Journal of Affective Disorders 229 (2018) 22–31

Contents lists available at ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Research paper

The neurobiology of self face recognition among depressed adolescents

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ARTICLE INFO

Keywords:
Adolescence
Depression
Self-face processing
Amygdala
Hippocampus
Fusiform
Neuroimaging

ABSTRACT

Objective: Depression is linked to alterations in both emotion and self-processing. The current study used functional magnetic resonance imaging (fMRI) to assess neural activation in healthy and depressed youth to a novel task that combined emotion processing with self-face recognition.

Methods: An fMRI study involving 81 adolescents (50.6% females; $M_{age} = 14.61$, SD = 1.65) comprised of depressed (DEP, $n = 43$), and healthy controls (HC, $n = 38$). Participants completed a clinical interview and self-report measures during an initial assessment. In the scanner, adolescents completed a face recognition task, viewing emotional (happy, sad, neutral) images of their own face (self) or the face of another youth (other).

Results: DEP youth showed higher activity in the cuneus ($F = 26.29$) and post and precentral gyri ($F = 20.76$), across all conditions compared to HC. Sad faces elicited higher posterior cingulate cortex, precuneus ($F = 10.36$) and inferior parietal cortex activity ($F = 11.0$), and self faces elicited higher precuneus, fusiform ($F = 16.39$), insula and putamen ($F = 16.82$) activity in all youth. DEP showed higher middle temporal activity to neutral faces but lower activity to sad faces compared to HC, who showed the opposite pattern ($F = 12.86$). DEP also showed hypoactive mid-temporal limbic activity relative to controls when identifying their self happy face vs. neutral face, yet showed hyperactivity when identifying the other happy face vs. neutral face, and HC showed the opposite pattern ($F = 10.94$).

Conclusions: The neurophysiology of self-face recognition is altered in adolescent depression. Specifically, depression was associated with decreased activity in neural areas that support emotional and associative processing for positive self-faces and increased processing for neutral self-faces. These results suggest that depression in adolescents is associated with hypoactive emotional processing and encoding of positive self-related visual information. This abnormal neural activity at the intersection of reward and self-processing among depressed youth might have long lasting impact in self-formation and future adult self-representations, given that adolescence is a sensitive period for self-development.

1. Introduction

Adolescence is a key developmental period for the emergence of depression, as well as for transformations in self-processing and identity (Auerbach et al., 2015). In particular, increased self-processing (e.g. heightened self-consciousness or self-awareness) has been associated with depression and this association is strongest during mid-adolescence (Chen et al., 1998), suggesting that self-processing changes increase risks for depression during the adolescent transition. Depression is characterized by self-processing disturbances, which are linked to hyperactivation in cortical midline structures (CMS) (Lemogne et al., 2012). Because changes in self-processing are linked to upsurges of depression during adolescence (Chen et al., 1998), it is key to study the neural bases of self-processing in depressed adolescents. Uncovering how self-processing differs between depressed and healthy youth at the neural level may shed light on neuropsychological processes linked to onset and maintenance of depression.

Self-processing is the ability to perceive, and judge one's own states, traits, and abilities. A facet of this overarching construct, and the focus of this research, is visual self-face recognition (Hu et al., 2016). Self-

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https://doi.org/10.1016/j.jad.2017.12.023
Received 25 August 2017; Received in revised form 20 November 2017; Accepted 16 December 2017
Available online 28 December 2017
0165-0327/

processing is altered in depression. Negative self-directed thoughts and emotions are core depression symptoms (Gaddy and Ingram, 2014). Yet most existing work on self-processing neurophysiology in depressed populations relied on tasks that require participants to verbally reflect on stimuli in relation to themselves (See for a review of such tasks Lieberman, 2007). These facets of self-processing are subserved by midline cortical structures (MCS) such as anterior and posterior cingulate cortices (ACC, PCC), precuneus, and medial prefrontal cortex (MPFC) (Bradley et al., 2016; Heatherton et al., 2006). While these tasks are informative for a verbally explicit and predominantly cognitive definition of self-processing (Harter, 1999), they seldom engage limbic regions that enable rapid, emotionally charged self-processing. In contrast, self-face recognition does not rely on language and is enabled by limbic regions such as fusiform gyrus, amygdala, and hippocampus. For example, Kircher et al. (2001) examined self-face recognition and found it was subserved by limbic structures in addition to MCS. We propose that studying self vs. other face recognition may shed light on the neurophysiology of rapid emotional self-processes that are not always part of our explicit, verbal awareness. Specifically, we hypothesize that self-face recognition will allow access to the function of limbic, emotion processing neural structures among depressed adolescents.

1.1. Face processing neurophysiology and emotional biases in depression

There is ample research demonstrating that depression is characterized by exaggerated attention, encoding and recall of negative versus positive stimuli across many modalities (e.g. words, images, phrases (Platt et al., 2017)). Notably these cognitive and emotional biases in depression also extend to processing of socially salient stimuli such as faces (Stuhrmann et al., 2011). For example, while controls show greater limbic activation in response to subliminal happy versus sad facial expressions, depressed individuals show the opposite pattern (Stuhrmann et al., 2013).

