

## **Cognitive Features of the Borderline Phenotype**

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Cognitive psychology/cognitive neuroscience involves identifying the basic component processes of perception, attention, categorization, discrimination, generalization, learning, memory, and thought in general, how these processes work together, how they are affected by emotion, and the conditions under which they are disrupted. Broadly defined, almost all symptomatic aspects of borderline personality reflect the outcome of some atypical cognition. For example, cognitive schemas of the self and others relate to identity disturbance, problematic interpersonal relationships, and to stress-related paranoid ideation or dissociation. Characteristic affective dysregulation can in part be understood as cognitive systems going “off-line,” as can impulsive behaviors (i.e., acting without thinking). What then, will help to clarify cognitive features of borderline pathology, systematically advance our understanding of the phenotype, and consequently improve the assessment of the disorder? In approaching these difficult questions, one helpful distinction is among types or levels of cognitive processing. For instance, higher order cognition occurs over a relatively long period of time and involves complex reflective operations. Many basic processes of perception and reflection occur on the order of milliseconds but nevertheless may accrue or interact to affect cognition, emotion, and behavior. A second key consideration is potential interactions between cognition and emotion.

Clinical research on “schemas” of self or others has provided an important empirical approach for studying the ways that borderline patients represent others and themselves. These approaches allow gross level specification of how the ways of thinking about self and others can aid the development of models of the disorder (see Horowitz, 1991). This line of work supports several profitable approaches for treatment, for instance, Cognitive Therapy (Beck, 1976; Beck, Freeman et al, 1990), Dialectical Behavioral Therapy (Linehan, 1993; Linehan et al, 2006), and Schema Therapy (Young, Klosko, & Weishaar, 2003; Kellogg & Young, 2006). These approaches share in common a goal to alter a patient’s cognitive schemas and change maladaptive behavior patterns to healthier ways of thinking and relating. These cognitive treatments have proven to be effective psychotherapies for borderline personality disorder, and recent findings in neuroscience corroborate the notion that emotions can be influenced by redirecting thoughts at this level (e.g., Ochsner, Bunge, Gross, & Gabrieli, 2002; see below). Interestingly, Beck, Freeman, and associates (1990) argue that by first addressing a relatively more basic process of dichotomous thinking typical in BPD, therapeutic interventions aimed at higher-order cognitive schemas and patterns of behavior will prove effective.

While there are many aspects of cognition relevant to the disorder, focusing on those most relevant to cognition-emotion interactions is likely a fruitful avenue for current efforts given the persistence of affective instability as an enduring, core feature of BPD (McGlashan et al, 2005; Zanarini et al, 2003). Further, clarifying cognition and cognition-emotion interactions and identifying discrete deficits (or specific combinations of deficits) characteristic of borderline may provide markers more proximal to biologic or

genetic factors. Because of their basic structure, there is also the possibility that they will be more *reliably* identifiable. Neuroimaging findings also document general heightened emotional sensitivity in BPD patients. Several studies have demonstrated hyper-reactive amygdala activity in response to emotionally charged stimuli for BPD patients compared to normal controls (e.g., Herpertz, et al, 2001; Donegan et al, 2003), and the amygdala, a structure in the limbic system, is not only involved with the generation of emotional states but also plays a role in perception of fear (Davis, 2000).

Cognitive studies have examined the processing of affective information in BPD patients. In the area of memory, data show that the presence of borderline features is associated with how patients respond toward affective material. For example, both general recognition memory deficits and mood congruent valence selectivity effects have been demonstrated in BPD patients. Kurtz and Morey (1999) noted affective selectivity differences between patients with BPD and those with major depression in recall memory, despite negligible differences between the two patient groups in the severity of depressive symptoms and in the general level of psychopathology. Results from a study of directed forgetting conducted by Korfine and Hooley (2000) showed BPD subjects were less able than normal controls to inhibit the recall of symptom related stimuli (words) when explicitly instructed not to remember them. Thus, BPD patients may be more likely to generate negative emotional material from memory. One hypothesis, based on the source-monitoring framework of memory (Johnson et al, 1993), would be that BPD patient's criteria for retrieving and evaluating autobiographical memories would be biased toward negative features and emotional features at the expense of other contextual features. Although these processes have not been directly linked to neuropathology, neuroimaging studies of BPD have shown abnormalities in brain areas implicated in relevant memory processes. For example, in dorsolateral prefrontal cortex (DLPFC) deficits in N-acetylaspartate, an indicator of neuronal integrity, have been shown (Tebartz van Elst et al, 2001) and hyperactivation in response to emotional stimuli (Schmahl et al, 2003, 2004). In hippocampus, lowered resting state activity has been reported (Juengling et al, 2003) as have reduced volumes (Driessen et al., 2000).

