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An analogue investigation of the relationships among perceived parental criticism, negative affect, and borderline personality disorder features: the role of thought suppression

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Abstract

The current study examined the relationships among biological predisposition, social environment, emotion regulation, and features characteristic of Borderline Personality Disorder (BPD). Using an analogue sample, we examined whether thought suppression mediated the relationship of negative affective intensity/reactivity and perceived parental criticism with a composite of BPD features including impulsivity, interpersonal sensitivity, and aggression. Results indicated that thought suppression fully mediated the relationship between negative affect intensity/reactivity and BPD features and partially mediated the relationship between BPD features and perceived parental criticism. Clinical implications, directions for further research, and limitations of the present study are discussed.

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1. Introduction

Borderline Personality Disorder (BPD) is a psychiatric disorder characterized by a range of severe behavioral (e.g., self-injury), emotional (e.g., marked reactivity), cognitive (e.g., dissociation), and interpersonal (e.g., chaotic relationships) problems (DSM-IV, American Psychological Association, 1994). Epidemiological studies estimate that 2–4% of the general population meets criteria for BPD (Swartz, Blazer, George, & Winfield, 1990) and that prevalence rates are highest among young adults (Stone, 1990). A model of BPD that recently has gained prominence is Linehan's biosocial theory (Linehan, 1993) which suggests that BPD symptomatology results from the reciprocal transaction among three primary factors: an invalidating environment, emotional vulnerability, and emotional dysregulation.

1.1. *An invalidating environment*

An invalidating environment encompasses sexual, physical, and emotional abuse (Wagner & Linehan, 1997). An invalidating environment is characterized more broadly by pervasive criticizing, minimizing, trivializing, punishing, erratically reinforcing communication of internal experiences (e.g., thoughts and emotions), and over-simplifying the ease of problem-solving, often coming from those close to the individual (e.g., parents). Additionally, the individual is routinely pathologized as having socially undesirable personality traits (e.g., too sensitive, paranoid, or lazy; Linehan, 1993). Perceived parental criticism, a type of invalidation, has been shown to be associated with poor prognosis across a wide variety of disorders including schizophrenia (Baker, Kazarian, Helmes, Ruckman, & Tower, 1987), substance use disorders (Fals-Stewart, O'Farrell, & Hooley, 2001), mood disorders (Hooley & Teasdale, 1989), and anxiety disorders (Chambless & Steketee, 1999; Renshaw, Chambless, & Steketee, 2001), although this construct has yet to be linked directly to personality disorders.

1.2. *Emotional vulnerability*

Emotional vulnerability refers to a biologically mediated predisposition for affective instability involving heightened sensitivity and reactivity to emotional stimuli, and a slow return to baseline level of emotional arousal. Indeed, several studies indicate that individuals with BPD experience greater intensity of emotions and/or greater variability in their emotional experiences relative to non-BPD controls (Koenigsberg et al., 2002; Levine, Marziali, & Hood, 1997; Stein, 1996). In support of the biosocial model, Yen, Zlotnick, and Costello (2002) administered the Affect Intensity Measure (AIM; Larsen & Diener, 1987) to a sample of BPD patients. These authors found that affect intensity was associated with the number of BPD symptoms endorsed, even after controlling for depressive symptoms. Negative affect intensity is considered within this model to be a central feature of emotional vulnerability in BPD. According to the biosocial model, it is in the context of environmental invalidation (e.g., perceived parental criticism) and emotional vulnerability (e.g., high negative affect intensity) that problems with emotion regulation develop and persist.

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