# The Impact of Emotional Distress on Cognitive Performance in Borderline Personality Disorder

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ABSTRACT

THE IMPACT OF EMOTIONAL DISTRESS ON COGNITIVE PERFORMANCE IN BORDERLINE PERSONALITY DISORDER

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Individuals with borderline personality disorder (BPD) are prone to intense emotional reactions and dysfunctional interpersonal relationships, which may be associated with disruptions in cognitive functioning. However, research comparing neurocognitive functioning in BPD compared to patients with comorbid disorders like MDD and healthy control groups has been inconclusive. This study was the first to directly measure BPD individuals' working memory capacities under stressful conditions, using an experimentally manipulated, in-vivo social stressor. The primary aims of this study were to investigate the impact of emotional distress on working memory performance in the context of a psychological stress procedure (Trier Social Stress Test) and to determine whether emotion-induced working memory disruption was stronger for participants with BPD ($n$ = 60) than for participants with MDD ($n$ = 30) or healthy controls ($n$ = 21). Results showed that emotional distress positively predicted working memory errors in the sample overall, with self-reported feelings of confusion and vigor accounting for this relationship. However, there were no basic working memory differences between BPD participants, MDD participants, and controls. BPD participants were also not more likely to have impairments in working memory as a consequence of emotional distress compared to participants with MDD. Participants with BPD were more likely to have had a history of self-injurious behavior, showed poorer psychosocial functioning, and showed higher levels of

depression, anxiety, aggression, and impulsivity. When the effects of emotional distress were controlled for, participants with BPD were shown to have superior working memory performance, while MDD participants were shown to have poorer working memory performance, compared to the sample mean. Findings from the current study underscore the need to account for emotional distress when examining working memory in BPD and MDD groups. Mood fluctuations and emotional reactivity may play a larger role than pathophysiological factors in characterizing neurocognitive performance in these groups. These findings could point to a deficit in MDD, perhaps characterized by insufficient reactivity to the mobilizing effects of mild stress. Alternatively, BPD individuals' greater attunement and sensitivity to others' emotional states may paradoxically confer an advantage when pure attentiveness and concentration are called for. Future research should aim to identify psychological and neurocognitive strengths among individuals with BPD. Given the equivocal and complex findings on neurocognitive performance in BPD to date, more research is needed to develop a clear profile.

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# CHAPTER I:

# INTRODUCTION AND LITERATURE REVIEW

The ability to maintain cognitive focus when under stress is of paramount importance in daily life, and is central to successful functioning in occupational and social domains. Few studies have investigated the impact of emotional distress on cognitive performance in patients with borderline personality disorder (BPD), and none have examined this in the context of negative evaluation fears and hypersensitivity to rejection. The primary aim of this study was to investigate the impact of emotional distress on working memory performance, and to determine whether emotion-induced working memory disruption was stronger for participants with BPD than for participants with major depressive disorder (MDD) or healthy controls. Our sample was drawn from a larger set of patients who participated in a research study at New York State Psychiatric Institute (NYSPI).

As part of their participation in a parent study, all participants underwent a modified psychological stress procedure in order to evaluate heart rate reactivity and cortisol levels. Two procedures were used to induce psychological stress and were administered by study confederates clad in white laboratory coats who displayed neutral affect and gave minimal feedback to participants. Participants were first asked to speak about themselves extemporaneously for five minutes, followed by four increasingly rapid trials of a working memory task in which they were instructed to add a series of single digits in sequence. Participants completed mood ratings immediately before and after the procedures, and at follow-up 25 minutes later.

As a result of these procedures, it is likely that participants were emotionally distressed by the first half of the psychological stress test while they participated in the working memory

task. Despite the fact that the purpose of the working memory task in the parent study was to induce further psychological stress, the accuracy of participants' performance on the working memory task could serve as a valuable proxy measure of the ability to regulate emotional responsiveness to stress and to allocate cognitive resources to achieve a specific goal—skills which are seen as key in BPD-oriented treatment models like dialectical behavior therapy (DBT).

Because the induction of stress in our study paradigm took place in an interpersonal context, participants may have been experiencing the acute sensitivity to rejection and negative evaluation that is a hallmark of BPD. In addition, it is unknown based on previous research whether patients with BPD experience more severe emotion-induced cognitive disruption than patients with other psychiatric disorders, particularly a highly comorbid disorder like MDD. Research in this area could help to identify patients who are most vulnerable to cognitive disruption and would benefit from focused treatment efforts targeting these deficits.

**Borderline personality disorder**

Borderline personality disorder (BPD) is a chronic and often debilitating psychiatric disorder characterized by a pervasive pattern of instability. Key areas of dysfunction include mood lability and reactivity; unstable identity and cognitive dysregulation, including paranoid ideation and dissociation when under stress; chaotic interpersonal relationships (i.e., intense, volatile relationships marked by alternating extremes of idealization and devaluation); and difficulties with impulse control and behavioral disinhibition. The disorder has been estimated to occur in 1-2% of the general population (Swartz, Blazer, George, & Winfield, 1990; Torgersen, Kringlen, & Cramer, 2011), but accounts for a disproportionately high number of patients utilizing inpatient psychiatric services (Widiger & Weissman, 1991).

One of the core underpinnings of the disorder is a highly sensitive and emotionally vulnerable temperament, a feature which manifests itself in a variety of contexts. Notable among these is the interpersonal nature of many of the difficulties encountered by these individuals. Striking differences have been observed in the phenomenology of suicidal behavior between individuals with BPD and those with MDD. For instance, patients with BPD are more likely to report feelings of relief and diminished distress after a suicide attempt, while individuals with MDD tend to report feelings of regret and no significant change in sadness or distress following an attempt (Stanley & Brodsky, 2005). This points to the role of suicidality as a means of affect regulation among those with BPD, even in response to stressors which individuals without the disorder would find far less troubling. In addition, triggers of suicidal attempts and behavior among individuals with BPD are far more likely to be interpersonal, and center around perceived abandonments, rejections, slights, and losses (Brodsky, Groves, Oquendo, Mann, & Stanley, 2006).

Empirical, clinical, and anecdotal literature has led some to characterize the temperament of individuals with BPD as 'exquisitely' sensitive (Stanley & Siever, 2010). An emotionally sensitive temperament is characterized by a low threshold for the detection of emotional cues, intense responses to emotional stimuli, and a slow return to baseline levels of distress (Linehan, 1993). That is, individuals with BPD are likely to possess a lower threshold for emotional distress and to exhibit stronger responses of longer duration than non-BPD individuals. The early invalidation of emotional experiences and insufficient labeling or recognition of emotional states are thought to contribute to this constellation of emotion regulation difficulties in BPD (Linehan, 1993). In addition, emotional sensitivity and impulsivity in childhood may be potentiated by life

experiences and risk factors that further exacerbate emotion regulation problems (Crowell, Beauchaine, & Linehan, 2009).

**Emotion regulation**

Emotion regulation refers to the ability to modulate emotional responses to internal or environmental stimuli, either through amplification, attenuation, or maintenance of emotional states (Gross, 2002). The ability to exercise effective control over emotions involves accessing cognitive resources (Ochsner & Gross, 2005), most commonly to engage in cognitive reappraisal of emotional stimuli or to recruit behavioral suppression or modification of emotional responses once they have already been triggered (Gross, 2002). The selection of particular emotion regulation strategies varies widely according to circumstances and individual differences, and may include either the 'up-regulation' or 'down-regulation' of emotions (Gross, 1998).

Due to their pervasive instability and difficulties with emotion regulation, as well as chaotic interpersonal relationships, individuals with BPD have difficulty achieving non-mood-dependent goals and inhibiting behavioral responses to powerful emotional stimuli, resulting in an escalation of volatile and emotionally triggering situations (Bohus, Schmahl, & Lieb, 2004). The overall capacity to modulate emotional responses and to flexibly adapt one's choice of appropriate regulation strategies is associated with an array of beneficial psychosocial outcomes, including academic achievement (Bandura, Caprara, Barbaranelli, Gerbino, & Pastorelli, 2003), prosocial behavior (Eisenberg, Fabes, Guthrie, & Reiser, 2000), social acceptance and likability (Eisenberg & Fabes, 1992), and psychological well-being (Gross & John, 2003).

**Neurocognitive functioning in BPD**

While the clinical features of BPD are well-described, there are a wide range of explanations as to neurobiological underpinnings of the disorder. Researchers have sought to

identify areas of neuropsychological functioning that may be distinct in BPD, both as a means of understanding the disorder and to guide targeted treatment efforts. Some studies on neurocognitive differences between individuals with BPD and healthy controls have shown frontal lobe dysfunction in BPD (Dinn et al., 2004; Ruocco & Trobst, 2003; Tebartz van Elst et al., 2003), with specific deficits observed in a range of functions. A meta-analysis (Ruocco, 2005), for example, examined 10 studies comparing neuropsychological functioning in BPD to healthy controls across six domains. Mean weighted effect sizes (Cohen's d) by domain were large for planning (d = -1.43, SE = 0.20); moderate for attention (d = -0.59, SE – 0.22), speeded processing (d = -0.68, SE = 0.14), visuospatial ability (d = -0.59, SE = 0.11), and learning and memory (d = -0.66, SE = 0.15), with greater impairments shown for nonverbal memory than for verbal memory; and small for cognitive flexibility (-0.29, SE = 0.13).

The fact that the largest deficit was observed for planning is consistent with the high levels of impulsivity found in this population (Links, Heslegrave, & Reekum, 1999), and points to potential deficits in executive functioning. Deficits in executive functioning have been found in a number of studies comparing participants with BPD to healthy controls. Specific deficits in this domain include decision-making and planning (Bazanis et al., 2002; Beblo et al., 2006; Black et al., 2009; Haaland & Landro, 2007; LeGris, Links, van Reekum, Tannock, & Toplak, 2012), response inhibition (Rentrop et al., 2007), and cognitive flexibility and abstraction ability (Lenzenweger, Clarkin, Fertuck, & Kernberg, 2004). Visual memory is another frequently reported neurocognitive deficit in BPD compared to healthy controls (Beblo, Mensebach, Wingenfeld, Rullkoetter, & Schlosser, 2011; Beblo et al., 2006; Harris, Dinn, & Marcinkiewicz, 2002; Judd & Ruff, 1993; O'Leary, Brouwers, Gardner, & Cowdry, 1991; Swirsky-Sacchetti et

al., 1993). Deficits in visual memory in BPD participants have been linked to reduced volume in the hippocampus (Driessen et al., 2000; Irle, Lange, & Sachsse, 2005).

Studies examining working memory processes in BPD are of particular significance, since working memory requires sustained attention and therefore is particularly vulnerable to interference from the intense emotional responses that are characteristic of BPD (Dolcos & McCarthy, 2006). Findings from working memory studies also help to shed light on the complexity of neuropsychological functioning in BPD. For example, BPD patients may show deficits in accuracy on classic working memory tasks such as the N-Back (Hagenhoff et al., 2013; Black et al., 2009; Lazzaretti et al., 2012), though it is not clear whether increased cognitive load worsens accuracy among these participants (Hagenhoff et al., 2013; Stevens, Burkhardt, Hautzinger, Schwarz, & Unckel, 2004). Black et al. (2009) executed a well-controlled study which compared BPD participants to controls and found that the BPD group's deficits in working memory, perseveration, and decision-making were not attributable to the effects of IQ, depression, or alcohol use.

Interestingly, BPD participants may sometimes show faster reaction times compared to controls on working memory tasks (Hagenhoff et al., 2013) but potentially at the expense of accuracy (Rentrop et al., 2007), resulting in a speed-accuracy tradeoff. However, other studies comparing BPD participants to controls in visual working memory have found no evidence for such a tradeoff, despite greater impairments in accuracy among the BPD group (Keilp et al., 2007; Stevens et al., 2004). One potential explanation for accelerated reaction times among BPD groups may lie in the relationship between BPD traits and harm avoidance (Ball, Tennen, Poling, Kranzler, & Rounsaville, 1997), such that negative emotions actually motivate individuals with BPD to avoid punishments and negative consequences. For example, negative affective states

have been shown to predict greater impulse control for individuals with BPD traits compared to those without BPD traits (Chapman, Leung, & Lynch, 2008). Alternatively, faster reaction times in these studies could simply reflect the impulsive response style that is characteristic of individuals with BPD, in both clinical and experimental settings (de Bruijn et al., 2006; Haaland & Landro, 2007).

Other studies comparing BPD participants to healthy controls on neurocognitive measures have found no differences. Sprock, Rader, Kendall, and Yoder (2000), for example, found no differences between groups of BPD participants and controls on measures of memory or executive functioning. Other studies have shown impairments for BPD participants in some neurocognitive domains but not others. Kunert, Druecke, Sass, and Herpertz (2003) found that although individuals with BPD demonstrated poorer planning and greater impulsivity than controls, they performed equally well on neuropsychological tests of learning and memory, vulnerability to interference, intelligence, and attentiveness. Another study compared global executive functioning and working memory capacities among BPD and control participants, and found deficits for the BPD group in decision-making but no other executive functioning areas (LeGris et al., 2012). Similarly, BPD participants have been shown to demonstrate poorer working memory performance, but with no accompanying deficits in sustained attention (Lazzaretti et al., 2012).

Other studies have yielded null findings across a number of neurocognitive domains, such as working memory and attention (Dinn et al., 2004; Fertuck et al., 2006b; Lenzenweger et al., 2004; Sprock et al., 2000), learning and memory (Cornelius et al., 1989; Dinn et al., 2004; Fertuck et al., 2006b; Bazanis et al., 2002), language abilities (Cornelius et al., 1989), intelligence (Fertuck et al., 2006b; Kunert et al., 2003), motor abilities (Cornelius et al., 1989;

Fertuck et al., 2006b), visuospatial abilities (Cornelius et al., 1989), processing speed (Fertuck et al., 2006b); and executive functioning (Dinn et al., 2004; Fertuck et al., 2006b; Hagenhoff et al., 2013).