At the same time, there is evidence that BPD may also be associated with criterion differences in the cognitive process of stimulus evaluation. Kurtz and Morey (1998) found that BPD patients made more negative evaluative judgments to ambivalent or neutral stimuli than either a depressed group or normal controls, which did not significantly differ from each other. Herpertz and colleagues (2000) found that BPD patients reported relatively neutral affective material as more unpleasant than other patients despite observing no between-group differences in physiological responsiveness. Wagner and Linehan (1999) found BPD patients better able to discriminate between emotional expressions, especially fearful faces, compared to controls and non-BPD subjects with a history of abuse, a finding that suggests that BPD patients are perhaps more attuned to negative emotional stimuli.

Neurocognitive deficits have long been hypothesized to underlie impulsivity in BPD (Hoffman-Judd, 2005) although findings have not been consistent. One reason for

these mixed findings may be that, in many studies, attentional factors are not controlled (Monarch, Saykin, & Flashman, 2004). Paris, Awlkwowitz, Guzder, Joseph, and Feldman (1999) studied higher order cognitive functions (learning from errors, conceptual-level responses, maintaining a conceptual set, flexibility) in children with borderline features and also included the Continuous Performance Test (CPT) to measure attention. Subjects evidenced deficits performing these higher-order cognitive operations, but inconsistent attentional skills assessed using the CPT suggested that in part these deficits were due to attentional factors.

Work by Posner and colleagues (2002) examined attentional mechanisms in BPD patients using a purely cognitive task that has been well studied in other domains, the Attention Network Test (ANT; Fan et al, 2002). The ANT requires participants to make judgments about a cue in the context of distracters or “flankers” that surround the cue thereby introducing conflict designed to produce varying levels of interference with the judgments to be made. BPD patients had significantly greater difficulty performing the task with higher levels of conflict relative to normal controls (a temperamentally matched contrast group did not differ from either the patients or controls in performance). For the BPD patients, response time to perform the task was also inversely correlated with a self-reported measure of effortful control. From these findings, it appears that BPD patients have difficulty in conflict monitoring even when emotion is not involved. Results from this behavioral study are suggestive of abnormalities in anterior cingulate cortex (ACC), a brain area associated with cognitive control and performance on the ANT task (Bush, Lu, & Posner, 2000; see Botvinick, Nystrom, Fissell, Carter, & Cohen, 1999). Results from PET neuroimaging studies have shown a failure to activate ACC in BPD subjects with childhood sexual abuse (CSA) compared to CSA without BPD in response to abandonment (Schmahl et al, 2003) and trauma (Schmahl et al, 2004) scripts. Volume reductions in this area have also been reported in BPD patients (Driessen et al, 2000; Hazlett et al, 2005).

In normal subjects, there is evidence that varying the instructions for viewing pictures of emotional faces (e.g., passively view, focus on the emotional elements, focus away from the emotional elements) can modulate the level of amygdala activity and this modulation is accompanied by activity in right prefrontal cortex (Hariri et al, 2003; Lange et al, 2003). Studies where normal subjects are instructed to reappraise negative emotional information help identify emotion regulation processes and suggest ways to clarify cognitive mechanisms that occur in clinical interventions as well as illustrate the value of decomposing complex cognitive processes. For example, Ochsner, Bunge, Gross, and Gabrieli (2002) distinguish between three different processes and note brain areas associated with each: (1) the active generation of a strategy for cognitively reframing an emotional event in unemotional terms (lateral prefrontal cortex); (2) monitoring interference between “top down” appraisals and “bottom up” generated affect (dorsal anterior cingulate cortex); and (3) re-evaluating internal states to monitor changes in emotional state during the reappraisal process (medial prefrontal cortex). In their study, brain regions activated during re-appraisal included lateral and medial prefrontal cortex. Activation of ventral-lateral prefrontal cortex was inversely correlated with activation of the amygdala and medial prefrontal

cortex, suggesting that this area is important for modulating emotional processing. In response to viewing pictures from the emotionally charged International Affective Picture Set (IAPS), Herpertz et al (2001) found simultaneous activations in both amygdala and medial areas of prefrontal cortex compared to normal controls. Finally, Oschner and colleagues (2002) noted a positive correlation between cingulate activation and effective reappraisal (measured by ratings of the negatively valenced stimuli). This is consistent with the role of anterior cingulate cortex in monitoring conflicts, for instance in this case between initial emotional appraisals and cognitively restructured appraisals. Together with findings noted above suggesting ACC activation deficits, these results hint that BPD individuals may be less able to consider alternatives when faced with difficult emotional situations.