In healthy populations, processing emotional faces is enabled by activity in the fusiform gyrus and in limbic areas such as the amygdala, insula and hippocampus (Fusar-Poli et al., 2009). Functional abnormalities in these areas during face processing have been noted among depressed compared to healthy individuals. In addition, depressed patients evidence limbic activations whereas controls show fronto-thalamic activations during processing of emotional faces (Lai, 2014; Stuhrmann et al., 2011). These reviews also show that depressed patients evidence abnormalities in the face processing network, specifically, hyperactivation to negative faces and hypoactivation to positive faces in the amygdala, insula, parahippocampal gyrus, fusiform face area, and putamen (Lai, 2014; Stuhrmann et al., 2011). Similar patterns of abnormal neural activity during face processing have been observed in depressed adults, adolescents and children. For example, depressed adults show biases towards negative emotional faces (Leppanen, 2006), as well as neural hypoactivation regarding positive faces in limbic brain regions (Barlow et al., 2012; Nejad et al., 2013). Depressed adolescents also exhibit amygdala hyperactivation versus healthy controls (Beesdo et al., 2009), and exposure to sad faces interferes with dorsolateral prefrontal cortex function during an inhibition task in depressed youth (Colich et al., 2016). This is again consistent with behavioral and imaging evidence that risk for major depression involves a bias to attend to negative and to neglect positive information.

In summary, past research suggests that there are depression specific biases toward negative faces (limbic hyperactivation) as well as biases away from positive faces (limbic hypoactivation) across development. Similar neurobehavioral biases have been noted in young healthy samples at risk for depression. Adolescents at risk for depression perceive mild happy expressions as less intense than do healthy youth (Kerestes et al., 2016) and amygdala hyperactivity to negative emotional faces has been observed in non-depressed adolescents at risk for depression (Monk et al., 2008). Additionally, children at-risk for depression show increased amygdala and cortical activation to fearful versus neutral, and decreased activation to happy versus neutral faces in the ACC and supramarginal gyrus (Chai et al., 2015). Notably, this reduced limbic activation to happy expressions was linked to anhedonia in depressed patients (Stuhrmann et al., 2013). Finally, slower identification of happy facial expressions and faster identification of sad faces predicted onset of depression in adolescents over an 8 year period (Vrijen et al., 2016), suggesting that these biases have predictive value and constitute risk factors for depression.

1.2. The neural activity of self-face processing and recognition

Self-face recognition is a special case of face and self-processing. Viewing and recognizing our own face is supported by neural areas that enable self-processing (i.e. MCS) and by structures that support face (fusiform) and emotion processing (i.e. amygdala, hippocampus) (Phan et al., 2004; Sergerie et al., 2008; Sugiura et al., 2005). Importantly, both limbic and MCS networks have been associated with abnormal neurophysiology in depression (Nejad et al., 2013). Limbic structures are known to underlie rapid processing and encoding of, and memory for emotionally charged information (Devue and Bredart, 2011; Habel et al., 2007; Sugiura et al., 2008). Therefore, recognition of emotionally charged self-faces elicits activation in limbic structures (amygdala and hippocampus for salient emotional facial information), in MCS (i.e. ACC, PCC, MPFC and precuneus for higher order processing of self- and emotion-related information) as well as in the fusiform (Sugiura, 2015). Given previously discussed self-processing abnormalities (in verbal and explicit modalities) as well as emotional biases among depressed patients, it would be reasonable to expect abnormal limbic activation to emotionally charged self faces relative to unfamiliar faces.

1.3. Current study

The aims of this study were to test whether patterns of brain activity during emotional self-face processing differed for depressed youth compared to healthy controls. Our primary hypotheses were that, during self-face processing, depressed youth would show less MCS and limbic activation to self happy expressions compared to healthy controls, and more MCS and limbic activation to self sad and neutral faces in this age group.

An additional complexity of studying the neurobiology of depression pertains to the effects of medication on brain function. Past authors have suggested that antidepressants modulate emotional biases by increasing positive emotional processing (Harmer et al., 2009). Here we also examine the putative effects of medication (primarily antidepressants) on brain activity among depressed adolescents engaged on a self-processing task.

2. Methods

2.1. Participants and procedure

Participants (N = 81) were recruited from the brief crisis inpatient unit and among youth assessed for depression at two Universities in the U.S., from local outpatient mental health clinics, and through radio and flyer advertisements. Of the participants included in analyses, 16 of the healthy and 12 depressed youth were scanned at Site1 (n = 28), while 22 controls and 31 depressed were scanned at Site 2 (n = 53). Similar numbers of depressed and healthy control youth were scanned at the two sites: χ^2 (1) = 1.80, p = 0.18. Participants with any of the following characteristics were excluded: IQ < 70, autism spectrum disorder, substance abuse or dependency, history of seizures, left handed, primary diagnosis other than depressive disorder. Diagnosis were assigned by two experimenters for all participants recruited as patients, kappa depressive disorders = 0.86, kappa anxiety disorders = 0.43. Diagnostic discrepancies were solved by the first author, a licensed clinical