Thus, studies comparing neuropsychological functioning in BPD to that of healthy controls have yielded mixed results. The influence of confounding variables, such as intelligence, has not been adequately addressed in most studies (Fertuck et al., 2006a), with some exceptions (Black et al., 2009). Furthermore, it is not known whether observed neurocognitive differences represent primary functional deficits, or whether they are better understood as transient fluctuations in neurocognitive functioning, owing to the chronic cognitive instability of the disorder. In their study of neurocognitive deficits in BPD, Black et al. (2009) evaluated the predictive utility of neuropsychological tests compared to personality traits in accounting for the variance in BPD and found that although the BPD group showed deficits, neuropsychological tests did not improve explained variance over and above personality traits. They suggested that even when neuropsychological impairments are found among individuals with BPD, the findings may be more attributable to factors such as impulsivity and mood reactivity.

**Impact of emotion on cognitive functioning**

One promising area of inquiry into the neurobiological substrates of BPD is research examining the impact of emotionality on cognitive functioning, and how this neural circuitry may be different in BPD than in healthy controls. This is particularly important given that the extant literature on neurocognitive functioning BPD has yielded equivocal results, such that the role of transient mood states and distress may be particularly important.

Empirical investigations into the neural circuitry of emotion regulation have suggested that orbitofrontal regions (Beer, Shimamura, & Knight, 2004) and subgenual cingulate regions

(Silbersweig et al., 2007) are involved in the cognitive evaluation of emotional experiences, while the dorsolateral prefrontal cortex (DLPFC) has been implicated in the executive control of responses to emotional stimuli (Johnson, Hurley, Benkelfat, Herpertz, & Taber, 2003). In addition, a key feature of emotion regulation is the ability to orient one's attention away from emotional stimuli when necessary, and to attend selectively to certain aspects of one's immediate experience but not others (Anderson, Christoff, Panitz, De Rosa, & Gabrieli, 2003). It has been well-documented that emotional stimuli can compete for attentional resources and can interfere with complex cognitive functions under some circumstances (Vuilleumier, 2005). In particular, researchers have suggested that working memory—the ability to temporarily store and manipulate new information (Baddeley, 1992)—is especially vulnerable to interference from emotional stimuli, since it requires uninterrupted focus on a particular goal or task (Dolcos & McCarthy, 2006).

Data from functional magnetic resonance imaging (fMRI) studies offer compelling evidence for a complex interactional process between cognitive and affective systems, which helps to explain why and how emotion interferes with cognitive functions. In one of the only imaging studies to examine the neural mechanisms of emotion-induced cognitive disruption, Dolcos and McCarthy (2006) suggested that the ventral system of 'hot' emotional processing (i.e., the amygdala and ventrolateral prefrontal cortex) interacts with the dorsal system of 'cold' executive functioning (i.e., the DLPFC and lateral parietal cortex). Specifically, they found that during emotion-based distraction of a cognitive task, activation in the ventral system significantly disrupted neural activity in the dorsal executive areas, regions which have been shown to correlate with working memory functions (Smith & Jonides, 1999) and with the ability to inhibit attention to irrelevant information (Shimamura, 2000).

**Impact of emotion on cognitive functioning in BPD**

Compared to neutral stimuli, negative emotional stimuli appear to exert a greater distracting influence on cognitive control in most individuals (Dolcos & McCarthy, 2006). However, because individuals with BPD possess a lower threshold for the detection of emotional content and experience more intense emotional reactions, they are likely to experience greater cognitive disruption when emotionally primed (Winter, Elzinga, & Schmahl, 2014). In addition, the lengthier duration of emotional responses in individuals with BPD delays the return of critical thinking and executive functioning, which may prolong their vulnerability to further emotionally dysregulating experiences.

A number of studies have begun to explore the ways in which cognitive resources become less available to individuals with BPD in moments of psychological stress, and have provided support for the cognitive-affective interaction mechanism described by Dolcos and McCarthy (2006). A recent study by Krause-Utz et al. (2012) found that, compared to healthy controls, individuals with BPD showed greater activation in the amygdala and reduced neural activity in the DLPFC—and exhibited slower reaction times on a working memory task—in response to emotionally distressing images. Studies examining components of the interaction system separately in BPD patients have implicated reduced prefrontal control or inhibition of emotion-induced interference (Domes et al., 2006; Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2006; Korfine & Hooley, 2000; Mensebach et al., 2009; Silbersweig et al., 2007) in the dorsal system and elevated activity in the amygdala (Donegan et al., 2003; Herpertz et al., 2001; Koenigsberg et al., 2009; Minzenberg, Fan, New, Tang, & Siever, 2007) and other limbic structures such as the left anterior cingulate cortex (Schnell, Dietrich, Schnitker, Dauman, & Herpertz, 2007) in the ventral system. The impact of emotional stimuli on cognitive processing

in BPD patients has been found to be related to self-reports of emotional appraisals, such as a non-accepting stance toward emotions (Gratz et al., 2006), but may also occur at a nonconscious or subliminal level (Arntz, Appels, & Sieswerda, 2000).

Emotion-induced cognitive disruption in BPD may be mediated by hypervigilance and hypersensitivity to rejection. Heightened sensitivity to rejection is a well-known clinical feature of BPD (Ayduk et al., 2008; Linehan, 1993; Staebler, Helbing, Rosenbach, & Renneberg, 2011), and accompanying feelings of abandonment, loss, and loneliness are highly related to suicide attempts and self-injurious behavior in this population (Brodsky et al., 2006; Stiglmayr, Shapiro, Stieglitz, Limberger, & Bohus, 2001; Welch & Linehan, 2002). Heightened rejection sensitivity in BPD has been linked to greater sensitivity to fearful stimuli (Wagner & Linehan, 1999) as well as the tendency to make negative interpretations of otherwise neutral interpersonal stimuli (Donegan et al., 2003). This process is associated with hypervigilance in scanning the environments for threats (Herpertz et al., 2001), a process which is heavily governed by the amygdala and other limbic circuits (Öhman, 2005). Thus, there may be additional strain on cognitive resources in individuals with BPD as a result of heightened rejection sensitivity, particularly in situations which prime the activation of negative evaluation fears. This process of threat detection may become even more vigorous and demand more attentional resources – and therefore lead to greater depletion of working memory – when stimuli are ambiguous and subject to multiple interpretations, as can occur in social situations.

Researchers have used various experimental methods to examine the direct impact of emotional stimuli on cognitive processes in BPD, most often involving lists of emotionally valenced words or images (e.g., Korfine & Hooley, 2000; Mensebach et al., 2009; Silbersweig et al., 2007). However, there has been a relative dearth of research examining the impact of direct

(i.e., in vivo) interpersonal triggers of emotional distress on cognitive functioning in BPD. Individuals with BPD may be especially sensitive to emotional priming in an interpersonal context (Berenson et al., 2009). The salience of interpersonal stressors in BPD pathology has been well-documented. For example, triggers of suicide attempts among BPD patients are most often interpersonal in nature (Brodsky, Groves, Oquendo, Mann, & Stanley, 2006).

**Comorbidity with MDD.** Discerning the impact of emotional stimuli on cognitive performance in BPD patients is often complicated by the comorbid presence of major depression. Indeed, major depressive disorder (MDD) is one of the most common comorbidities observed in BPD (Grant et al., 2008; Zanarini et al., 1998). The effects of depressive symptomatology on cognitive performance, independently of personality disorders, have received a great deal of empirical attention (e.g., Landrø, Stiles, & Sletvold, 2001; Paelecke-Habermann, Pohl, & Leplow, 2005). One of the most consistent findings has been a mood-congruence bias for memory and attention, in which individuals with MDD are more likely than controls to recall information related to negative emotions (Bradley, Mogg, & Millar, 1996; Bradley, Mogg, & Williams, 1995; Direnfeld & Roberts, 2006; Gotlib et al., 2004) and to orient attention toward negative emotional cues (Elliott, Zahn, Deakin, & Anderson, 2011; Rinck & Becker, 2005).

However, disentangling the contributions of Axis I and Axis II pathologies in neurocognitive functioning can be difficult (Fertuck et al., 2006a). In addition, some studies have shown that observed deficits in neurocognitive functioning among BPD patients do not appear to be different than those found in patients with MDD (Veiel, 1997; Zakzanis, Leach, & Kaplan, 1998), though findings in this area have been mixed. Impairments in visual information processing have been shown in MDD patients with comorbid BPD but not in uncomplicated MDD (Keilp et al., 2007). However, Beblo et al. (2011) noted that neuropsychological

performance on visual learning tasks, including one which included an emotional interference paradigm, did not distinguish BPD and MDD groups from one another. One factor which may help to explain this difference is that the visual information processing task used in Keilp et al.'s (2007) study was a backward masking paradigm, a design in which impulsivity was associated with poorer performance. This again points to the role of impulsivity in differentiating BPD participants from MDD participants in neurocognitive functioning.

In a landmark study, Fertuck et al. (2006b) noted the existence of mixed findings on neurocognitive impairments in BPD and sought to determine whether such impairments in BPD are primarily due to the influence of comorbid depression or borderline personality traits. They compared participants with BPD and comorbid MDD, uncomplicated MDD, and healthy controls on neuropsychological measures of attention, memory, working memory, intelligence, psychomotor speed, motor skill, and executive functions. Notably, all participants also completed mood ratings assessing anxiety, depression, confusion, vigor, fatigue, and anger. There were no differences between BPD patients with concurrent MDD and those with uncomplicated MDD across all seven domains of neurocognitive performance. Although there was a strong relationship between anxiety and neurocognitive performance in both patient groups, BPD-MDD participants reported greater past-week distress and had higher levels of aggression, impulsivity, anger, and anxiety. Interestingly, controlling for the effects of tension and anxiety in the sample actually revealed *superior* cognitive performance among BPD patients compared to those with uncomplicated MDD. The authors suggested that anxiety may have a larger impact on neuropsychological functioning in BPD than in MDD. Moreover, since intellectual functioning was measured in the study, they were able to rule out the possibility that the BPD-MDD group was simply more intelligent. Rather, they noted that "depression-like

deficits in BPD participants may be more closely related to characteristic mood fluctuations than to the effects of depression per se" (Fertuck et al., 2006b, p. 65).

**Summary of existing research**

Research examining neurocognitive functioning in BPD has struggled to yield consistent findings across domains and across samples. One explanation for this might be that few studies have accounted for the role of transient mood states and momentary distress in neurocognitive performance in BPD. In Fertuck et al.'s study (2006b), emotional distress had a differential impact on the neuropsychological performance of depressed participants with and without BPD, such that the BPD-MDD group showed superior performance when distress was held constant. This indicates that distress must be accounted for to obtain a meaningful estimate of neuropsychological functioning in BPD.

A growing number of studies, then, have examined cognitive disruption from experimentally-induced emotion dysregulation in patients with BPD, and some have examined stress responses to interpersonal triggers. However, none have examined cognitive performance in the context of negative evaluation fears and hypersensitivity to rejection. Given that vulnerability to intense emotional experiences and hypersensitivity to rejection are core features of BPD, it is likely that, compared to MDD participants and controls, participants with BPD would experience stronger emotional distress following a psychological stress procedure, and that this emotional response would be associated with intensified cognitive impairment.

In the current study, we sought to address gaps in the existing literature in two important ways. First, we evaluated performance on a working memory task administered in the context of a psychological stress procedure among BPD participants with or without comorbid MDD, MDD participants with no comorbid BPD, and healthy controls. The psychological stress procedure

required participants to give a short speech and perform the working memory task in front of two study confederates, who wore white laboratory coats and showed no affect nor provided any facial feedback. Given BPD individuals' heightened sensitivity to rejection, as well as their tendency to deplete cognitive resources by assessing neutral stimuli for potential threats or rejection cues, we expected that this stress paradigm would result in elevated levels of distress and derail their cognitive functioning to a greater extent than would the more impersonal stimuli that have been used in previous studies, such as words and images.

Second, as has been previously discussed, neurocognitive functioning in BPD may owe more to transient mood states and distress levels than to primary deficits per se. Accounting for the effects of distress may therefore be critical in allowing neurocognitive differences to emerge between BPD participants and MDD or control groups, as was found in Fertuck et al.'s study (2006b). Because participants in our psychological stress procedure underwent the working memory task *while under stress*, we could therefore account for the impact of stress on participants' cognitive performance.

**Aims and Hypotheses**

**Aim 1:** To examine working memory performance in the context of a psychological stress procedure.

***Hypothesis 1a:*** BPD participants will show significantly poorer working memory performance compared to MDDs and controls. A one-way ANOVA comparing BPD, MDD, and control participants on working memory performance will reveal significant differences across groups. Post-hoc tests will show that BPD participants' working memory performance was significantly poorer than MDDs' and controls'.

***Hypothesis 1b:*** Participants with MDD will show significantly better working memory performance compared to participants with BPD. Post-hoc tests will show that MDD participants' working memory performance was significantly better than BPDs'. Based on the existing literature (e.g., Keilp et al., 2010), it is difficult to predict how MDD participants will perform on the working memory task compared to controls.

***Hypothesis 1c:*** The impact of diagnosis on working memory performance will be mediated by emotional distress. When emotional distress is included in a regression of working memory performance onto diagnostic category, the association between working memory performance and diagnosis will weaken.

**Aim 2:** To examine the impact of emotional distress on working memory performance.

***Hypothesis 2a:*** Higher levels of emotional distress post-procedure will be associated with greater number of errors on the working memory task. Pearson correlation coefficients showing the strength of association between emotional distress and number of errors on the working memory task will be positive and significant.

***Hypothesis 2b:*** The relationship between emotional distress post-procedure and working memory performance will be stronger for BPD participants than for healthy controls. A z-test comparing Pearson correlation coefficients for working memory performance and emotional distress among BPD participants and healthy controls will be significant. In addition, diagnosis will moderate the relationship between emotional distress and working memory performance, such that emotional distress will be shown to have a larger impact on working memory for BPD participants than for controls.

