Research paper

Altered time course of amygdala activation during speech anticipation in social anxiety disorder

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ABSTRACT

Background: Exaggerated anticipatory anxiety is common in social anxiety disorder (SAD). Neuroimaging studies have revealed altered neural activity in response to social stimuli in SAD, but fewer studies have examined neural activity during anticipation of feared social stimuli in SAD. The current study examined the time course and magnitude of activity in threat processing brain regions during speech anticipation in socially anxious individuals and healthy controls (HC).

Method: Participants (SAD n=58; HC n=16) underwent functional magnetic resonance imaging (fMRI) during which they completed a 90 s control anticipation task and 90 s speech anticipation task. Repeated measures multi-level modeling analyses were used to examine group differences in time course activity during speech vs. control anticipation for regions of interest, including bilateral amygdala, insula, ventral striatum, and dorsal anterior cingulate cortex.

Results: The time course of amygdala activity was more prolonged and less variable throughout speech anticipation in SAD participants compared to HCs, whereas the overall magnitude of amygdala response did not differ between groups. Magnitude and time course of activity was largely similar between groups across other regions of interest.

Limitations: Analyses were restricted to regions of interest and task order was the same across participants due to the nature of deception instructions.

Conclusions: Sustained amygdala time course during anticipation may uniquely reflect heightened detection of threat or deficits in emotion regulation in socially anxious individuals. Findings highlight the importance of examining temporal dynamics of amygdala responding.

1. Introduction

Excessive anxiety in both the presence and anticipation of social situations is a central feature of social anxiety disorder (SAD). Exaggerated anticipatory anxiety can lead socially anxious individuals to avoid social situations or engage in safety behaviors, thus maintaining SAD symptoms by preventing new learning and reinforcing the maladaptive belief that social apprehension is warranted (Hofmann, 2007; Wells et al., 1995). Cognitive models of SAD posit that socially anxious individuals engage in negatively biased anticipatory processing prior to entering social situations (e.g., expecting a negative outcome from an interaction), which enhances anxiety and increases avoidance behaviors (Clark and Wells, 1995; Hinrichsen and Clark, 2003). Given the role of anticipatory anxiety as a maintenance factor for SAD, it is important to better understand the neural bases of anticipatory processing in social anxiety.

Studies of the functional neuroanatomy of anxiety and emotional reactivity in SAD have revealed altered neural activity in response to social stimuli, including heightened amygdala responses to harsh (e.g., angry, disgusted) faces compared to happy faces (Phan et al., 2006; Stein et al., 2002), exaggerated amygdala reactivity to harsh faces compared to healthy controls (e.g., Klumpp et al., 2010), and greater amygdala and insula activity in response to faces with angry expressions compared to neutral ones (Straube et al., 2004). Indeed, amygdala and insula regions frequently show hyperactivation across provocation and affective processing study designs in individuals with anxiety disorders, including SAD (Etkin and Wager, 2007; Miskovic and Schmidt, 2012). In addition to amygdala and insula, anterior
The time course of amygdala activity may be of particular importance for anxious populations. A previous study found that SAD individuals exhibit altered amygdala temporal response patterns to negative and positive emotional faces, such that amygdala responses occurred later in SAD versus control participants (Campbell et al., 2007). In another fMRI study of 120 participants, heightened trait neuroticism was associated with more prolonged amygdala activation following the presentation of negative images, but was not associated with initial amygdala reactivity (Schuyler et al., 2012). In other words, slower amygdala “recovery” rather than elevated amygdala reactivity to negative images correlated with trait neuroticism. Based on these findings, we expected that SAD individuals would have not only more elevated but also more sustained or prolonged amygdala activation to threat during speech anticipation compared to healthy controls. Additionally, we expected that heightened and more sustained amygdala activity would be associated with more severe social anxiety symptoms. For other ROIs, we hypothesized that SAD individuals would show heightened insula activity and reduced ventral striatum activity compared to controls. We also examined whether dACC activation in SAD was reduced (replicating Lorberbaum et al. [2004] results) or elevated (e.g., Phan et al., 2006) compared to controls. Beyond main effects of group, hypotheses regarding the time course of activity in non-amygdala regions were largely exploratory.

2. Methods

2.1. Participants

Participants were recruited as part of a study comparing two behavioral treatments for SAD (see Craske et al., 2014). SAD participants met DSM-IV criteria for principal or co-principal SAD with a clinical severity rating (CSR) of 4 or higher according to the Anxiety Disorders Interview Schedule (Brown et al., 1994). HC participants could not meet DSM-IV criteria for any Axis I disorder. All participants were between 18 and 45 years of age, either medication free or stabilized on medication, not undergoing behavioral therapy, English-speaking, and right-handed. Exclusion criteria included active suicidal ideation or severe depression (CSR > 6), psychiatric hospitalization within the past five years, serious medical conditions or pregnancy, history of psychosis or bipolar disorder, substance abuse or dependence within the past 6 months, claustrophobia, and non-removable metal in body.

Seventeen HC participants and 71 SAD participants entered the study and completed the fMRI scan. Of these, 1 HC and 11 SAD participants did not complete the speech anticipation task due to technical errors; thus 16 HC and 60 SAD participants were included in the present study. Participants were 50% female with a mean age of 27.8 years (SD=6.6) and were 49.3% Caucasian, 24.0% Asian/Pacific Islander, 14.7% Hispanic/Latino, 2.7% Black/African American, and 9.3% other race. HC and SAD participants did not differ by gender, age, or ethnicity (ps > .26). The majority (82.5%) of SAD participants were unmedicated.

2.2. Procedure

Participants completed the ADIS-IV and a battery of questionnaires, including the Liebowitz Social Anxiety Scale (Liebowitz, 1987). Eligible participants then completed a laboratory assessment, which included a computer dot probe task and public speaking task, followed by an fMRI scan approximately one week later. During the fMRI, participants completed several tasks to assess emotional reactivity and emotion regulation, including the control and speech anticipation tasks below.

2.3. Control anticipation task

Prior to entering the scanner, participants were told that they would...
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