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Emotion avoidance and fear bradycardia in patients with borderline personality disorder and healthy controls

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ABSTRACT

Background and objectives: Exaggerated emotional reactivity is supposed to be essential in the etiology of borderline personality disorder (BPD). More specifically, models of defensive behavior would predict reduced freezing behavior —indicated by fear bradycardia-in response to threat. This study examined automatic fear bradycardia responses in BPD versus healthy controls and the role of emotion dysregulation, more specifically tendencies to avoid emotions.
Methods: Patients with BPD ($n = 23$) and healthy controls ($n = 18$) completed questionnaires and then watched neutral, pleasant and unpleasant pictures while heart rate was assessed.
Results: Emotion avoidance interacted with group: it was associated with distinct autonomic responses in healthy controls but not in BPD patients. Controls with lower emotion avoidance tendencies showed bradycardia in response to unpleasant pictures, while controls with higher emotion avoidance tendencies did not. BPD patients showed no bradycardia, irrespective of their emotion avoidance tendencies.
Limitations: This study is limited by a small sample size. Comorbidity or medication intake were not controlled for.
Conclusions: The results may suggest impaired automatic defense responses in BPD. Further understanding of the regulation of distress and defense responses might improve BPD treatment.

1. Introduction

Borderline personality disorder (BPD) is characterized by a pervasive pattern of instability in interpersonal relationships, self-image, and affects, and marked impulsivity (American Psychiatric Association, 2013). Inadequate regulation of emotions is proposed to be a key deficit in BPD (Linehan, 1993; Selby & Joiner, 2009). Dysregulated emotional responses and affective instability are thought to be affected by two factors: emotional reactivity (hyperreactivity to emotional stimuli) and emotion regulation strategies (attempts to affect the emotion) (Carpenter & Trull, 2013; Gross & Jazaieri, 2014). With regard to emotional reactivity, empirical findings are contradictory. Increased emotional reactivity is usually operationalized by enhanced subjective and/or physiological responses to stress-inducing stimuli compared to positive and neutral stimuli. With respect to subjective responses, some studies indeed found enhanced self-reported emotional reactivity in BPD patients (Glaser, Van Os, Mengelers, & Myin-Germeys, 2008) but others did not (e.g., Arntz, Klokman, & Sieswerda, 2005; Herpertz, Kunert, Schwenger, & Sass, 1999; Herpertz et al., 2001) or only in reaction to stimuli with a specific BPD-relevant content such as social interaction or abandonment (Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2010; Sauer, Arens, Stopsack, Spitzer, & Barnow, 2014). Findings on physiological reactivity are contradictory too. Some authors found increased autonomic reactivity in BPD patients on some, but not on other parameters (Baschnagel, Coffey, Hawk, Schumacher, & Holloman, 2013; Ebner-Priemer et al., 2005;

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Herpertz et al., 2001), or no increased reactivity at all (Kuo & Linehan, 2009; Schmahl et al., 2004; Vitale & Newman, 2012). Interestingly, none of the studies found the expected increase in heart rate in BPD patients compared to normal controls (e.g. Baschnagel et al., 2013; Ebner-Priemer et al., 2005; Herpertz et al., 2001; Kuo, Fitzpatrick, Metcalfe, & McMain, 2016).

Lack of a comprehensive theoretical framework of affective dysregulation has been proposed to complicate the interpretation of these contradictory findings, and to cause problems in designing the right experimental methods (Ebner-Priemer et al., 2015). In the present study, we examined whether the automatic defense cascade model (Lang, Bradley, & Cuthbert, 1997) would be an adequate framework for increased emotional reactivity in BPD. This model - describing freezing, fight and flight responses to threat - is based on animal threat responses and may therefore adequately represent the emotional reactivity factor of emotion regulation. Importantly, the defense model proposes that threat-related behavior is flexible and changes depending on threat imminence and magnitude (Blanchard & Blanchard, 1988; Bracha, 2004; Fanselow & Lester, 1988; Lang, Simons, & Balaban, 1997; Ratner, 1967; Rivers, 1920). Such distance-related threat responses have been found in humans too (e.g., Mobbs et al., 2007; Richter et al., 2012). Physiological correlates differ, depending on (perceived) threat proximity. Importantly, for optimal survival chances an organism should be able to shift between fight/flight (with predominant sympathetic activation) and freezing (with sympathetic and parasympathetic co-activation), following environmental changes (Eilam, 2005). Following this model, maladaptive regulation might present itself as inadequate regulation of freezing and fight/flight, rather than exaggerated fight/flight, which has been used as an indicator of reactivity-deficits in many studies. Thus, the defense model may help in interpreting findings in terms of whether the response is appropriate for the specific defense stage.