# CHAPTER II:

# METHOD

## Participants

This study was a secondary data analysis. Study participants were a subset ($n$ = 118) of a larger study conducted at New York State Psychiatric Institute (PI: Barbara Stanley, Ph.D.). This parent study was a prospective, naturalistic examination of biological and clinical predictors of suicidal behavior. The parent sample was comprised of 100 participants with BPD (50 suicide attempters and 50 non-attempters), a psychiatric control group of 100 participants with MDD and no Axis II disorder (50 suicide attempters and 50 non- attempters), and 50 healthy controls with no current or past Axis I or Axis II diagnoses. Male and female clinical participants were eligible to participate in the parent study if they had a diagnosis of either MDD or BPD, were between the ages of 18 and 55, and were able and willing to provide informed consent. Clinical participants were excluded from participation if they exhibited organic mental syndromes, current psychoactive substance dependence, or mental retardation; schizophrenia, schizoaffective disorder, delusional disorder, or any other psychotic disorder; or history of vasovagal syncope, heart disease, or myocardial infarction. Healthy controls were eligible to participate in the parent study if they were between the ages of 18 and 55, had no current or past Axis I or Axis II disorders, had no alcohol or substance use disorder within the last three months, did not take any medications, and were able and willing to provide informed consent.

Participants whose data were eligible for analysis in the current study were males and females between the ages of 18 and 55 who did not meet any of the above exclusion criteria. For inclusion in the current study, participants also had to have participated in psychological stress test procedures at baseline (described in "Procedure"). Participants with a primary diagnosis of

BPD ($n$ = 60) were compared to patients with MDD ($n$ =30) as a psychiatric control group and to healthy controls ($n$ = 21). Modifications to the sample used for analysis are described in "Statistical Analysis."

**Measures**

**Working memory.** Working memory was assessed using the PASAT-C. The PASAT was originally designed as a neuropsychological assessment of auditory processing speed, flexibility, and general working memory in patients with traumatic brain injury (Gronwall, 1977). The task has been used to study the impact of various conditions on cognitive functioning, such as multiple sclerosis (Benedict et al., 2002), lupus (Shucard et al, 2004), and chronic fatigue syndrome (Tombaugh, 2006). The PASAT involves the rapid presentation of a series of single-digit numbers, in which the two most recent digits must be summed. For example, if the digits '2', '8', and '5' were presented, the correct sums would be '10' and then '13.' It is noteworthy that participants must ignore the first sum to recall the second digit in the sequence and add it to the next one, thereby contributing to the task's difficulty. Participants must provide the correct sum before the next digit in the series is presented in order for the response to be scored.

Although digits were presented auditorily in the original version of the task, many recent studies have used visually presented digits on computer screens (e.g., Fos, Greve, South, Mathias, & Benefield, 2000; Royan, Tombaugh, Rees, & Francis, 2004; Tombaugh, Rees, Baird, & Kost, 2004). While some studies have employed consistent latency intervals between digits, most have used increasingly rapid inter-stimulus intervals (ISIs; e.g., Tombaugh et al., 2004). Tombaugh's (2006) comparison of these studies indicated that the most rapid ISIs are less sensitive to the detection of between-group differences.

Factor analytic studies have indicated that the PASAT measures components of working memory. Specifically, the task has been shown to measure attention switching (Bate, Mathias, & Crawford, 2001), divided attention (van Zomeran & Brouwer, 1994), mental tracking (Lezak, 1995), concentration (Gronwall & Wrightson, 1981), speed of information processing (Haslam, Batchelor, Fearnside, Haslam, & Hawkins, 1995), immediate memory (Larrabee & Curtiss, 1995), and a latent factor termed Freedom from Distractibility (Crawford et al., 1998; Deary et al, 1991; Sherman et al., 1995). More recently, researchers have suggested that successful performance on the PASAT draws on several simultaneous cognitive abilities, most notably sustained attention and speed of information processing (Cicerone, 1997; Madigan et al., 2000). Studies have demonstrated moderate to high correlations between the PASAT and other measures of working memory components, particularly attention; these include Digit Span ($r$ = .27 to .49; Fisk & Archibald, 2001), Trails ($r$ = .58; O'Donnell, MacGregor, Dabrowski, Oestreicher, & Romero, 1994), and Arithmetic ($r$ = .63; Crawford et al., 1998). The PASAT has been shown to possess a high degree of internal consistency, with Cronbach's alphas ranging from .76 to .95 (MacLeod & Prior, 1996; Ponsford & Kinsella, 1992; Sherman et al., 1997); test-retest coefficients have ranged from .90 to .97 (McCaffrey, Westervelt, & Haase, 2001; Stuss et al., 1989).

Researchers have long encountered difficulties administering the PASAT, particularly in clinical populations (Iverson, Lovell, & Smith, 2000), due to the task's high level of difficulty and aversive nature (e.g., Diehr et al, 2003). Participants have reported elevated levels of anxiety (Roman, Edwall, Buchanan, & Patton, 1991), sadness (Holdwick & Wingenfeld, 1999), and stress (Stuss, Stethem, Hugenholtz, & Richard, 1989) after performing the task. Accordingly, the PASAT was adapted by Lejuez, Kahler, and Brown (2003) for use as an experimental induction

of moderate psychological stress rather than a neuropsychological assessment. Lejuez et al. (2003) adapted the PASAT for use as a stress induction tool by giving unpleasant auditory feedback when incorrect answers were given, and by increasing precision through computerized measurements of inter-stimulus interval times and participant errors. In their pilot study of this modified, computerized version of the task (PASAT-C), it was shown to induce self-reported anxiety, difficulty concentrating, and irritability (Lejuez et al., 2003).

Researchers have adapted the PASAT-C further (Keilp et al, 2010) to incorporate it into the Trier Social Stress Test (TSST; Kirschbaum, Pirke, & Hellhammer, 1993), a standardized social stress procedure which in turn has been modified for use in clinical populations and was used in the parent study (described in "Procedure"). Participants in the parent study were administered the PASAT-C for the purposes of inducing psychological stress. However, given the long history and well-validated use of the PASAT as a neuropsychological measure, participants' actual performance (measured by total number of errors) on the PASAT-C was used as the dependent variable in the current study to examine the ability to sustain working memory abilities while under stress. The total number of errors was summed from errors made on each of four trials. Digits were presented with ISIs of 2.6 seconds in Trial 1, 2.4 seconds in Trial 2, 2.0 seconds in Trial 3, and 1.6 seconds in Trial 4. Change scores (Trial 1 errors subtracted from Trial 4 errors) were calculated to estimate deterioration of performance from Trial 1 to Trial 4.

**Distress.** The Profile of Mood States (POMS; McNair, Lorr, & Droppleman, 1981) was used to assess subjective mood and levels of emotional distress at three time-points: 10 minutes before (pre-TSST), immediately following (post-TSST), and 20 minutes after (follow-up) the 15-minute psychological stress procedure. The POMS is a 65-item self-report measure which assesses subjective experiences across six domains (tension/anxiety, depression/dejection,

anger/hostility, confusion/bewilderment, fatigue/inertia, and vigor/activity). All subscales except vigor are negatively scaled, with higher scores indicating greater distress. Participants are presented with a list of 65 words that describe various feelings and are asked to indicate how well the words describe how they have been feeling during the past week, including today. Items are scored on a 5-point Likert scale, ranging from 0 ("Not at all") to 4 ("Extremely"). Sample words include "angry", "peeved", and "grouchy" for the anger subscale; "confused" and "unable to concentrate" for confusion; "unhappy", "sorry for things done", and "sad" for depression; "worn out", "listless", and "fatigued" for fatigue; "tense", "shaky", and "on edge" for tension; and "lively", "active", and "energetic" for vigor. A total distress score is obtained by summing scores from the tension, depression, anger, confusion, and fatigue scales, and then subtracting scores on the vigor scale. The POMS has been shown to possess good internal consistency (Cronbach's alpha of .78 to .92; Morfield, Petersen, Kruger-Bodeker, von Mackensen, & Bullinger, 2007).

**Participant experience.** Participants' experiences of the TSST were measured by visual analog scales (VAS) administered immediately following the completion of the psychological stress test (post-TSST). Participants were asked to rate their experience of the psychological stress procedure by indicating on a horizontal continuum their level of stress and concern during the task, as well as perceived difficulty of the task and their level of involvement in the task. VAS scores are participants' marks on the scales, which are measured in millimeters from 0 to 100.

**Diagnosis.** Structured diagnostic interviews, along with a battery of clinical assessments, were administered over two to three baseline visits prior to beginning study procedures. Axis I diagnoses were ascertained using the Structured Clinical Interview for DSM-IV Axis I Disorders

(SCID-I; First, Gibbon, Spitzer, & Williams, 1996), a clinician-administered instrument designed for differential diagnosis of DSM-IV psychiatric disorders. The SCID-I has been shown to possess good interrater reliability, with mean Cohen's kappas ranging from 0.57 (Zanarini et al., 2000) to 0.71 (Lobbestael, Leurgans, & Arntz, 2011). Axis II diagnoses were ascertained using the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II; First, Gibbon, Spitzer, Williams, & Benjamin, 1997). The SCID-II is a clinician-administered instrument which has been shown to possess good interrater reliability (.48 to .98) and satisfactory internal consistency coefficients, or ICCs (.71 to .94; Maffei et al., 1997).

In the parent and current study, BPD participants were classified as having an Axis II diagnosis of borderline personality disorder, regardless of the presence or absence of current or lifetime major depressive disorder. MDD participants were classified as having a current or lifetime diagnosis of major depressive disorder, and *no* diagnosis of BPD. Healthy controls were classified as having no current or lifetime Axis I or Axis II diagnosis.

**Clinical symptoms.** Clinical symptoms were included for analysis to compare and fully characterize the BPD and MDD participant groups, but were not included in primary analyses of working memory. Clinical measures assessed global psychosocial functioning using the Global Assessment Scale (GAS; American Psychiatric Association, 2000); symptoms of depression using the Hamilton Depression Rating Scale (HAM-D; Hamilton, 1960) and Beck Depression Inventory (BDI-II; Beck, Steer, & Brown, 1996); anxiety, using the Hamilton Anxiety Rating Scale (HAM-A; Hamilton, 1959) and State-Trait Anxiety Inventory, State Version (STAI; Spielberger, 1983); aggression, using the Brown-Goodwin Lifetime History of Aggression (BGHA; Brown, Goodwin, Ballenger, Goyer, & Major, 1979); hopelessness, using the Beck Hopelessness Scale (BHS; Beck, Weissman, Lester, & Trexler, 1974); general psychiatric

(particularly psychotic) symptoms using the Brief Psychiatric Rating Scale (BPRS; Overall & Gorham, 1962); and impulsivity, using the Barratt Impulsivity Scale (BIS; Patton, & Stanford, & Barratt, 1995).

**Procedure**

Patients were referred to the parent study by referrals from physicians, emergency rooms, and state hospitals, and through advertising across the New York City area by direct mailings, in-service presentations, public affairs programming, and phone contact with local psychiatric facilities. Interested patients were informed about the study using a standardized telephone script. Participants received initial screenings for eligibility and underwent informed consent procedures. Baseline clinical assessments were conducted prior to the psychological stress procedure, at the same or a previous visit. The parent study was entitled "Prospective Study of Predictors of Suicidal Behavior in Borderline Personality Disorder (BPD)—Biological Measures" and was approved by the NYSPI-Columbia University Department of Psychiatry Institutional Review Board (IRB). Given that the current study was a secondary data analysis and involved no new recruitment of subjects, it was considered exempt from committee review by the Teachers College IRB.

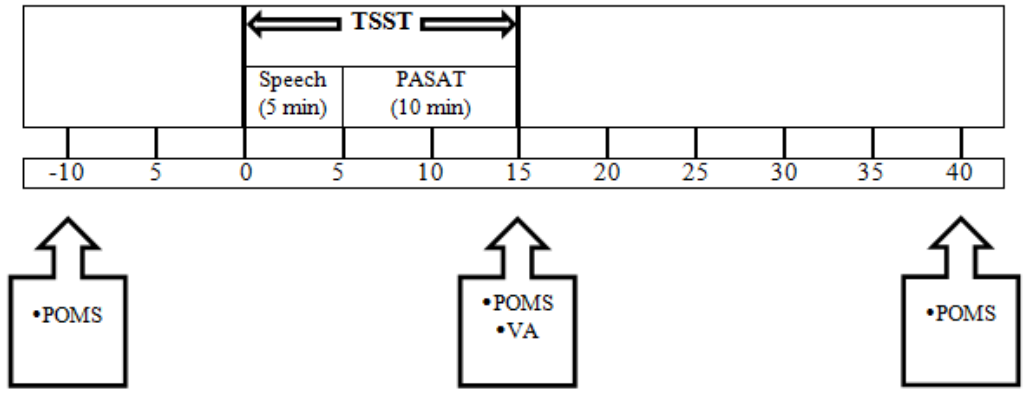
The psychological stress procedure used in the parent study was a version of the TSST, a tool which has been used extensively to study biobehavioral responses to stress in a laboratory setting. This task has also been modified for high-risk populations (Keilp et al., 2010). Traditionally, the TSST consists of a 10-minute anticipation period and a 10-minute test period. During the test period, participants are asked to stand in front of a microphone and deliver a persuasive five-minute speech as a job applicant, followed by performing rapid mental arithmetic (such as subtracting serial sevens) in front of an audience of study confederates. The TSST has

been shown to reliably induce psychological stress (Nater et al., 2010; Simeon, Knutelska, Smith, Baker, & Hollander, 2007; Takahashi et al., 2004). Studies have suggested that the TSST induces psychological stress primarily by elevating participant concerns about social evaluation and by exposing participants to a task that is uncontrollable (Dickerson & Kemeny, 2004; Gruenewald, Kemeny, Aziz, & Fahey, 2004).

