10	45.5	0.38

Note: CDI = Children's Depression Inventory; MASC = Multidimensional Anxiety Scale for Children; BDI = Beck Depression Inventory.