The orbitofrontal cortex (OFC) has been implicated in emotion regulation (see Rule, Shimamura, & Knight, 2002). Neuroimaging studies of BPD have reported a failure to activate OFC in response to emotional stimuli (Schmahl et al., 2004) and PET studies have reported less OFC activation for BPD patients, relative to normal controls, in resting studies (Soloff et al, 2003) and challenge studies (Soloff et al, 2000). Using a variety of self-report measures, Berlin, Rolls, and Iversen (2005) compared BPD patients to patients with OFC lesions, patients with lesions other than OFC-lesions, and normal control subjects. On a variety of self-report measures and cognitive tasks aimed at assessing impulsivity, BPD and OFC-lesion patients reported greater levels of impulsivity and anger, and less happiness relative to patients with other lesions and normal controls, and BPD patients were significantly different from OFC-lesion patients in these respects as well. Interestingly, BPD and OFC-lesion patients did not differ from normals on a spatial working memory task (whereas the patients with non-OFC lesions showed a deficit in this regard). However, BPD patients reported more inappropriate social behaviors than the OFC patients consistent with the idea that other brain areas, e.g., amygdala and ACC, may play a role as well.

In summary, brain areas implicated in conflict detection (anterior cingulate cortex), emotional control (orbitofrontal cortex), emotional generation/perception (amygdala), and the consolidation (hippocampus) and retrieval (dorsolateral prefrontal cortex) of memories are areas that have been found in BPD neuroimaging studies to activate (or deactivate depending on the task) during emotional processing tasks (for reviews and efforts to integrate these findings, see Brendel, Stern, & Silbersweig, 2005; Johnson et al, 2003; McCloskey, Phan, & Coccaro, 2005; Putnam & Silk, 2005; Schmahl & Bremner, 2006). In addition, reduced volumes are found in many of these areas (e.g., Driessen et al, 2000; Hazlett et al, 2005; Irle, Lange, & Sachsse, 2004; Rusch et al, 2003; Tebartz van Elst et al, 2003). Future work aimed at clarifying patterns of connectivity among these brain regions would benefit from tasks that isolate and control specific component processes of cognition and their associated neural activation patterns. There may be certain advantages to also adopting an approach of examining basic *component processes* of cognition in investigating the borderline phenotype. For example, the Multiple Entry Modular Memory System (MEM; Johnson, 1992) is a component process approach to cognition that could provide a theoretical framework for studying borderline psychopathology. MEM describes elementary perceptual and

reflective processes (e.g., *locating, identifying, refreshing, noting, reactivating, rehearsing*) that constitute attention, working memory, long-term memory, problem solving, and other higher-order cognition. Understanding which aspects of cognition are aberrant could clarify the nature of the BPD phenotype and distinguish it from other forms of psychopathology.

### Conclusions

The goal to clarify cognitive features specific to the BPD phenotype can, in turn, help to establish reliable markers that reflect the mechanisms of the disorder and that are likely to have biological correlates. While there are many aspects of cognitive function relevant to the disorder, focusing on cognition-emotion interactions is likely a fruitful area to focus current efforts in light of the persistence of affective instability as a core feature of the BPD phenotype. A critical consideration for progress in this area concerns the manner in which cognitive features are studied. Often, tasks are used that engage multiple processes of cognition. For instance, attention, executive control, and memory are required on certain neuropsychological tests. Isolating these factors by employing experimental paradigms using tasks designed to tap into more discrete components or processes of cognition may help to resolve conflicting findings. For instance, is poor emotion regulation due to attention to emotional material, or an inability to disengage from emotional material, or by generating emotional material from memories of past experience, or some combination of these aspects of perception and thinking? It has been argued that greater attention to *component processes* of cognition is a productive approach to clarifying the cognitive features of the BPD phenotype. Finally, it is important to note that the way that these features “fit together” and interact with development throughout the life span in the clinical expression of the BPD phenotype is important as well.

A cautionary note with regard to *assessment* of BPD cognitive features: these experimental approaches to study cognitive features of BPD are at the investigative stage. Identification of reliable group differences can inform the way that we conceptualize and understand the BPD phenotype, provide important clues about the underlying mechanisms of borderline pathology, and suggest neurocognitive deficits. However, there are limitations in regard to individual differences and to consider these experimental paradigms assessment tools may be inaccurate. To move from the realm of experimental studies to reliable assessments of cognitive features will require validation procedures such as those employed for psychometric development of neuro- or other types of psychological testing. For instance, validation studies would include test-retest procedures to develop reliability and longitudinal studies to distinguish state effects from trait effects. Questions might include: Are there certain cognitive features that are stable whether or not a patient is “in episode” while other features appear to be state dependent? The former may be suggestive of a vulnerability to the disorder whereas the latter a manifestation of the disorder. Results from experimental cognitive studies guide where to look for reliable deficits when assessing patients, but determining under what circumstances they occur (e.g., what mood states, or with what co-occurring disorders) will be part of the work.

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