The version of the TSST which was modified for clinical populations and used in the parent study consisted of a five-minute speaking exercise and a 10-minute working memory task (PASAT-C), both of which were administered in the presence of two study confederates clad in white laboratory coats who displayed neutral affect. This was intended to induce socially evaluative stress. The TSST was administered at the same time of day (the afternoon) for all participants to minimize the effects of normal diurnal fluctuations in cortisol levels and heart rate. Ten minutes before the beginning of the stress procedure, heart rate monitors were placed on participants' wrists and participants completed mood (POMS) ratings; they also received training in the PASAT-C task. One minute before the procedure, participants were asked to stand and were informed that they would remain standing for the duration of the task. In the first part of the stress procedure, participants were asked to speak about themselves extemporaneously for five minutes. In the second half of the stress procedure, participants were administered the PASAT-C working memory task. In this version of the PASAT-C, participants stood in front of a laptop computer. Series of single digits were presented visually on the screen and participants were to mentally sum the two most recent digits, and speak their answers aloud. The PASAT-C was administered four times, with the ISI becoming shorter in each trial (2.6, 2.4, 2.0, and 1.6 seconds, respectively). Participants' spoken responses were recorded on clipboards by an examiner and a study confederate. Both the examiner and the confederate scored the PASAT.

Immediately following the TSST, participants completed distress ratings (POMS) and visual analog ratings of their perceived involvement in, difficulty of, concern about, and stressfulness of the TSST. They repeated POMS ratings 25 minutes later. If participants reported feeling distressed by the procedure, they were given the opportunity to speak with a licensed mental health professional on staff. A timeline of study procedures is presented in Figure 1.

Figure 1. Timeline of study procedures (in minutes)




*Note.* TSST – Trier Social Stress Task. PASAT – Paced Auditory Serial Addition Task. POMS – Profile of Mood States. VA – Visual analog scales.

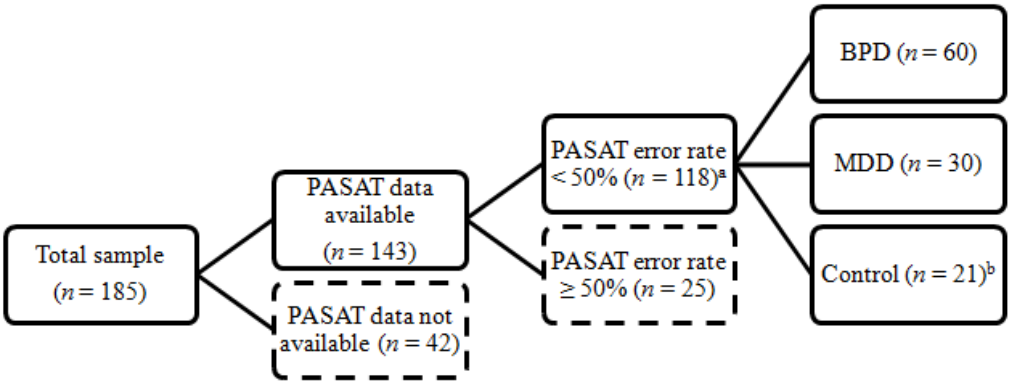
Clinical assessments and study procedures were performed either by licensed psychologists or trained research assistants. All interviewers are required to have completed ethics training and have had experience working with clinical populations. Clinical interviewers who did not have a doctorate degree were supervised closely by a licensed psychologist.

**Statistical Analysis**

All statistical analyses were performed using SPSS (version 17.0). Data was available for 185 individuals who participated in the parent study. Cases were then eliminated ($n$ = 25) if they demonstrated an error rate of greater than 50% (≥ 124 errors) in total PASAT scores, as this would indicate that they were no longer attending to the task and providing reliable data. Clinical characteristics of participants who prematurely ended the task were explored. Participants who ended the task prematurely had fewer years of education than those who completed the task ($F(1,131) = 13.02$, $p < .001$). They also showed higher baseline levels of clinician-rated anxiety ($F(1,127) = 4.08$, $p = .045$), were less involved in the task ($F(1,141) = 14.13$, $p < .001$), and perceived the task as being more difficult ($F(1,141) = 5.17$, $p = .025$) than those who completed the task. PASAT completers were no more likely than non-completers to have a BPD diagnosis ($\chi^2(2) = 1.712$, $p = .425$).

Forty-two participants had missing PASAT data on all four trials and were also removed from the sample. This resulted in a final sample of 118 participants, of whom 60 had a diagnosis of BPD, 30 had a diagnosis of MDD, and 21 were healthy controls. There were an additional seven participants whose diagnostic information was not available. Data elimination procedures are presented in Figure 2.

Figure 2. Data elimination procedures for the study sample




*Note*. [a] Cases were excluded from analysis if they had error rates of greater than 50% (≥ 124 errors) in total PASAT scores, as this would indicate that they were no longer attending to the task and providing reliable data. Thus, the highest possible PASAT score in this sample was 123. [b] Seven participants had missing diagnostic information.

The sample used for analysis in the current study ($n$ = 118) was evaluated for normality using SPSS Regression and Explore functions. Hypotheses were examined using a combination of Pearson correlations, chi-squares, one-way ANOVAs, and multiple regression analyses. To test the main hypothesis that the impact of emotional distress on working memory is moderated by diagnosis, several multiple regression analyses were used (described in "Results"). Assumptions of linearity, independence of residuals, and homoscedasticity in the multiple regression analyses were not violated. To avoid potential problems of multicollinearity with the interaction term, mean-subtracted (centered) continuous predictor variables were used (Aiken & West, 1991) and multiplicative interaction terms were created.

Effects coding was used to create two contrast variables for the three diagnostic groups (BPD, MDD, and control) in order to test for between-group differences. The first contrast variable (BPD = 1, MDD = 0, control = -1) compared BPD participants and controls to the grand mean of the sample. The second contrast variable (BPD = 1, MDD = -1, control = 0) compared BPD participants and MDD participants to the grand mean of the sample. Probabilities reported are two-tailed and significance was established at $p < .05$. Trends were reported at $p < .10$.

# CHAPTER III:

# RESULTS

**Descriptive statistics.** The mean age of the sample was 32.66 years ($SD$ = 10.49) with a range from ages 18 to 57. Participants in the sample had 15.20 years of education on average ($SD$ = 2.38). Seventy-eight percent of the sample was female. Approximately 45% of the sample was employed. There were slight differences in race across diagnostic groups ($\chi 2$ (10) = 17.135, $p$ = .071).; z-tests examining column proportions revealed that there was a slightly higher prevalence of black participants and lower prevalence of white participants in the MDD group than in the

BPD group. MDD participants had slightly fewer years of education than BPD participants or controls ($F(2,101) = 3.05$, $p = .052$). Among categories of marital status, the BPD participants were slightly more likely to be married, while MDD participants were slightly more likely to be separated or widowed ($\chi 2\ (8) = 13.491$, $p = .096$). However, there were no significant differences on any demographic variables across diagnostic groups. These data are presented in Table 1.

Table 1. Demographic Characteristics

| | BPD (*n* = 60) | MDD (*n* = 30) | Control (*n* = 21) | Total (*n* = 111) | ANOVA or Pearson Chi-Square | |
|---|---|---|---|---|---|---|
| | *M (SD)* or *n* (%) | *M (SD)* or *n* (%) | *M (SD)* or *n* (%) | *M (SD)* or *n* (%) | Test | *p* |
| Age | 32.22 (9.66) | 33.00 (10.03) | 32.67 (11.87) | 32.66 (10.49) | $F(2,106) = 0.060$ | .942 |
| Sex | | | | | | |
| Male | 8 (13.3%) | 8 (28.6%) | 6 (28.6%) | 22 (18.6%) | $\chi 2\ (2) = 3.888$ | .143 |
| Female | 52 (86.7%) | 20 (71.4%) | 15 (71.4%) | 92 (78.0%) | | |
| Race/ Ethnicity | | | | | | |
| Asian | 3 (5.0%) | 1 (3.6%) | 2 (9.5%) | 6 (5.5%) | | |
| Black | 5 (8.3%) | 8 (28.6%) | 5 (23.8%) | 18 (16.5%) | | |
| White | 41 (68.3%) | 10 (35.7%) | 13 (61.9%) | 64 (58.7%) | $\chi 2\ (10) = 17.135$ | .071 |
| Hispanic | 6 (10.0%) | 3 (10.7%) | 0 (0.0%) | 9 (8.3%) | | |
| Multiracial | 1 (1.7%) | 2 (7.1%) | 1 (4.8%) | 4 (3.7%) | | |
| Unknown | 4 (6.7%) | 4 (14.3%) | 0 (0.0%) | 8 (7.3%) | | |
| Years of education | 15.25 (2.42) | 14.46 (2.42) | 16.10 (1.45) | 15.20 (2.38) | $F(2,101) = 3.049$ | .052 |
| Marital status | | | | | | |
| Single | 39 (65.0%) | 21 (75.0%) | 19 (90.5%) | 82 (69.5%) | | |
| Married or conjugal living | 12 (20.0%) | 1 (3.6%) | 1 (4.8%) | 14 (11.9%) | $\chi 2\ (8) = 13.491$ | .096 |
| Separated | 1 (1.7%) | 2 (7.1%) | 0 (0%) | 4 (3.4%) | | |
| Divorced | 8 (13.3%) | 3 (10.7%) | 1 (4.8%) | 12 (10.2%) | | |
| Widowed | 0 (0%) | 1 (3.6%) | 0 (0%) | 1 (0.8%) | | |
| Currently employed | | | | | | |
| Yes | 24 (40.7%) | 13 (48.1%) | 14 (66.7%) | 53 (44.9%) | $\chi 2\ (2) = 4.197$ | .123 |
| No | 35 (59.3%) | 14 (51.9%) | 7 (33.3%) | 59 (40.0%)[a] | | |

*Note.* * $p < .05$. ** $p < .01$. *** $p < .001$. [a] 17 of the 59 participants (28.8%) classified as unemployed were students.

**Clinical characteristics.** To fully characterize our sample, chi-squares and one-way ANOVAs were used to analyze differences across diagnostic groups on baseline clinical measures collected in the parent study but not incorporated for further analysis in the present study. Significant differences were observed in participants' number of comorbid diagnoses across the two groups ($\chi 2$ (3) = 9.045, $p$ = .029), with BPD participants being more likely to carry three or more Axis I diagnoses. BPD participants were also more likely to have had a history of self-injurious behavior ($\chi 2$ (1) = 7.236, $p$ = .007) and they demonstrated lower GAS scores ($F$(1,84) = 4.99, $p$ = 0.28). In addition, compared to MDD participants, BPD participants had higher scores on depression measures; however, only scores on the clinician-rated measure of depression ($F$(1,86) = 4.360, $p$ = .040) were significant; self-reported depression ($F$(1,84) = 2.412, $p$ = .124) was not. BPD participants also showed higher levels of anxiety compared to MDD participants. In contrast to the depression findings, however, only self-reported anxiety ($F$(1,83) = 10.688, $p$ = .002) was significant, while clinician-rated anxiety ($F$(1,78) = 1.806, $p$ = .183) was not. BPD participants showed higher levels of aggression ($F$(1,77) = 6.636, $p$ = .012), greater overall psychiatric symptoms ($F$(1,71) = 5.307, $p$ = .024), and higher levels of impulsivity ($F$ (1,82) = 14.119, $p$ < .001). There were no other significant differences on clinical measures across diagnostic groups. These data are presented in Table 2. The prevalence of comorbid psychiatric diagnoses in the two clinical groups is presented in Table 3.

Table 2. Clinical Characteristics

| | BPD ($n$ = 60) | MDD ($n$ = 30) | | ANOVA/Pearson Chi-Square | |
|---|---|---|---|---|---|
| | *M (SD)* or *n* (%) | *M (SD)* or *n* (%) | Potential Range | Test | *p* |
| Number of comorbid Axis I disorders | | | | | |
| None (0) | 2 (3.3%) | 1 (3.4%) | | | |
| One (1) | 17 (28.3%) | 14 (48.3%) | | $\chi 2$ (3) = 9.045 | .029*[a] |
| Two (2) | 27 (45.0%) | 14 (48.3%) | | | |
| Three or more (≥ 3) | 14 (23.3%) | 0 (0%) | | | |
| Age of first hospitalization | 23.64 (7.73) | 18.75 (6.07) | | $F$ (1,34) = 2.706 | .109 |
| Number of previous hospitalizations | 4.07 (5.41) | 3.00 (1.85) | | $F$ (1,34) = 0.298 | .588 |
| Total cumulative months in hospital | 2.76 (4.78) | 4.28 (14.88) | | $F$ (1,47) = 0.290 | .593 |
| Physical abuse | | | | | |
| Yes | 20 (33.3%) | 8 (27.6%) | | $\chi 2$ (1) = 0.299 | .584 |
| Sexual abuse | | | | | |
| Yes | 25 (41.7%) | 11 (37.9%) | | $\chi 2$ (1) = 0.113 | .736 |
| Lethality of suicide attempts | 2.45 (1.06) | 3.10 (1.73) | | $F$ (1,37) = 2.012 | .164 |
| History of self-injurious behavior | | | | | |
| Yes | 28 (65.1%) | 7 (30.4%) | | $\chi 2$ (1) = 7.236 | .007** |
| Functioning (GAS) | 57.84 (8.71) | 62.03 (7.45) | 1-100[b] | $F$ (1,84) = 4.99 | .028* |
| Depression (HAM-D 24) | 15.19 (7.59) | 11.60 (7.74) | 0-74 | $F$ (1,86) = 4.360 | .040* |
| Depression (BDI-II) | 18.52 (9.51) | 14.93 (11.40) | 0-63 | $F$ (1,84) = 2.412 | .124 |
| Anxiety (HAM-A) | 10.36 (6.09) | 8.50 (5.824) | 0-56 | $F$ (1,78) = 1.806 | .183 |
| Anxiety (STAI) | 51.68 (15.59) | 40.69 (12.74) | 20-80 | $F$ (1,83) = 10.688 | .002** |
| Aggression (BGHA) | 21.10 (5.54) | 17.73 (5.81) | 11-44 | $F$ (1,77) = 6.636 | .012* |
| Hopelessness (BHS) | 9.20 (5.72) | 7.41 (5.32) | 0-20 | $F$ (1,80) = 1.860 | .176 |
| Psychiatric symptoms (BPRS) | 28.98 (5.58) | 25.97 (5.36) | 18-126 | $F$ (1,71) = 5.307 | .024* |
| Impulsivity (BIS) | 63.04 (18.49) | 48.33 (14.50) | 30-120 | $F$ (1,82) = 14.119 | <.001*** |

*Note.* GAS – Global Assessment Scale; HAM-D 24 – Hamilton Depression Rating Scale, 24 Item; BDI-II – Beck Depression Inventory; HAM-A – Hamilton Anxiety Rating Scale; STAI – State-Trait Anxiety Inventory (State Form); BGHA – Brown-Goodwin Assessment for Lifetime History of Aggression; BHS – Beck Hopelessness Scale; BIS – Barratt Impulsiveness Scale.

