

A preliminary study of cortisol and norepinephrine reactivity to psychosocial stress in borderline personality disorder with high and low dissociation

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Abstract

The goal of the current study was to investigate subjective and neurohormonal reactivity to acute psychosocial stress in borderline personality disorder (BPD) as a function of dissociative symptoms. Five BPD subjects with high dissociation, 8 BPD subjects with low dissociation, and 11 healthy control subjects were compared in basal urinary cortisol and norepinephrine, as well as in plasma cortisol and norepinephrine reactivity to the Trier Social Stress Test (TSST). Subjective stress rating and emotional response to the TSST were also measured. The three groups differed significantly in cortisol stress reactivity, with the high-dissociation BPD group demonstrating the most robust response. The three groups did not significantly differ in norepinephrine stress reactivity. In the combined BPD sample, dissociation severity tended to be inversely correlated with basal urinary norepinephrine, was positively correlated with norepinephrine stress reactivity. Childhood trauma was inversely correlated with basal urinary cortisol. In conclusion, despite its small sample size this pilot study suggests that dissociative symptomatology may be a marker of heightened biological vulnerability to stress in BPD, and merits further study.

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1. Introduction

Dissociative symptoms are a prominent feature in a subgroup of borderline personality disorder (BPD) individuals, and comprise one of the nine current diagnostic criteria for the disorder (“transient, stress-related paranoid ideation or severe dissociative symptoms”). It has been suggested that the addition of this criterion was the most significant revision of the BPD diagnosis instituted by the DSM-IV, and the criterion

carries excellent specificity for the disorder (Skodol et al., 2002). In one study of a large sample of 290 BPD patients, 32% had low, 42% moderate, and 26% high levels of dissociation (Zanarini et al., 2000a). Dissociation severity was predicted by inconsistent caretaking, sexual abuse by a caretaker, childhood witnessing of sexual violence and adult rape (Zanarini et al., 2000b). Simeon et al. (2003b) found that dissociation in a BPD sample was specifically related to emotional neglect rather than total childhood trauma, as well as to fearful attachment style. Goodman et al. (2001) on the other hand reported that pathological dissociation in BPD was unrelated to childhood trauma, but was possibly related to genetic factors.

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One compelling reason for greater attention to dissociative symptoms in BPD is the strong association between dissociation and high-risk BPD behaviors such as self-mutilation (Brodsky et al., 1995; Zlotnick et al., 1999); greater depressive, anxious, posttraumatic, behavioral dyscontrol and alcohol abuse symptomatology (Shearer, 1994; Brodsky et al., 1995; Wildgoose et al., 2000); and greater overall utilization of psychiatric services (Brodsky et al., 1995). Specifically, Zlotnick et al. (1999) showed that a higher level of dissociation was associated with self-injuring behavior, even when controlling for borderline personality disorder diagnosis and for childhood abuse. In a study by Brodsky et al. (1995), self-injury, childhood trauma, depression, and psychiatric treatment were all significant predictors of dissociation when all other variables were controlled for.

It remains unknown whether a dissociative symptom domain can be reliably defined in BPD, with associated neurobiological features, particularly perturbations in stress-related neurohormones such as the HPA axis and the noradrenergic system which are widely implicated in stress-related psychopathology. Indeed, a hallmark feature of BPD that has been relatively ignored in clinical research is BPD patients' pronounced stress intolerance. It has been argued that the proneness to regression may be a hallmark feature of the condition, and possibly should comprise a diagnostic criterion (Skodol et al., 2002); such regression typically occurs under conditions of diminished structure and constancy and increasing stress. In a naturalistic study that investigated the correlates of stress in BPD, participants were monitored over a 24-h period in order to examine self-reported states associated with the experience of "aversive tension," i.e. feeling overwhelmingly stressed unlinked to a specific clearly identifiable affect (Stiglmayr et al., 2001); substantial inter-individual differences were found in the duration and intensity of subjectively perceived tension, and this tension correlated strongly with dissociative symptoms. To our knowledge, there are no studies that have examined HPA axis or noradrenergic functioning in BPD in relation to dissociative symptoms. Other BPD symptom domains, e.g. impulsive aggression and affective instability, have been much better characterized to date, phenomenologically, neurobiologically, and in terms of treatment response (Best et al., 2002; Koenigsberg et al., 2002; Hollander et al., 2003).

The HPA axis has been the subject of some investigation in BPD. Several studies have examined dexamethasone suppression in BPD and have fairly consistently shown that the DST is neither particularly sensitive to the diagnosis nor helpful in differentiating

BPD from other affective spectrum disorders (Lahmeyer et al., 1988; Korzekwa et al., 1991; De la Fuente and Mendlewicz, 1996). In personality-disordered subjects, cortisol hypersuppression in response to dexamethasone has been found to be associated with comorbid PTSD rather than with trauma exposure or with the BPD diagnosis per se (Grossman et al., 2003). In a more naturalistic design, Lieb et al. (2004) conducted sequential ambulatory salivary cortisol monitoring over a 3-day period and reported significantly higher ambient cortisol levels in BPD compared to healthy controls. Another study examined cortisol reactivity to traumatic and abandonment scripts, and found that BPD patients had enhanced cortisol reactivity compared to PTSD and control subjects (Elzinga et al., 2002). Unlike BPD, the HPA axis has been minimally investigated in dissociative disorders; Simeon et al. (2001) reported elevated basal cortisol and dexamethasone suppression resistance in depersonalization disorder compared to healthy controls.

Norepinephrine also plays a central role in regulating the organism's response to stress, via its effects on arousal, selective attention, vigilance, and the encoding of emotional memory. It has been proposed that childhood trauma may contribute to BPD patients' hyperreactivity to stress and interpersonal hypersensitivity, and that these heightened responses may be in part mediated by trauma-related noradrenergic dysregulation (Figuroa and Silk, 1997). Indeed, unmedicated patients with BPD were reported to have significantly decreased platelet alpha 2-adrenergic receptor binding sites (Southwick et al., 1990). Another study of personality-disordered subjects showed that plasma MHPG, the major metabolite of NE, was inversely correlated with lifetime history of aggression (Coccaro et al., 2003). These two conflicting studies are suggestive of heightened versus blunted noradrenergic tone, respectively, in BPD, and to our knowledge there are no other studies examining the noradrenergic system in BPD and its responsivity to stress.

With respect to dissociative symptoms and the noradrenergic system, the few pertinent studies in the literature are suggestive of autonomic blunting. After rape, women scoring high in dissociation showed diminished heart rate and galvanic skin responses (Griffin et al., 1997). Sierra et al. (2002) found that, compared to anxiety disorder and healthy control subjects, individuals with depersonalization exhibited reduced magnitude and increased latency of skin conductance response to unpleasant compared to neutral stimuli suggestive of autonomic hyporesponsivity. Delahanty et al. (2003) reported that in motor vehicle

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