The relevance of fight/flight responses (e.g., avoidance) for the etiology of threat-related disorders has been recognized (Brewin & Holmes, 2003; Porges, 2001). From a cognitive perspective, emotional hyperreactivity is proposed to be the result of increased threat perception and/or intolerance for distress (Leyro, Zvolensky, & Bernstein, 2010). Increased threat perception (or stimulus evaluation) has in turn been associated with (automatic fight/flight) action (Cacioppo & Gardner, 1999; Chen & Bargh, 1999; Neumann, Förster, & Strack, 2003; Rinck & Becker, 2007). Some recent studies suggested that distorted freezing behavior might be a relevant factor in the maintenance of threat-related psychiatric disorders as well (Blanchard, Griebel, Pobbe, & Blanchard, 2011; Fragkaki, Stins, Roelofs, Jongedijk, & Hagenaars, 2016; Hagenaars, Oitzl, & Roelofs, 2014).

Freezing, in animal studies defined as having a motionless posture (Fanselow, 1994), is a reaction to threat that serves survival (Hagenaars, Oitzl et al., 2014; Lang et al., 1997). It occurs after threat detection, can be prolonged in the absence of escape options, and has been associated with moderate threat. It is considered to serve orienting, enhance attentional processing, reduce detection by predators and prepare for action (Hagenaars, Oitzl et al., 2014). In freezing, parasympathetic and sympathetic systems are both active, resulting in its key characteristic: fear bradycardia (Hagenaars, Oitzl et al., 2014; Kozlowska, Walker, McLean, & Carrive, 2015; Walker & Carrive, 2003). Moreover, freezing is associated with risk assessment and the selection of appropriate action (Blanchard et al., 2011). Being associated with risk assessment, freezing should be especially present when using an emotion regulation strategy targeting the problem (thus taxing the situation), whereas *exaggerated* use of avoidance or suppression strategies should be associated with automatic flight behavior, i.e., decreased freezing (see also page 9 for further explanation).

Note that freezing should not be confused with orienting, a brief and immediate attentional response to novel stimuli. Freezing should also be distinguished from tonic immobility ("playing dead"), a threat response that occurs in case of physical contact with a predator, which includes immobility but also additional symptoms such as analgesia and possibly dissociation (Abrams, Carleton, & Asmundson, 2012; Kozlowska et al., 2015). Finally, learned helplessness can also be expressed by immobility. However, freezing is an active response with increased muscle tonus as a feature (parasympathetic and sympathetic co-activation), whereas learned helplessness is shown by muscle weakness.

Freezing responses have recently been elicited successfully in humans using a passive viewing paradigm with bradycardia as an important indicator of freezing (Azevedo et al., 2005; Hagenaars, Stins, & Roelofs, 2012; Roelofs, Hagenaars, & Stins, 2010). In such a paradigm, participants typically watch pictures with different valences without action-instruction so that spontaneous responses to unpleasant pictures can be compared to responses to neutral and pleasant pictures. This setup also proved adequate to detect individual differences in freezing responses (Hagenaars et al., 2012; Roelofs et al., 2010). It is also sensitive to threat expectancy, induced by for example preceding mental imagery interventions (Hagenaars, Mesbah, & Cremers, 2015). Importantly, pictures in the passive viewing paradigm are usually presented in blocks (usually 1 min in total), as freezing is considered a sustained response (Hagenaars, Roelofs, & Stins, 2014). Picture presentation times are usually shorter in experiments that investigate emotional reactivity. Using the passive viewing paradigm, healthy participants typically show fear bradycardia in response to unpleasant relative to neutral and pleasant pictures. The next step would be to test freezing behavior in threat-related psychiatric disorders such as BPD and posttraumatic stress disorder (PTSD). Adenauer, Catani, Keil, Aichinger, and Neuner (2010) used a passive viewing paradigm with traumatized participants with and without PTSD and healthy controls. They found bradycardia in response to aversive pictures in healthy controls, but not in traumatized participants with PTSD. Traumatized participants without PTSD showed bradycardia in response to all picture categories, suggesting that immediate bradycardia may be associated with resilience. The authors suggested that the patients reacted with rapid fight/flight responses without prior exploration of the stimulus. Note that Adenauer et al., (2010) used brief picture presentations, probably eliciting orienting rather than freezing responses. However, given that risk-assessment is highly associated with freezing, the same conclusion might apply. Moreover, these findings would be in line with the findings of Hagenaars et al. (2012) who found enhanced freezing-like responses (reduced body sway and bradycardia) in traumatized but healthy participants. It also matches the results of another study, in which healthy participants showed greater heart rate decreases during unpleasant pictures after a mental imagery intervention with a negative outcome that was related to these pictures, relative to mental imagery interventions with a positive outcome (related and unrelated to the pictures; Hagenaars et al., 2015). Speculatively, bradycardia after negative related imagery might indicate further exploration of the stimulus or adequate vigilance whereas immediate action and associated sympathetic activity is shown in case of unexpected threat. One other study including psychiatric patients (panic disorder; Lopes et al., 2009) found reduced body sway (interpreted as freezing-like behavior) throughout the experiment for patients with panic disorder relative to healthy controls. However, this effect was independent of picture category (neutral, unpleasant, and panic disorder-related content), and heart rate was not assessed.

In conclusion, although emotional hyperreactivity is considered to be a key factor in the etiology of BPD, empirical findings are