



Executive neurocognition, memory systems, and borderline personality disorder

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Abstract

Borderline Personality Disorder (BPD) is a common, disabling, and burdensome psychiatric condition. It is characterized by turbulent fluctuations of negative emotions and moods, unstable and conflictual interpersonal relationships, an incoherent and often contradictory sense of self, and impulsive, potentially lethal self-injurious behaviors. The neurobehavioral facets of BPD have not been extensively studied. However, clinical theoreticians and researchers have proposed that the symptoms and behaviors of BPD are, in part, associated with disruptions in basic neurocognitive processes. This review summarizes and evaluates research that has investigated the relationship between executive neurocognition, memory systems, and BPD. Three historical phases of research are delineated and reviewed, and the methodological and conceptual challenges this body of investigation highlights are discussed. Laboratory-based assessment of executive neurocognition and memory systems is integral to an interdisciplinary approach to research in BPD. Such an approach holds promise in elucidating the

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neurobehavioral facets, development, diagnostic boundaries, prevention, and optimal interventions for this debilitating and enigmatic disorder.

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Borderline personality disorder (BPD; *Diagnostic and Statistical Manual, 4th Edition; DSM-IV; American Psychiatric Association, 1994*) is characterized by turbulent, anxious, angry, and depressive emotional states, unstable interpersonal relationships, an incoherent and often contradictory self-concept, and impulsive and often dangerous behaviors such as self-injury and drug abuse. Following the pioneering clinical descriptions of borderline personality (*Grinker, Werble, & Drye, 1968; Kernberg, 1967*), empirical research of BPD has progressed over the last 20 years. Based on recent population prevalence estimates (0.3–0.7%), between six-hundred thousand and 1.4 million adults in the United States meet diagnostic criteria for BPD (*Lenzenweger, Loranger, Korfine, & Neff, 1997; Samuels et al., 2002; Torgersen, Kringlen, & Cramer, 2001*). The suicide rate for BPD individuals is about 10% (*Paris, 2002; Stone, 1993*), comparable to the other psychiatric disorders such as schizophrenia and major depression. In addition, 69% to 75% of individuals with BPD engage in self-injurious behaviors (*Clarkin, Widiger, Frances, Hurt, & Gilmore, 1983; Cowdry, Pickar, & Davies, 1985*), and the frequency of self-injurious behaviors is more than in any other psychiatric diagnosis (*Stanley, Winchel, Molcho, Simeon, & Stanley, 1992*). Individuals with BPD exhibit high drop out rates and variable improvement in psychotherapy (*Clarkin, 1996*), and respond only partially to psychopharmacological therapies (*Soloff, 2000*). Additionally, this diagnostic group utilizes health care services more frequently than any other psychiatric group (*Bender et al., 2001*). Despite this troubling clinical picture, BPD has not received research attention and widespread clinical focus commensurate with the suffering and mortality it causes.

There is considerable reason to suspect that there are disruptions in basic executive neurocognition and memory processes in BPD. Central to the symptoms of BPD is unstable and dysregulated inhibitory control over behavior, emotion, and cognition. The acquisition of executive neurocognition is inextricably linked to emotion and personality development, and inhibitory capacity influences the acquisition of prosocial behaviors, affect regulation, and problem solving abilities (*Derryberry & Reed, 1994; Posner & Rothbart, 2000*). These capacities are commonly impaired in BPD. From a clinical perspective, neurocognitive function predicts response to intervention in other clinical groups (e.g., *Smith, Hull, Romanelli, Fertuck, & Weiss, 1999*). Should BPD individuals, or subgroups of them, evidence a characteristic constellation of inhibitory and memory characteristics, treatment could be tailored to complement them, leading to improved treatment planning and interventions. From a scientific vantage point, BPD is a diagnostic entity that can be used to elaborate the understanding of basic cognitive and affective processes. The study of emotion, mood, temperament, affect regulation, and their relationship with personality, cognition, and psychopathology are at the center of rapid, exciting developments in cognitive (*Gazzaniga, 2000*) and affective science (*Davidson, Scherer, & Goldsmith, 2003*).

Previous reviews have emphasized the clinical implications of neurocognitive functioning in BPD and other personality disorders (see *Gorton, Swirsky-Sacchetti, Sobel, Samuel, & Gordon, 1999; O'Leary & Cowdry, 1994*). This review, by contrast, focuses on research that has attempted to systematically

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