



Abnormal prefrontal cortical response during affective processing in borderline personality disorder

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ABSTRACT

Emotion dysregulation is a hallmark feature of borderline personality disorder (BPD) and is associated with a dysfunction of prefrontal (PFC)–limbic systems. The purpose of the present study was to examine PFC function in BPD during the experience and suppression of sadness. Subjects were females with BPD ($N=9$) and age-, gender-, and IQ-matched non-psychiatric comparison subjects ($N=8$). Evoked hemodynamic oxygenated hemoglobin (oxy-Hb) was examined in PFC using functional near-infrared spectroscopy while subjects viewed neutral or sad images and were instructed to either maintain or suppress their emotional reactions. No group differences in behavioral ratings of sadness suppression or mean levels of evoked oxy-Hb were observed. BPD and control subjects, however, recruited homologous regions of lateral PFC during emotional suppression, with right lateral PFC activation for BPD subjects associated with difficulty suppressing sadness, whereas an inverse relationship was observed in left lateral PFC for healthy controls. Exploratory analyses revealed that the slope of the rise in oxy-Hb in medial PFC during transient sadness was positive and steep for healthy controls. Conversely, BPD subjects showed a negative and shallow slope, which was associated with severity of clinical symptoms. These results suggest that BPD subjects may show abnormal evoked oxy-Hb in medial PFC during transient sadness, with recruitment of right lateral PFC in BPD associated with reported difficulty in suppressing emotion. This abnormal cortical response, possibly in tandem with subcortical–limbic regions, may underlie symptoms of emotion dysregulation in BPD.

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1. Introduction

Borderline personality disorder (BPD) is a severe psychiatric condition characterized by chronic difficulties with emotion regulation. Functional neuroimaging techniques have been used to elucidate the neural circuitry underlying emotion dysregulation in BPD. A relatively consistent finding in studies of passive viewing of negatively valenced emotional stimuli is of elevated blood oxygen-level dependent (BOLD) functional magnetic resonance imaging (fMRI) signal in the amygdala, sometimes bilaterally, in BPD subjects compared with non-psychiatric controls (Herpertz et al., 2001; Donegan et al., 2003; Minzenberg et al., 2007; Schnell et al., 2007; Koenigsberg et al., 2009a; Guitart-Masip et al., 2009). These studies also typically identify activation differences in regions of the frontal cortex, most notably medial prefrontal cortex (PFC) and inferior frontal gyrus, as well as other regions of the brain typically involved in emotion (i.e., anterior cingulate cortex, insula) and face processing (i.e., fusiform gyrus, primary visual cortex). Similar findings have been

found in studies probing emotion systems using aurally presented scripts containing such themes as abandonment, self-injury, and unresolved life events (Schmahl et al., 2003; Beblo et al., 2006; Kraus et al., 2010). A disconnection between amygdala and PFC activity has been proposed as a possible mechanism underlying emotion dysregulation and impulsivity in BPD (New et al., 2007). Taken together, these findings are suggestive of a disruption of frontolimbic circuitry in BPD, the suggestion being that PFC systems (i.e., medial and ventrolateral PFC) are disturbed in their effortful regulation of emotion which originates from subcortical limbic regions in an automatic fashion.

Persons with BPD have frequent fluctuations in mood, even more so compared with patients who have depressive disorders (Trull et al., 2008), and these unstable moods typically involve alternations between sadness and anxiety (Reisch et al., 2008). Given the vulnerability of these patients to negative mood states, understanding which cortical systems may be involved in processing and regulating sadness may shed light on the neural substrate of emotion dysregulation in BPD. Discrete brain regions are involved in the processing of sad images in healthy individuals. Relative to neutral conditions, limbic and paralimbic areas activated during sadness include medial and ventrolateral PFC, orbitofrontal cortex, cingulate, and mesial temporal cortex (George et al., 1995; Pelletier et al., 2003).

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The effortful regulation of negative affect appears to activate a distributed network of structures which includes areas of the frontal cortex (orbitofrontal and dorsomedial PFC), anterior cingulate, and amygdala (Levesque et al., 2003; Phan et al., 2005; Banks et al., 2007; Goldin et al., 2008; Kross et al., 2008). There is limited information about the neural circuitry underlying effortful emotion regulation in persons with BPD. One study found that BPD subjects who were instructed to use a psychological distancing strategy to regulate responses to pictures depicting negative social interactions showed less activation in dorsal anterior cingulate cortex and intraparietal sulcus, less deactivation in the amygdala, and greater activation in the superior temporal sulcus and superior frontal gyrus (Koenigsberg et al., 2009b). Given these findings and in conjunction with neuropsychological evidence of frontal-system dysfunction in BPD (for a review, see Ruocco, 2005), it seems plausible to suggest that emotion dysregulation in BPD may result, at least in part, from a difficulty with top-down or effortful suppression of negative affect.