[a]Bonferroni-corrected contrast: BPD > MDD, three or more Axis I disorders. [b] Lower scores indicate poorer functioning.

* $p$ < .05. ** $p$ < .01. *** $p$ < .001.

Table 3. Prevalence of comorbid psychiatric disorders among clinical participants

| Comorbid psychiatric diagnosis | BPD ($n$ = 60) | | MDD ($n$ = 30) | | Total ($n$ = 90) | |
|---|---|---|---|---|---|---|
| | Current[a] | Lifetime | Current | Lifetime | Current | Lifetime |
| Mood disorder | | | | | | |
| Major depression | 30 (50.0%) | 27 (45.0%) | 11 (36.7%) | 26 (86.7%) | 41 (45.6%) | 53 (58.9%) |
| Dysthymia | 5 (8.3%) | 4 (6.6%) | 2 (6.7%) | 2 (6.7%) | 7 (7.8%) | 6 (6.7%) |
| Bipolar disorder | 3 (5.0%) | -- | -- | -- | 3 (3.3%) | -- |
| Other bipolar disorder | 1 (1.7%) | 3 (5.0%) | -- | -- | 1 (0.8%) | 3 (2.5%) |
| Mood disorder in remission | 11 (18.3%) | -- | 16 (53.3%) | -- | 27 (30.0%) | -- |
| Anxiety disorder | | | | | | |
| Panic | 3 (5.0%) | 2 (3.3%) | 2 (6.7%) | 2 (6.7%) | 5 (4.2%) | 4 (4.4%) |
| Agoraphobia | 3 (5.0%) | -- | -- | -- | 3 (2.5%) | -- |
| Social phobia | 2 (3.3%) | -- | -- | -- | 2 (1.7%) | -- |
| Simple phobia | 4 (6.7%) | 3 (5.0%) | 1 (3.3%) | 2 (6.7%) | 5 (4.2%) | 5 (4.2%) |
| OCD | 6 (10.0%) | 1 (1.7%) | -- | -- | 6 (5.1%) | 1 (0.8%) |
| GAD | 3 (5.0%) | 1 (1.7%) | -- | -- | 3 (2.5%) | 1 (0.8%) |
| PTSD | 3 (5.0%) | 8 (13.3%) | 2 (6.7%) | 3 (10.0%) | 5 (4.2%) | 11 (12.1%) |
| Eating disorder | | | | | | |
| Bulimia nervosa | 1 (1.7%) | 3 (5.0%) | -- | 1 (3.3%) | -- | -- |
| Binge eating disorder | -- | 1 (1.7%) | 1 (3.3%) | 1 (3.3%) | 1 (0.8%) | 2 (1.7%) |
| Personality disorder | | | | | | |
| Borderline | 60 (100%) | | -- | | 60 (66.7%) | |
| Paranoid | 7 (11.7%) | | 2 (6.7%) | | 9 (9.9%) | |
| Schizotypal | 5 (8.3%) | | -- | | 5 (5.5%) | |
| Schizoid | -- | | 1 (3.3%) | | 1 (0.8%) | |
| OCPD | 6 (10.0%) | | 4 (13.3%) | | 10 (11.0%) | |
| Avoidant | 9 (15.0%) | | 4 (13.3%) | | 13 (14.3%) | |
| Antisocial | -- | | 1 (3.3%) | | 1 (0.8%) | |

*Note.* Percentage sums less than 100% were due to missing data. Percentage sums greater than 100% were due to the presence of multiple diagnoses in individual participants.

**Preliminary analyses.** Psychometric properties of independent and dependent variable measures are presented in Table 4. This table design was adapted from Shoum (2011). Values for kurtosis (-0.83, *SE* = 0.44) and skewness (0.09, *SE* = 0.22) in the distribution of the dependent variable (PASAT errors) in the total sample were converted to *z*-scores by dividing by their standard errors in order to examine their statistical significance. Absolute values of both z-scores fell below the 95% critical value of 1.96, suggesting that skewness and kurtosis in PASAT errors did not deviate significantly from the normal curve. Although skewness and kurtosis were high for PASAT Trial 1, this suggests that most participants performed fairly well on the easiest trial of the task, which is consistent with the test's intended design. Closer examination of total PASAT errors by diagnostic group using the Kolmogorov-Smirnov test revealed normal distributions for BPD participants ($D(60) = 0.89$, $p = .20$), MDD participants ($D(30) = 0.064$, $p = .20$) and controls ($D(21) = 0.103$, $p = .20$). Levene's test revealed homogeneous variances in PASAT errors across diagnostic groups ($F(2,108) = 0.92$, $p = .402$). Frequencies, histograms, and boxplots revealed no significant outliers in PASAT errors across diagnostic groups.

Table 4. Psychometric properties of working memory and emotional distress measures

| | | | | | | Range | | Skewness | | Kurtosis | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | *n* | *M* | *SE* | *SD* | α | Potential | Actual | Statistic | SE | Statistic | SE |
| Working memory (PASAT) | | | | | | | | | | | |
| Trial 1 (2.6 s) | 118 | 8.37 | 0.78 | 8.50 | | 0-60 | 0-49 | 1.94 | 0.22 | 5.04 | 0.44 |
| Trial 2 (2.4 s) | 118 | 10.00 | 0.66 | 7.18 | | 0-60 | 0-35 | 0.66 | 0.22 | 0.08 | 0.44 |
| Trial 3(2.0 s) | 118 | 14.89 | 0.84 | 9.12 | .87 | 0-60 | 0-34 | 0.10 | 0.22 | -1.10 | 0.44 |
| Trial 4 (1.6 s) | 118 | 21.86 | 1.00 | 10.87 | | 0-60 | 0-49 | -0.23 | 0.22 | -0.63 | 0.44 |
| Total errors | 118 | 55.12 | 2.80 | 30.44 | | 0-240[a] | 2-123 | 0.09 | 0.22 | -0.83 | 0.44 |
| Emotional distress (POMS) | | | | | | | | | | | |
| Pre-TSST | 106 | 26.30 | 3.96 | 40.80 | .94 | -32-228 | -30-178 | 1.16 | 0.24 | 1.26 | 0.47 |
| Post-TSST | 110 | 36.27 | 4.25 | 44.55 | .95 | -32-228 | -31-186 | 1.01 | 0.24 | 0.81 | 0.46 |
| Follow-up | 109 | 12.93 | 3.02 | 31.50 | .94 | -32-228 | -32-119 | 1.03 | 0.23 | 0.77 | 0.46 |
| Visual analog scales | | | | | | | | | | | |
| Involvement | 118 | 81.33 | 1.70 | 18.50 | | 0-100 | 15-100 | -1.64 | 0.23 | 2.67 | 0.44 |
| Stress | 118 | 65.85 | 2.37 | 26.03 | .62 | 0-100 | 2-100 | -0.73 | 0.22 | -0.48 | 0.44 |
| Concern | 118 | 62.51 | 2.92 | 31.75 | | 0-100 | 0-100 | -0.62 | 0.22 | -1.01 | 0.44 |
| Difficulty | 118 | 63.08 | 2.26 | 24.54 | | 0-100 | 2-100 | -0.68 | 0.22 | -0.27 | 0.44 |

*Note*. PASAT – Paced Auditory Serial Addition Task; POMS – Profile of Mood States; α = Cronbach's alpha.

[a] Cases were excluded from analysis if they had error rates of greater than 50% (≥ 124 errors) in total PASAT scores, as this would indicate that they were no longer attending to the task and providing reliable data. Thus, the highest possible PASAT score in this sample was 123.

Analyses of skewness revealed positive skew for the POMS measures, indicating that scores clustered on the lower end of the distribution, and negative skew for the visual analog scales, indicating that scores clustered on the higher end of the distribution. Skewness presents greater difficulty when tests are norm-referenced, and may even be desirable when the test references a criterion rather than a norm (Brown, 1996). In this case, significant negative skewness for participants' involvement in the TSST indicates a relatively high degree of involvement in the task, lending greater validity to the working memory data obtained. Similarly, negative skewness for participants' stress, concern, and perceived difficulty of the TSST confirms that the task induced a moderate degree of psychological stress as intended, thereby serving as a manipulation check.

Reliability analyses of dependent and independent measures revealed a high degree of internal consistency for working memory (Cronbach's $\alpha = .87$), pre-TSST distress (Cronbach's $\alpha = .94$), post-TSST distress (Cronbach's $\alpha = .95$), and distress at follow-up (Cronbach's $\alpha = .94$), and an acceptable degree of internal consistency for visual analog scales (Cronbach's $\alpha = .62$). Pearson correlations between working memory and emotional distress pre-TSST, post-TSST, and at follow-up are presented in Tables 5, 6, and 7, respectively.

Table 5. Pearson correlations between working memory and pre-TSST emotional distress

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. PASAT 1 (2.6 s) | -- | | | | | | | | | | | |
| 2. PASAT 2 (2.4 s) | .62*** | -- | | | | | | | | | | |
| 3. PASAT 3 (2.0 s) | .51*** | .71*** | -- | | | | | | | | | |
| 4. PASAT 4 (1.6 s) | .48*** | .67*** | .80*** | -- | | | | | | | | |
| 5. PASAT (Total) | .75*** | .86*** | .90*** | .89*** | -- | | | | | | | |
| 6. POMS (Ang) | .02 | -.03 | -.07 | -.03 | -.03 | -- | | | | | | |
| 7. POMS (Conf) | .15 | .10 | .09 | .12 | .13 | .60*** | -- | | | | | |
| 8. POMS (Dep) | .07 | .01 | -.04 | .12 | .02 | .67*** | .80*** | -- | | | | |
| 9. POMS (Fat) | .02 | .05 | .03 | .04 | .04 | .53*** | .76*** | .75*** | -- | | | |
| 10. POMS (Ten) | .02 | .01 | .01 | .02 | .02 | .53*** | .70*** | .66*** | .57*** | -- | | |
| 11. POMS (Vig) | -.19* | -.28** | -.27** | -.23* | -.28** | -.07 | -.40*** | -.30*** | -.37*** | -.30** | -- | |
| 12. POMS (Total) | .06 | .04 | .01 | .05 | .05 | .77*** | .88*** | .92*** | .84*** | .79*** | -.46*** | -- |

*Note.* PASAT – Paced Auditory Serial Addition Task; POMS – Profile of Mood States; POMS (Ang) – Anger subscale; POMS (Conf) – Confusion subscale; POMS (Dep) – Depression subscale; POMS (Fat) – Fatigue subscale; POMS (Ten) – Tension/anxiety subscale; POMS (Vig) – Vigor subscale (reverse scored); VA-I – Visual Analog, Involvement; VA-S – Visual Analog, Stress; VA-C – Visual Analog, Concern; VA-D – Visual Analog, Difficulty.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 6. Pearson correlations between working memory and post-TSST emotional distress

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | 13 | 14 | 15 | 16 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. PASAT 1 (2.6 s) | -- | | | | | | | | | | | | | | | |
| 2. PASAT 2 (2.4 s) | .62*** | -- | | | | | | | | | | | | | | |
| 3. PASAT 3 (2.0 s) | .51*** | .71*** | -- | | | | | | | | | | | | | |
| 4. PASAT 4 (1.6 s) | .48*** | .67*** | .80*** | -- | | | | | | | | | | | | |
| 5. PASAT (Total) | .75*** | .86*** | .90*** | .89*** | -- | | | | | | | | | | | |
| 6. POMS (Ang) | .19* | .10 | .23* | .22* | .23* | -- | | | | | | | | | | |
| 7. POMS (Conf) | .24** | .29** | .31** | .32*** | .34*** | .62*** | -- | | | | | | | | | |
| 8. POMS (Dep) | .33*** | .24** | .22* | .22* | .30** | .75*** | .79*** | -- | | | | | | | | |
| 9. POMS (Fat) | .15 | .19* | .26** | .22* | .24** | .63*** | .75*** | .79*** | -- | | | | | | | |
| 10. POMS (Ten) | .05 | .12 | .17 | .16 | .15 | .62*** | .77*** | .66*** | .64*** | -- | | | | | | |
| 11. POMS (Vig) | -.21* | -.31** | -.39*** | -.30** | -.36*** | -.19* | -.39*** | -.26** | -.38*** | -.29** | -- | | | | | |
| 12. POMS (Total) | .24* | .25** | .31** | .29** | .32** | .82*** | .89*** | .90*** | .86*** | .83*** | .46*** | -- | | | | |
| 13. VA-I | -.01 | -.08 | -.23* | -.25** | -.18 | -.07 | -.23* | -.06 | -.11 | .01 | .20* | -.11 | -- | | | |
| 14.VA-S | .25** | .24** | .28** | .22* | .29** | .31** | .38*** | .28** | .38*** | .55*** | .27** | .45*** | .15 | -- | | |
| 15.VA-C | .22* | .19* | .17 | .15 | .21* | .13 | .25** | .20* | .12 | .34*** | -.07 | .22* | .09 | .56*** | -- | |
| 16. VA-D | .33*** | .41*** | .44*** | .40*** | .46*** | .22* | .32*** | .22* | .30** | .24** | .32*** | .34*** | .01 | .51*** | .31** | -- |

*Note*. PASAT – Paced Auditory Serial Addition Task; POMS – Profile of Mood States; POMS (Ang) – Anger subscale; POMS (Conf) – Confusion subscale; POMS (Dep) – Depression subscale; POMS (Fat) – Fatigue subscale; POMS (Ten) – Tension/anxiety subscale; POMS (Vig) – Vigor subscale (reverse scored); VA-I – Visual Analog, Involvement; VA-S – Visual Analog, Stress; VA-C – Visual Analog, Concern; VA-D – Visual Analog, Difficulty.

* $p < .05$. ** $p < .01$. *** $p < .001$.

