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# Affective dysregulation and dissociative experience in female patients with borderline personality disorder: a startle response study

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**Abstract**

Affective dysregulation and dissociation are currently discussed as core features of borderline personality disorder (BPD). Affective dysregulation is hypothesized to be correlated with increased amygdala functioning and dissociation is linked to inhibited processing on the amygdala and dampened autonomic output, according to the corticolimbic disconnection model of dissociation from Sierra and Berrios [Biological Psychiatry 44 (1998) 898]. We assessed startle response, which is mainly mediated by the amygdala, to investigate the relationship between affective dysregulation and dissociation. We hypothesized that patients with BPD would reveal enhanced responses to startling tones, but that these would be lessened by the presence of state dissociative experiences. 21 unmedicated female patients with BPD and 21 healthy female controls listened to 15 startling tones (95-dB, 500-ms, 1000-Hz) while heart rate, skin conductance and orbicularis oculi electromyogram responses were measured. Covariance analysis showed that the BPD group had a significantly higher startle response in the electromyogram as compared to controls. Furthermore, present-state dissociative experiences significantly influenced the startle response. Patients with low dissociative experiences revealed enhanced startle responses whereas patients with high dissociative experiences showed reduced responses. Our data support affective dysregulation in BPD as well as the corticolimbic disconnection model of dissociation, at least for EMG. Furthermore, it highlights the importance of assessing present-state dissociation in basic research as well as psychotherapy.


*Keywords:* Borderline personality disorder; Startle response; Affective dysregulation; Dissociation; Electromyography; Psychophysiology

## 1. Introduction

This study focuses on the interrelation of two DSM-IV criteria in borderline personality disorder (BPD): affective dysregulation or instability and dissociative symptoms. Affective dysregulation is currently depicted in the scientific literature as the core feature of BPD (Linehan, 1993; Sanislow et al., 2002; Skodol et al., 2002a,b; Siever et al., 2002). Support for this view has been found by several studies (Levine et al., 1997; Dougherty et al., 1999; Stein, 1996). Although the neurobiological basis of affective dysregulation is unknown some research suggests that affective dysregulation might be caused by higher activity of the amygdala (Corrigan et al., 2000). For example Herpertz et al. (2001a) found significantly higher activity of the amygdala to unpleasant visual stimuli within BPD patients compared to healthy controls, as assessed by fMRI. Although the self-report of valence and arousal did not differ between groups, the authors suggested that enhanced amygdala activation in BPD might reflect intense and slowly subsiding emotions. In addition alterations of the amygdala in BPD were found, using MRI-based volumetric measurements (Driessen et al., 2000; Tebartz van Elst et al., 2001). However, there have also been laboratory studies using potentiated startle

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that provided no evidence for emotional hyperreactivity or hypersensitivity in individuals diagnosed with BPD (Herpertz et al., 1999, 2001b) neither on a psychological nor on a physiological level.

Despite the central role of emotional reactivity in BPD, the restriction or absence of emotional experiencing, in the form of dissociative behavior, is also common in BPD (Zanarini et al., 2000) and is one of the defining DSM-IV criteria. Sierra and Berrios (1998) published a neurobiological model for depersonalization disorder, proposing bilateral corticolimbic disconnection during dissociation. In their model the medial prefrontal cortex inhibits processing on the amygdala, causing a reduced emotional experience and a dampening of autonomic output. Recent studies confirm autonomic blunting in dissociation. Sierra et al. (2002) showed that subjects with depersonalization disorder exhibited reduced magnitude and increased latency of skin conductance response to unpleasant stimuli, but not to non-specific stimuli suggesting a selective inhibition of emotional processing. Lanius and colleagues (2002) using a traumatic script-driven symptom provocation paradigm posttraumatic stress disorder (PTSD) could also partially support the model of Sierra and Berrios. The dissociated PTSD subgroup (present state) revealed reduced heart rate, increased activation in the dorsolaterala and medial frontal cortex and did not exhibit increased amygdala activation. Studies on peritraumatic dissociation and physiological response revealed controversial results. Griffin et al. (1997) reported decreased heart rate and galvanic skin response in female rape victims with high dissociation compared to a group with low dissociation during baseline and while reporting about the trauma. In contrast, Kaufmann et al. (2002) could not show any physiological differences to trauma-relevant stimuli between PTSD patients with low and high peritraumatic dissociation. Ladwig et al. (2002) investigated startle response and peritraumatic dissociation in survivors of life-threatening cardiac events. Using several subgroup analyses, the biggest influence on startle response was the diagnosis of PTSD. However, we think that the disconnection model of dissociation from Sierra and Berrios (1998) refers rather to present state than to peritraumatic dissociation.

Consistent with the disconnection model, clinicians have observed that dissociation is characterized by decreased emotional experiencing (Maldonado and Spiegel, 1998). Similarly, behavioral therapists (Foa and Kozak, 1986; Wagner and Linehan, 1999) view dissociation as functioning to regulate emotional engagement in e.g. exposure therapy.

To explore these questions related to the neurobiological underpinnings of affective dysregulation and dissociation in BPD, we employed the acoustic startle response paradigm (ASR). The neural pathway of the EMG in the ASR involves three neuronal "relay stations": The cochlear root neuron, the caudal pontine reticular nucleus (PnC), and motoneurons in the facial motor nucleus (Davis et al., 1999). The PnC is mainly controlled by the medial part of the central nucleus of the amygdala (Rosen et al., 1991). Enhanced amygdala activation (e.g. via electrical stimulation) therefore leads to enhanced startle response in EMG (Davis et al., 1999; Rosen and Davis, 1988). Along these lines, studies have shown that trauma survivors with chronic PTSD demonstrate both exaggerated amygdala response (Rauch et al., 2000) and elevated responses to startling tones (Metzger et al., 1999; Shalev et al., 1997, 2000).

Because of the afore mentioned linkage between BPD, emotional dysregulation, enhanced amygdala activation (Herpertz et al., 2001a) and enhanced startle response in EMG (Davis et al., 1999; Rosen and Davis, 1988), the ASR is an appropriate paradigm for investigating affective dysregulation in this population. The first specific hypothesis is: (1) Patients with BPD will show enhanced responses to startling tones. According to the corticolimbic disconnection model of dissociation from Sierra and Berrios (1998) we examined whether dissociation is linked with reduced physiological responsiveness. The second specific hypothesis is: (2) Present-state dissociative experiences will reduce the startle response in BPD. To ensure that reduced startle response is related to present-state dissociative experiences and not to other psychological variables, we investigated anxiety, depression, trait dissociation and comorbid PTSD as a confounding variables.

## 2. Materials and methods

### 2.1. Subjects

Twenty one female patients with BPD (10 inpatients, 11 outpatients) and a comparison group of 21 female healthy controls (HC) participated in this study. The BPD sample was recruited from consecutively admitted patients to a DBT-treatment program (Bohus et al., 2000). Patients were randomly assigned to inpatient or outpatient treatment. All patients fulfilled DSM-IV criteria for BPD, assessed by the appropriate segment of the Structured Clinical Interview for DSM-IV Personality Disorders (SCID-II; First et al., 1996) and scored a minimum of 8 points on the Revised Diagnostic Interview for Borderlines (DIB-R; Zanarini et al., 1989). Axis I comorbidity was assessed by the Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I; First et al., 1997). Patients with a lifetime history of schizophrenia, bipolar I disorder or alcohol and drug addiction were excluded. Trained psychologists administered all diagnostic instruments. Eighteen patients from the BPD group had current comorbid Axis I disorders, including major depressive disorder ($n = 6$), anxiety dis-