The purpose of the present study was to examine evoked hemodynamic oxygenation (oxy-Hb) in PFC using functional near-infrared spectroscopy (fNIRS) while BPD subjects viewed sad images and were instructed to either feel the emotion (i.e., experience transient sadness) or suppress the emotion (i.e., use a psychological distancing strategy to regulate their emotion). It was hypothesized that BPD subjects would report greater difficulties than controls in regulating their subjective emotional responses to the sad images during the Suppression condition. With regard to the neuroimaging data, in light of past fMRI studies of emotion processing in healthy individuals, an extended pattern of activation involving the medial and ventrolateral PFC was predicted during transient sadness for all subjects. No mean differences in evoked oxy-Hb, however, were anticipated between BPD and control subjects during transient sadness given the lack of any specific PFC findings (but robust amygdala activation) for BPD subjects in a similar fMRI study of emotion processing (Donegan et al., 2003). During the Suppression condition, activation of right dorsolateral PFC was expected for all subjects, as was found in the Levesque et al. (2003) study which employed similar procedures. BPD subjects, however, were predicted to show less activation in this region compared with controls. This hypothesis is consistent with previous findings of aberrant right superior frontal gyrus activation in BPD subjects relative to controls during emotional suppression (Koenigsberg et al., 2009b), and is also predicated on the notion that frontal-system dysfunction may underlie emotion dysregulation in BPD.

fNIRS affords a high degree of temporal resolution compared to other neuroimaging techniques (e.g., fMRI, positron emission tomography [PET]), which allows for a precise characterization of the temporal features of the hemodynamic response to stimuli of interest. Capitalizing upon this advantage, a series of exploratory analyses were planned to examine the slope of the evoked oxy-Hb response to the block of sad images for each experimental condition (i.e., Sad and Suppression). Whereas we are unaware of any studies examining this parameter of the hemodynamic response in subjects with BPD, we predicted that a shallower response slope would be seen for BPD subjects within medial PFC during transient sadness, consistent with previous fMRI findings of inefficient recruitment of medial PFC during emotion processing in BPD. Other areas of PFC, most notably dorsomedial and orbitofrontal, were hypothesized as possible areas in which shallower hemodynamic response slopes could be identified in BPD subjects, although these are speculations given limited information about the neural substrates of effortful emotion regulation in BPD.

Apart from its strong temporal resolution, fNIRS is also advantageous because it provides a quantification of the relative concentration of oxygenated hemoglobin in cortical tissue. The oxy-Hb parameter provides information about cerebral hemodynamics unique to that provided by the fMRI BOLD signal, which is more

strongly correlated with deoxygenated hemoglobin measurements (Steinbrink et al., 2006). Thus, the results of the present study have the potential to illuminate the role of evoked oxy-Hb in PFC during emotion processing, a parameter which has received comparatively less attention in functional neuroimaging studies of emotion.

2. Materials and methods

2.1. Subjects

The study received approval from the Drexel University Institutional Review Board. Nine females with BPD and eight healthy female controls were recruited from a university counseling center and from the community. All BPD subjects endorsed the criterion of affective instability; six endorsed difficulty controlling anger. The groups did not differ by age, years of education, ethnicity, or Full Scale IQ (see Table 1). All subjects completed a comprehensive screen for DSM-IV Axis I disorders. BPD subjects additionally met criteria for the following Axis I disorders: major depressive disorder, recurrent ($n=3$); dysthymic disorder ($n=1$); social phobia ($n=2$); and alcohol dependence, in partial remission ($n=1$). Three BPD subjects were currently receiving outpatient treatment: psychotherapy alone ($n=1$) and pharmacologic treatment alone ($n=2$). Subjects receiving pharmacologic treatment were taking stimulant ($n=1$) and combined stimulant and antidepressant ($n=1$) medications.

To be eligible to participate, all subjects met the following inclusion criteria: 18 years of age or older at the time of recruitment, female, English-speaking, right-handed, and able and willing to provide written informed consent. BPD subjects were required to meet DSM-IV criteria for current BPD. Subjects were ineligible for participation if they met DSM-IV criteria for schizophrenia or any psychotic disorder, bipolar disorder, current eating disorder or lifetime eating disorder that required hospitalization, mental retardation, neurological or severe somatic disorder, or significant head trauma (> 5 min loss of consciousness). Additionally, healthy subjects were excluded if they had a current diagnosis of any Axis I or Axis II disorder. In the week prior to testing, all subjects were asked to abstain from cannabis or any other illicit drug use, and severe alcohol consumption. In addition, all subjects were instructed to abstain from coffee and nicotine 24 h prior to testing. After complete description of the study to the subjects, written informed consent was obtained.

2.2. Clinical assessment

Subjects completed the BPD module of the Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV) (Zanarini et al., 1996). The DIPD-IV is a semi-structured interview to categorically assess DSM-IV personality disorder. Reliability statistics (Cronbach's alpha) for each criterion ranged from 0.57 (impulsivity) to 0.97 (affective instability) in the present study. Subjects also completed the Structured Clinical Interview for DSM-IV Axis I Disorders (First et

Table 1

Demographic characteristics of borderline personality disorder and healthy control subjects.

	BPD ($n=9$)	Healthy controls ($n=8$)
Age (years) ^a	20.78 (6.30)	18.88 (0.84)
Education (years) ^b	12.89 (1.54)	12.88 (0.84)
Ethnicity (n) ^c		
White (not of Hispanic Origin)	7	6
Black (not of Hispanic Origin)	0	1
Asian or Pacific Islander	2	1
Full Scale IQ M (S.D.) ^d	111.0 (9.79)	107.0 (14.03)

^a $t(15) = -0.85, P = 0.41$.

^b $t(15) = -0.02, P = 0.98$.

^c $\chi^2(2) = 1.36, P = 0.51$.

^d $t(15) = -0.659, P = 0.50$.

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