Table 7. Pearson correlations between working memory and emotional distress at follow-up

| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1. PASAT 1 (2.6 s) | -- | | | | | | | | | | | |
| 2. PASAT 2 (2.4 s) | .62*** | -- | | | | | | | | | | |
| 3. PASAT 3 (2.0 s) | .51*** | .71*** | -- | | | | | | | | | |
| 4. PASAT 4 (1.6 s) | .48*** | .67*** | .80*** | -- | | | | | | | | |
| 5. PASAT (Total) | .75*** | .86*** | .90*** | .89*** | -- | | | | | | | |
| 6. POMS (Ang) | .27** | .12 | .19* | .12 | .21* | -- | | | | | | |
| 7. POMS (Conf) | .31** | .27** | .27** | .22* | .31** | .58*** | -- | | | | | |
| 8. POMS (Dep) | .32** | .17 | .13 | .11 | .21* | .67*** | .77*** | -- | | | | |
| 9. POMS (Fat) | .19* | .19* | .23* | .15 | .22* | .53*** | .76*** | .70*** | -- | | | |
| 10. POMS (Ten) | .17 | .14 | .16 | .11 | .17 | .58*** | .74*** | .71*** | .62*** | -- | | |
| 11. POMS (Vig) | -.22* | -.30** | -.31** | -.21* | -.30** | -.14 | -.42*** | -.23** | -.32*** | -.29** | -- | |
| 12. POMS (Total) | .28** | .23** | .26** | .19 | .28** | .70*** | .89*** | .87*** | .83*** | .81*** | -.57*** | -- |

*Note.* PASAT – Paced Auditory Serial Addition Task; POMS – Profile of Mood States; POMS (Ang) – Anger subscale; POMS (Conf) – Confusion subscale; POMS (Dep) – Depression subscale; POMS (Fat) – Fatigue subscale; POMS (Ten) – Tension/anxiety subscale; POMS (Vig) – Vigor subscale (reverse scored); VA-I – Visual Analog, Involvement; VA-S – Visual Analog, Stress; VA-C – Visual Analog, Concern; VA-D – Visual Analog, Difficulty

* $p < .05$. ** $p < .01$. *** $p < .001$.

**Aim 1: Working memory under stress**

**Working memory by diagnosis.** The mean number of total errors on the PASAT in the sample overall was 55.13 (*SD* = 30.44). Total PASAT errors were unrelated to age ($r(112) = .14$, $p = .144$), years of education ($r(107) = -.13$, $p = .191$), race ($F(5,108) = 0.51$, $p = .765$), or sex ($F(1,112) = 0.32$, $p = .574$). However, there was a modest inverse relationship between years of education and PASAT errors on the easiest trial ($r(107) = -.19$, $p = .049$). There were no significant differences across diagnostic groups in total PASAT errors ($F(2,108) = 1.497$, $p = .228$) and no group differences in errors on any of the four trials or in change scores. These findings did not support Hypothesis 1a, which predicted that BPD participants would show poorer working memory performance compared to MDD participants and controls. Hypothesis 1b, which predicted that MDD participants would show significantly better working memory performance compared to BPD participants, was also not supported. PASAT errors by diagnostic group and for the total sample are presented in Table 8. Errors for each PASAT trial are presented for each diagnostic group in Figure 3. The impact of diagnosis on working memory is clarified further in the results section titled "Emotional distress and working memory by diagnosis."

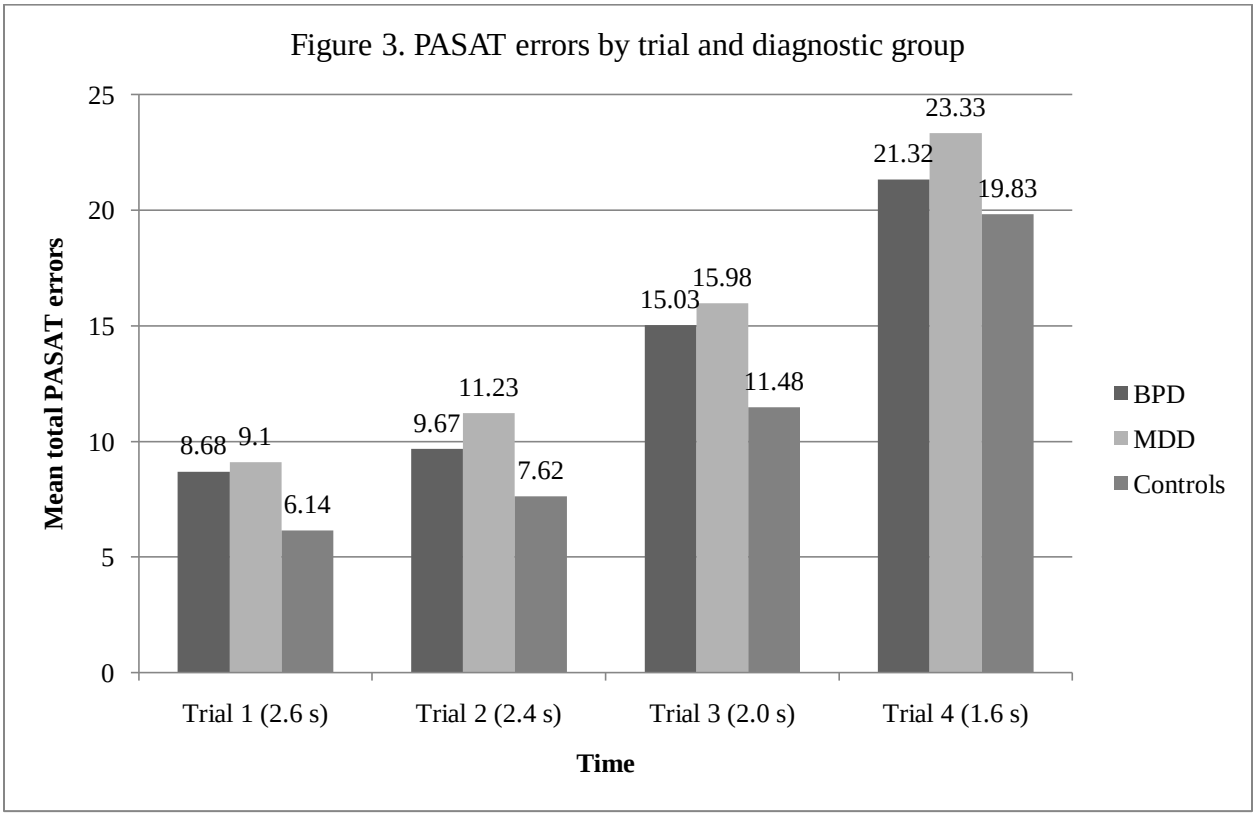
Table 8. Working memory errors by diagnostic group

| | BPD (*n* = 60) | MDD (*n* = 30) | Control (*n* = 21) | Total (*n* = 111) | ANOVA | |
|---|---|---|---|---|---|---|
| | *M (SD)* | *M (SD)* | *M (SD)* | *M (SD)* | *F* | *p* |
| PASAT (no. of errors) | | | | | | |
| Trial 1 (2.6 s) | 8.68 (9.07) | 9.10 (8.07) | 6.14 (8.17) | 8.37 (8.50) | $F(2,108) = 0.841$ | .434 |
| Trial 2 (2.4 s) | 9.67 (7.27) | 11.23 (6.20) | 7.62 (5.95) | 10.00 (7.18) | $F(2,108) = 1.766$ | .176 |
| Trial 3(2.0 s) | 15.03 (9.07) | 15.98 (8.53) | 11.48 (8.47) | 14.89 (9.12) | $F(2,108) = 1.760$ | .177 |
| Trial 4 (1.6 s) | 21.32 (10.99) | 23.33 (9.22) | 19.83 (12.11) | 21.86 (10.87) | $F(2,108) = 0.692$ | .503 |
| Total score | 54.70 (31.40) | 59.65 (27.39) | 45.07 (28.18) | 55.13 (30.44) | $F(2,108) = 1.497$ | .228 |
| Change scores (T4-T1) | 12.63 (10.05) | 14.23 (8.04) | 13.69 (12.42) | 13.49 (10.08) | $F(2,108) = 0.276$ | .759 |

*Note.* PASAT – Paced Auditory Serial Addition Task

* $p < .05$ ** $p < .01$ *** $p < .001$

Figure 3. PASAT errors by trial and diagnostic group

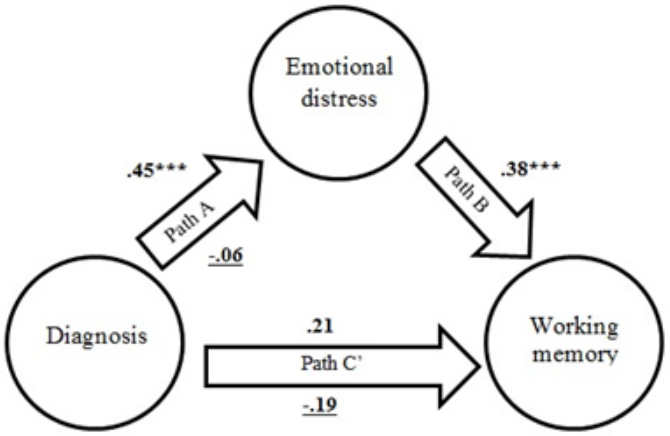


**Distress as proposed mediator.** To test Hypothesis 1c, which predicted that the impact of diagnosis on working memory performance would be mediated by emotional distress, the causal steps approach outlined by Baron and Kenny (1986) was followed. According to the causal steps approach, mediation is established in four steps. In Step 1, an independent variable (X) is shown to significantly predict a dependent variable (Y) (X → Y). In Step 2, the independent variable (X) is shown to significantly predict a mediator (M) (X → M). In Step 3, the mediator is shown to significantly predict the dependent variable when the independent variable is controlled for (M|X → Y). Finally, in Step 4, the presence of the mediator results in the non-significance or reduction in magnitude of the relationship between the independent variable (X) and the dependent variable (Y) (X|M → Y).

Diagnosis was entered as the independent variable (X). Due to the presence of more than two levels in the variable, the mediation analysis was performed twice using two contrast-coded variables for diagnosis (described in "Method" section). The first mediation analysis was performed with the BPD-versus-control contrast variable as the independent variable and the BPD-versus-MDD contrast variable as a covariate, and the second analysis was performed with the BPD-versus-MDD variable as the independent variable and the BPD-versus-control variable as the covariate (Hayes & Preacher, 2013). Post-TSST POMS scores (emotional distress) were used as the mediator (M) in the analysis, and total PASAT errors (working memory) were used as the dependent variable (Y). In the temporal sequence of this procedure, the proposed mediator of distress (M) was assessed after the working memory task was implemented (Y). However, the use of mediators which are measured after the dependent variable in temporal sequence is justified when the mediator is theoretically presumed to occur between the independent and dependent variables (Warner, 2013).

In Step 1 of the mediation model, the impact of diagnosis on working memory, ignoring the mediator of distress, was non-significant ($R^2$ = .02, $F$(2,105) = 1.32, $p$ = .273). This failed to support Hypothesis 1c, which predicted that the relationship between diagnosis and working memory would be mediated by emotional distress. On an exploratory basis, however, Steps 2 through 4 of the mediation analysis were carried out to clarify the relationships among the other variables in the model. Step 2 showed that diagnosis significantly predicted distress ($R^2$ = .16, $F$(2,105) = 10.62, $p$ < .001). Further examination of the contrast-coded diagnosis variables revealed an effect for the BPD v. controls comparison ($\beta$ = .45, $t$(104) = 3.84, $p$ < .001), such that BPD participants had significantly higher emotional distress ($M$ = 50.75, $SD$ = 48.43) and controls had lower distress ($M$ = 1.11, $SD$ = 23.73) compared to the sample mean ($M$ = 36.27, $SD$ = 44.55). Step 3 showed that emotional distress, controlling for diagnosis, significantly predicted working memory ($\beta$ = .38, $t$(103) = 3.77, $p$ < .001). Step 4 showed that diagnosis, controlling for emotional distress, did not significantly predict working memory, either in the BPD v. controls comparison ($\beta$ = .03, $t$(103) = 0.19, $p$ = .847) or the BPD v. MDD comparison ($\beta$ = -.16, $t$(103) = -1.27, $p$ = .206). Relationships among all variables are depicted in Figure 4.

Figure 4. Emotional distress as a proposed mediator of the impact of diagnosis on working memory




*Note.* Diagnosis was analyzed with two contrast-coded variables: BPD versus controls and BPD versus MDD. Underlined values represent standardized regression coefficients for the comparison between BPD and MDD.

*$p < .05$, **$p < .01$, ***$p < .001$

## Aim 2: Impact of distress on working memory

**Relationship between distress and working memory.** In the sample overall, there was a significant positive zero-order correlation between total PASAT errors and total POMS scores post-TSST ($r(108) = .324$, $p = .001$) and at follow-up ($r(107) = .277$, $p = .004$), but not pre-TSST ($r(104) = .047$, $p = .634$). This supported Hypothesis 2a, which predicted that higher levels of emotional distress post-TSST would be associated with greater number of errors on the working memory task. All post-TSST POMS subscales, with the exception of tension, were significantly correlated with total PASAT errors; the strongest association was found for vigor, which was negatively associated with PASAT errors ($r(115) = -.38$, $p < .001$).

Total PASAT errors were also positively correlated with POMS change scores from pre-TSST to post-TSST ($r(98) = .420$, $p < .001$) and negatively correlated with POMS change scores from post-TSST to follow-up ($r(100) = -.199$, $p = .045$). Zero-order correlations indicated that visual analog ratings of stress ($r(116) = .287$, $p = .002$), concern ($r(116) = .210$, $p = .022$), and difficulty ($r(116) = .463$, $p < .001$) associated with the psychological stress task were positively associated with total PASAT errors. This was not true for self-reports of involvement in the stress task ($r(116) = -.181$, $p = .050$).

## Emotional distress and working memory by diagnosis

**Simple correlations.** The relationship between emotional distress and working memory was first examined by comparing Pearson correlation coefficients between these two variables within diagnostic groups. The relationship between emotional distress post-TSST and working memory errors was positive and significant among BPD participants ($r(54) = .443$, $p = .001$) but not among MDD participants ($r(27) = .137$, $p = .480$) or controls ($r(17) = .282$, $p = .242$). A similar pattern was observed at follow-up; the relationship between emotional distress and

working memory errors was again positive and significant for BPD participants ($r(53) = .302$, $p = .025$) but not for MDD participants ($r(27) = .104$, $p = .607$) or controls ($r(18) = .375$, p = .103).

To compare the strength of these correlations obtained from within diagnostic groups, correlation coefficients were converted into z-scores using Fisher's *r*-to-*z* transformation. This test revealed that BPD participants did not show a stronger relationship between emotional distress and working memory than did MDD participants or controls. This was found post-TSST (BPD versus MDD, $Z = 1.412$, $p = .158$; BPD versus controls, $Z = 0.652$, $p = .514$) and at follow-up (BPD versus MDD, $Z = 0.863$, $p = .388$; BPD versus controls, $Z = -0.295$, $p = .768$). This test failed to support Hypothesis 2b, which predicted that the relationship between post-TSST distress and working memory performance would be stronger for BPD participants than for healthy controls.

**Impact of distress on working memory.** For the second test of Hypothesis 2b, which predicted that the relationship between post-TSST distress and working memory performance would be stronger for BPD participants than for healthy controls, we conducted a hierarchical multiple regression analysis. The regression model sought to determine whether diagnosis moderated the relationship between distress and working memory. In this process, variables were added sequentially to examine their unique ability to explain the variance in working memory. In Model 1, POMS scores pre-TSST (hereafter referred to as "baseline emotional distress") were included as a covariate to control for the significantly higher scores among BPD and MDD participants compared to controls ($F(2,97) = 11.694$, $p < .001$). In Model 2, two effects-coded contrast variables were used to make diagnostic comparisons. The first contrast variable (BPD = 1, MDD = 0, control = -1) compared BPD participants' and controls' PASAT errors against mean PASAT errors in the sample. The second contrast variable (BPD = 1, MDD = -1, control =

0) compared BPD participants' and MDD participants' PASAT errors against mean PASAT errors in the sample. The addition of diagnosis to the model did not improve explained variance in PASAT errors ($\Delta R^2 = .017$, $p = .449$) and diagnosis alone did not significantly predict PASAT errors ($F(3,93) = 0.694$, $p = .558$), even when controlling for baseline emotional distress. Thus, diagnosis alone did not appear to impact working memory performance.

In Model 3, post-TSST POMS scores (hereafter referred to as "emotional distress") were added to the model, in order to examine the impact of emotional distress and diagnosis together on working memory. The addition of emotional distress significantly improved model fit ($\Delta R^2 = .230$, $p < .001$), and emotional distress by itself predicted working memory ($\beta = .793$, $t(91) = 5.317$, $p < .001$) when controlling for baseline emotional distress. The addition of emotional distress also resulted in a main effect for diagnosis, such that the comparison of BPD participants' and MDD participants' PASAT errors against mean PASAT errors in the sample became significant ($\beta = -.271$, $t(91) = -2.183$, $p = .032$). This indicated that, after controlling for emotional distress at both time-points, BPD participants made significantly fewer errors on the PASAT ($M = 54.70$, $SD = 31.40$), while MDD participants showed greater errors ($M = 59.65$, $SD = 27.39$), compared to the mean number of errors in the sample ($M = 55.13$, $SD = 30.44$). The comparison of BPD participants' and controls' PASAT errors to mean PASAT errors in the sample was not significant ($\beta = .114$, $t(97) = 0.851$, $p = .397$), suggesting that BPD participants' working memory performance was not significantly different from that of controls.

Finally, Model 4 tested the hypothesis that diagnosis would moderate the impact of emotional distress on working memory performance. Two multiplicative interaction terms (between centered POMS scores post-TSST and both diagnosis contrast variables) were added to the model. This did not result in a significant improvement of explained variance in PASAT

errors ($\Delta R^2$ = .017, $p$ = .356). Both interactions tested were non-significant: the impact of emotional distress on working memory was not significantly different for BPD participants or controls than it was for the sample overall ($\beta$ = -.269, $t$(99) = -1.031, $p$ = .305). Similarly, the impact of emotional distress on working memory was not significantly different for BPD participants or MDD participants than it was for the overall sample ($\beta$ = .303, $t$(99) = 1.437, $p$ = .154). Emotional distress by itself remained a significant predictor of working memory performance in this model ($\beta$ = .758, $t$(99) = 3.539, $p$ = .001). Diagnosis by itself was no longer a significant predictor of working memory in this model, either in the comparison of BPD participants and controls to overall PASAT errors ($\beta$ = -.042, $t$(99) = -0.194, $p$ = .847) or in the comparison of BPD and MDD participants to overall PASAT errors ($\beta$ = -.169, $t$(99) = -1.065, $p$ = .290). These findings are presented in Table 9.

Table 9. The impact of emotional distress and diagnosis on working memory performance

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Variable | *B* | SE *B* | *β* | *B* | SE *B* | *β* | *B* | SE *B* | *β* | *B* | SE *B* | *β* |
| (Covariate)[a] | 0.05 | 0.73 | .07 | 0.02 | 0.08 | .03 | -0.40*** | 0.11 | -.55 | -0.36** | 0.11 | -.51 |
| BPD v. MDD | | | | -6.12 | 5.01 | -.17 | -9.73* | 4.46 | -.27 | -6.06 | 5.70 | -.17 |
| BPD v. control | | | | 6.19 | 5.72 | .16 | 4.29 | 5.05 | .114 | -1.60 | 8.25 | -.04 |
| Distress[b] | | | | | | | 0.52*** | 0.10 | .793 | 0.50** | 0.14 | .758 |
| BPD v. MDD x distress | | | | | | | | | | 0.23 | 0.16 | .30 |
| BPD v. control x distress | | | | | | | | | | -0.20 | 0.20 | -.27 |
| $\Delta R^2$ | .005 | | | .017 | | | .230 | | | .017 | | |
| *F* for $\Delta R^2$ | 0.47 | | | 0.81 | | | 28.27*** | | | 1.05 | | |

*Note.* [a] POMS scores at baseline were entered as a covariate in all models. [b] Distress – POMS scores post-TSST

* $p < .05$ ** $p < .01$ *** $p < .001$

In summary, emotional distress and diagnosis (BPD-versus-MDD) each significantly predicted working memory performance. However, there was no interaction between emotional distress and diagnosis in predicting working memory. That is, the impact of emotional distress on working memory did not appear to differ significantly by diagnosis.

**Impact of specific emotions on working memory.** To determine which emotional state (i.e., POMS subscale) was the best predictor of working memory, an additional multiple regression analysis was conducted in which anger, confusion, depression, tension/anxiety, fatigue, and vigor were entered as predictors of PASAT errors. These findings are shown in Table 10. Confusion ($\beta$ = .40, $t(105)$ = 2.16, $p$ = .033) positively predicted PASAT errors, while tension ($\beta$ = -.29, $t(105)$ = -2.04, $p$ = .044) and vigor ($\beta$ = -.30, $t(105)$ = -3.13, $p$ = .002) negatively predicted PASAT errors. Anger, depression, and fatigue did not significantly predict working memory. A stepwise multiple regression analysis was performed to determine which emotional states best predicted working memory. This analysis confirmed that confusion and vigor were the best predictors of working memory, such that confusion positively predicted PASAT errors ($\beta$ = .23, $t(109)$ = 2.49, $p$ = .014) and vigor negatively predicted PASAT errors ($\beta$ = -.27, $t(105)$ = -2.88, $p$ = .005). Tension no longer predicted working memory in this analysis, based on a stepwise exclusion criterion of $p > .100$.

Table 10. Summary of simple regression analysis for specific predictors of working memory

| Variable | *B* | SE *B* | *β* | *t* | *p* |
|---|---|---|---|---|---|
| Anger | 0.37 | 0.40 | .12 | 0.92 | .362 |
| Confusion | 1.76 | 0.81 | .40 | 2.16 | .033* |
| Depression | 0.36 | 0.41 | .15 | 0.88 | .379 |
| Fatigue | -0.77 | 0.62 | -.19 | -1.24 | .217 |
| Tension | -0.91 | 0.45 | -.29 | -2.04 | .044* |
| Vigor | -1.19 | 0.38 | -.30 | -3.13 | .002** |
| $R^2$ | | .22 | | | |
| *F* | | 5.13*** | | | |

*Note.* * $p < .05$ ** $p < .01$ *** $p < .001$

To determine whether the impact of confusion and vigor on working memory differed for diagnostic groups, the hierarchical multiple regression analysis described above was repeated; one analysis was conducted for confusion, and one analysis was conducted for vigor. In the first hierarchical regression, controlling for baseline levels of confusion, main effects were observed for post-TSST confusion and diagnosis. Confusion positively predicted working memory ($\beta$ = .61, $t(101) = 4.34$, $p < .001$). Diagnosis predicted working memory, but only when controlling for confusion ($\beta$ = -.27, $t(101) = -2.13$, $p = .035$). BPD participants made significantly fewer errors on the PASAT ($M = 54.70$, $SD = 31.40$) than did MDD participants ($M = 59.65$, $SD = 27.39$) compared to the mean number of errors in the sample ($M = 55.13$, $SD = 30.44$). There was no interaction between confusion and diagnosis in predicting working memory. These findings are presented in Table 11.

In the second hierarchical regression, controlling for baseline levels of vigor, a main effect for vigor but not diagnosis was observed. Vigor negatively predicted working memory errors ($\beta$ = -.41, $t(101) = -2.66$, $p = .009$). There was no interaction between vigor and diagnosis in predicting working memory. These findings are presented in Table 12.

Table 11. The impact of POMS confusion scores and diagnosis on working memory performance

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Variable | *B* | SE *B* | *β* | *B* | SE *B* | *β* | *B* | SE *B* | *β* | *B* | SE *B* | *β* |
| (Covariate)[a] | 0.81 | 0.54 | .14 | 0.59 | 0.60 | .10 | -1.88 | 0.80 | -.33* | -1.54 | 0.82 | -.27 |
| BPD v. MDD | | | | -5.45 | 4.72 | -.15 | -9.50 | 4.46 | -.27* | -5.99 | 5.43 | -.17 |
| BPD v. control | | | | 5.77 | 5.46 | .15 | 5.68 | 5.04 | .15 | 1.20 | 7.20 | .03 |
| Confusion[b] | | | | | | | 2.68 | 0.62 | .61*** | 2.31 | 0.79 | .52** |
| BPD v. MDD x confusion | | | | | | | | | | 1.45 | .93 | .29 |
| BPD v. control x confusion | | | | | | | | | | -1.03 | 1.10 | -.20 |
| $\Delta R^2$ | .02 | | | .01 | | | .15 | | | .02 | | |
| $F$ for $\Delta R^2$ | 2.29 | | | 0.74 | | | 18.84*** | | | 1.31 | | |

*Note.* [a] POMS confusion scores at baseline were entered as a covariate in all models. [b] Confusion – POMS confusion scores post-TSST

* $p < .05$ ** $p < .01$ *** $p < .001$

Table 12. The impact of POMS vigor scores and diagnosis on working memory performance

| | Model 1 | | | Model 2 | | | Model 3 | | | Model 4 | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Variable | *B* | SE *B* | *β* | *B* | SE *B* | *β* | *B* | SE *B* | *β* | *B* | SE *B* | *β* |
| (Covariate)[a] | -1.07 | 0.36 | -.28** | -1.01 | 0.39 | -.26* | 0.27 | 0.61 | .07 | 0.27 | 0.62 | .07 |
| BPD v. MDD | | | | -4.54 | 4.45 | -.13 | -4.20 | 4.32 | -.12 | -4.30 | 4.48 | -.12 |
| BPD v. control | | | | 3.10 | 5.12 | .08 | 3.05 | 4.50 | .08 | 3.00 | 5.31 | .08 |
| Vigor[b] | | | | | | | -1.61 | 0.60 | -.41** | -1.61 | 0.62 | -.41* |
| BPD v. MDD x vigor | | | | | | | | | | -0.13 | 0.54 | -.03 |
| BPD v. control x vigor | | | | | | | | | | 0.03 | 0.57 | .01 |
| $\Delta R^2$ | .08 | | | .01 | | | .06 | | | .001 | | |
| *F* for $\Delta R^2$ | 8.77** | | | 0.53 | | | 7.08** | | | 0.04 | | |

*Note.* [a] POMS vigor scores at baseline were entered as a covariate in all models. [b] Vigor – POMS vigor scores post-TSST

* $p < .05$ ** $p < .01$ *** $p < .001$

# CHAPTER IV:

# DISCUSSION

The primary aims of this study were to investigate the impact of emotional distress on working memory performance and to determine whether emotion-induced working memory disruption was stronger for BPD participants than for MDD participants or healthy controls. This was examined in the context of a psychological stress procedure, in which participants were asked to give a short speech and perform rapid mental arithmetic calculations in front of study confederates. Participants completed subjective ratings of distress before, immediately following, and at follow-up after the stress procedure. The number of errors on the arithmetic task performed during the stress procedure was used to assess participants' working memory performance. Broadly speaking, this study aimed to assess the extent to which individuals with BPD may be more vulnerable to cognitive interference from intense emotional experiences, particularly those generated by negative evaluation fears and hypersensitivity to rejection. This section will review and interpret key findings.

## Demographic characteristics

Participants in the current sample were predominantly female and highly educated, and the majority of participants identified as white. Approximately half the participants were employed, although one-third of unemployed participants were students. The fact that the sample was highly educated (most had completed some college) likely reflects the population surrounding a university teaching hospital in an urban setting. Greater educational attainment may also have been made more likely by the fact that many clinical participants were referred by mental health or medical providers, and were therefore receiving effective management of their psychiatric symptoms. Despite the high level of education in the sample overall, it was not

controlled for in the current study because educational attainment did not differ significantly across diagnostic groups. The fact that the current sample was predominantly female is consistent with the higher prevalence of BPD among women found in previous research (*DSM-IV-TR*, 2000; Sansone & Sansone, 2011; Skodol & Bender, 2003).

**Clinical characteristics**

Analyses revealed significant clinical differences at baseline between BPD and MDD participants in the sample. BPD participants had a higher degree of comorbidity with other psychiatric disorders than did MDD participants, and were more likely to carry three or more Axis I diagnoses. This is consistent with the higher prevalence of Axis I comorbidity in BPD than in MDD found in previous studies (Zanarini et al., 1998; Zimmerman & Mattia, 1999). Compared to MDD participants, BPD participants in this sample were also more likely to have had a history of self-injurious behavior, and showed higher levels of aggression and impulsivity. In addition, BPD participants demonstrated lower psychosocial functioning and greater overall psychiatric symptoms than did MDD participants. These findings suggest that the participants with BPD were more severely ill than those with MDD in a number of areas. The high prevalence of comorbid current depression (50%) and lifetime depression (45%) among the BPD patients in this sample, a rate consistent with previous estimates (e.g., McGlashan et al., 2000), indicates further that this was a particularly ill sample. Indeed, co-occurring BPD and MDD have been shown to relate to poorer clinical outcomes (Gunderson et al., 2004) and greater likelihood of suicide attempts and completions (Corbitt, Malone, Haas, & Mann, 1996; Soloff, Lynch, Kelly, Malone, & Mann, 2000). It is notable that BPD and MDD participants did not differ in their reported degree of hopelessness, since previous studies comparing these groups have found greater hopelessness in BPD (Fertuck et al., 2006b). However, the parent study from which this

sample was drawn recruited equal numbers of suicide attempters in the BPD and MDD groups. Given that hopelessness is known to be a strong predictor of suicidality (Beck, 2006; Beck, Steer, Kovacs, & Garrison, 1985; Mann, 2002), this may have accounted for the equivalent degree of hopelessness between the two groups.

BPD participants also had higher levels of depression and anxiety than did MDD participants, though findings varied depending on the measures that were used to assess these symptoms. BPD participants showed significantly higher scores on the HAM-D (24-item version), a clinician-rated measure that assesses neurovegetative, behavioral, cognitive, and motivational domains of depression. In contrast, BPD participants' elevation in scores on the BDI-II – an instrument which emphasizes cognitive symptoms of depression – was not significant. This suggests that, despite the high prevalence of current or lifetime diagnoses of depression within the BPD group in this sample, individuals with BPD versus MDD may experience different qualitative features of depression. For example, depression in BPD is more likely to be characterized by mood reactivity and interpersonal sensitivity than in MDD (Fertuck et al., 2006; Feske et al., 2004; Joyce et al., 2003). These domains are more likely to be probed by the HAM-D than the BDI.

For anxiety, the opposite pattern in measures was found. BPD participants scored higher on the STAI, a self-report measure of current distress, but not the HAM-A, a clinician-rated measure of psychological and somatic symptoms of anxiety in the past week. BPD participants may have rated themselves as being in greater momentary distress in part due to the pervasive and intense negative affectivity that characterizes the disorder (Stiglmayr et al., 2001; Zanarini et al., 1998). In addition, some researchers have noted that the HAM-A does not reliably distinguish between generalized anxiety disorder and depression (Koerner, Antony, & Dugas,

2010; Roemer, 2001). Given the overlap in the measurement of cognitive and affective symptoms between the HAM-A and BDI, it is perhaps not surprising that the equivalence of BPD and MDD participants was consistent across these measures.

**Aim 1: Working memory under stress**

**Working memory by diagnosis.** Working memory performance in the sample was unrelated to age, years of education, race, or sex. Research examining age differences in working memory performance on the PASAT has been inconclusive. Some studies have demonstrated that older age is associated with poorer working memory performance (Brittain, LaMarche, Reeder, Roth, & Boll, 1991; Diehr et al., 2003), while others have found that older participants actually perform better than their younger counterparts (Ward, 1997). In part, these contradictory findings could be attributable to different cognitive strengths across the life span. Working memory performance might be superior in younger individuals, but older individuals could have greater familiarity and ease with routine mathematical calculations (Tombaugh, 2006). Moreover, the oldest participant in this sample was only 57, making it less likely to detect significant age-related deterioration in working memory.

Research on sex differences in PASAT performance has yielded more consistent findings, with most studies showing no significant differences by sex (Diehr et al., 2003; MacLeod & Prior, 1996; Wingenfeld, Holdwick, Davis, & Hunter, 1999). Thus, the finding that PASAT performance was unrelated to sex in this sample is consistent with this body of literature. However, in contrast to the finding that overall PASAT performance was unrelated to education, many studies have documented a relationship between greater education and superior PASAT performance (e.g., Brittain et al., 1991; Diehr, Heaton, Miller, & Grant, 1998; Wiens, Fuller, & Crossen, 1997). Greater education was slightly associated with fewer PASAT errors on the

easiest trial of the task in our sample. However, previous research examining demographic differences in PASAT performance has been performed in healthy samples, and has not examined performance on the PASAT when administered under stressful conditions. It is therefore difficult to clarify the precise nature of the relationship between education and working memory in this sample. Participants' high degree of stress while taking the PASAT could have obscured the impact of other demographic variables. Studies examining the relationship between race and PASAT performance have yielded inconclusive results (Brittain et al., 1991; Diehr et al., 1998; Wiens et al, 1997).

Contrary to Hypotheses 1a and 1b, no differences were observed between BPD participants, MDD participants, and controls in working memory performance on the PASAT, both in total number of errors, in individual performance on each trial, and in change scores (i.e., degree of deterioration) from Trial 1 to Trial 4. However, when emotional distress was controlled in subsequent analyses, superior working memory performance was observed among BPD participants compared to MDD participants. Put another way, BPD participants would show better working memory performance, and MDD participants poorer performance, if both groups were equally distressed. Given the complex relationship between diagnosis and distress in predicting working memory performance, these findings are discussed further in the "Impact of distress on working memory" section of this chapter.

**Distress as proposed mediator.** Contrary to Hypothesis 1c, the impact of diagnosis on working memory was not mediated by emotional distress because a simple relationship between diagnosis and working memory (i.e., Step 1 of the causal steps approach to mediation; Baron & Kenny, 1986) was not found. Mediational analyses are sensitive to small sample sizes which contribute to lack of statistical power (Fritz & MacKinnon, 2007). In the current study, unequal

sample sizes may have contributed to this statistical underpowering. Moreover, in traditional meditational analyses, mediators are variables which occur in temporal sequence between the occurrence of the independent and dependent variables. The mediator assessed in the current study (i.e., distress) was measured *following* the dependent variable (i.e., working memory). Although this was justified because distress was theoretically presumed to occur before the working memory task took place, it was nonetheless a limitation of the analysis. These findings are discussed further in the "Impact of distress on working memory" section of this chapter.

**Aim 2: Impact of distress on working memory**

**Relationship between distress and working memory.** Emotional distress immediately following the stress task and at follow-up was positively correlated with working memory errors in the sample overall. It is noteworthy that there was no relationship between baseline emotional distress and working memory, and that this relationship only emerged after the onset of the stressor. Of the six POMS subscales, vigor was most strongly associated with working memory, such that it was negatively correlated with working memory errors. Participant ratings of stress, concern about their performance, and perceived difficulty of the task were also positively associated with working memory errors. In addition, working memory errors were positively correlated with distress change scores from pre-TSST to post-TSST, and negatively correlated with distress change scores from post-TSST to follow-up. That is, greater increases in distress were associated with poorer working memory performance in the sample, while more pronounced decreases in distress after the TSST (i.e., a fuller return to baseline) were associated with superior working memory performance.

This finding helps to shed light not only on the rises in distress that were associated with poorer cognitive functioning, but on the effect of the post-task regulation period. One would

expect that the gradual reduction in emotional distress following a stressful experience would occur more slowly for individuals with BPD, given the slower return to baseline that is considered a hallmark of their emotional reactivity (Linehan, 1993; Herpertz, 2003). Although it was beyond the scope of the current study to compare the magnitude of change in POMS scores across diagnostic groups, future studies should seek to determine whether changes in distress are more strongly associated with working memory in BPD compared to MDD or healthy controls.

**Relationship between distress and working memory by diagnosis.** Exploratory correlational analyses revealed that working memory errors and emotional distress post-TSST and at follow-up and were positively associated for BPD participants, but not for MDD participants or controls, when the groups were analyzed separately. However, when the strength of correlation coefficients were compared across the three groups, the correlations found between distress and working memory were not significantly different. That is, the relationship between distress and working memory did not appear to be stronger for BPD participants than for MDD participants or controls.

**Impact of distress on working memory.** Controlling for baseline emotional distress (which was significantly higher in BPD and MDD participants compared to controls), emotional distress post-TSST was found to be a strong predictor of working memory performance in the sample overall. Diagnosis alone did not predict working memory performance, even when controlling for baseline emotional distress. However, controlling for post-TSST emotional distress resulted in *superior* working memory performance for BPD participants and poorer performance for MDD participants when compared to the overall sample. Controlling for post-TSST emotional distress did not reveal significant differences in working memory for BPD participants compared to controls, indicating that distress had a unique impact on working

memory in the clinical groups but not in the control group. No interaction between distress and diagnosis was observed in predicting working memory; that is, the deleterious impact of distress on working memory performance did not appear to be greater for BPD participants than for MDD participants or controls. Contrary to the predictions made for the mediation analysis, in which it was anticipated that distress would mediate the relationship between diagnosis and working memory, it appears as though distress acted as a *suppressor* in the relationship between diagnosis and distress, such that its large impact on cognitive performance had to be statistically controlled before any group differences in working memory were found. This finding is discussed further in the "General discussion" section of this chapter.

**Impact of specific emotions on working memory.** To further explore the finding that post-TSST total POMS scores predicted working memory, multiple regression analyses were conducted to determine which emotional states (i.e., POMS subscale) accounted for this relationship. POMS subscales included tension/anxiety, depression/dejection, anger/hostility, confusion/bewilderment, fatigue/inertia, and vigor/activity. Of the six emotional states assessed by the POMS, confusion and vigor were the strongest predictors of working memory performance, such that confusion positively predicted errors and vigor negatively predicted errors. BPD participants again showed superior working memory performance, and MDD participants poorer performance, when confusion was controlled for. However, the same was not true for vigor; controlling for vigor did not result in superior performance for BPD participants and poorer performance for MDD participants. This suggests that working memory performance was roughly the same across groups when all participants were equally invigorated, but that when the groups were equally confused, BPD participants performed better on the working memory measure. It is noteworthy that different relationships between diagnosis and working

memory were found for positively valenced versus negatively valenced emotional states. BPD participants appeared to show better performance when equally as confused, but not when equally invigorated. Negative emotional states may be more salient for BPD participants than for MDD participants, and in some cases may actually spur motivation that improves performance. This finding is consistent with a previous study indicating that negative affective states predict *greater* impulse control for individuals with BPD traits compared to those without BPD traits (Chapman, Leung, & Lynch, 2008), which has been theorized to relate to harm avoidance traits in BPD (Ball et al., 1997).

**General discussion**

The results of this study and others (Fertuck et al., 2006b; Sprock et al., 2000) suggest that the role of emotional distress may be critical when evaluating neurocognitive functioning in BPD individuals compared to controls and other psychiatric populations. Ignoring the impact of distress, there were no basic neurocognitive differences between BPD participants, MDD participants, and controls in this sample. This is consistent with a large body of literature, though findings on neurocognitive differences between BPD participants and other groups have been equivocal. For instance, numerous studies comparing BPD participants to healthy controls on neurocognitive measures have found no differences in various domains, such as working memory and attention (Dinn et al., 2004; Fertuck et al., 2006b; Kunert et al., 2003; Sprock et al., 2000), learning and memory (Cornelius et al., 1989; Dinn et al., 2004; Fertuck et al., 2006b; Bazanis et al., 2002; Kunert et al., 2003), language abilities (Cornelius et al., 1989), intelligence (Fertuck et al., 2006b; Kunert et al., 2003), motor abilities (Cornelius et al., 1989; Fertuck et al., 2006b), visuospatial abilities (Cornelius et al., 1989), processing speed (Fertuck et al., 2006b),

and executive functioning (Fertuck et al., 2006b; Kunert et al., 2003; LeGris et al., 2012; Sprock et al., 2000).

However, other studies examining neurocognitive differences between individuals with BPD and healthy controls have shown deficits consistent with frontal lobe dysfunction in BPD, such as attention, memory, executive functioning, processing speed, and visuospatial abilities (Posner et al., 2002; Lenzenweger et al., 2004; Ruocco & Trobst, 2003; Tebartz van Elst et al., 2003). Indeed, a meta-analysis determined that there was a mean effect size in the moderate range for attention (a constituent component of working memory) across 10 studies comparing neurocognitive performance in BPD and control groups (Ruocco, 2005). However, the neurocognitive deficits that have been found in BPD are often similar to those found in MDD (Veiel, 1997; Zakzanis et al., 1998), making it difficult to determine whether their neurocognitive profiles are distinct. In addition, Fertuck et al. (2006a) noted that studies which have not found neurocognitive differences between BPD and control groups were more methodologically rigorous than studies detecting significant differences. For example, studies which did not find differences tended to control for the confounding influence of other variables, such as IQ.

One possible explanation for the lack of consensus in this area is that very few studies have accounted for the role of distress in neuropsychological functioning in BPD, given that cognitive performance in BPD may owe more to the impact of transient mood states than to underlying cognitive deficits per se. As discussed in Chapter I, Fertuck et al. (2006b) incorporated distress ratings in their neurocognitive comparisons of depressed participants with and without BPD, and found that controlling for anxiety resulted in *superior* neurocognitive performance for the BPD group in several domains (attention, psychomotor speed, and general

IQ). This is strikingly consistent with the current finding that controlling for distress revealed superior working memory performance for BPD participants and poorer working memory performance for MDD participants, although there were some slight differences. For example, vigor (in an inverse relationship) was the strongest POMS predictor of working memory in our sample, whereas the Fertuck et al. (2006b) study implicated the tension/anxiety subscale. Their study also assessed multiple neurocognitive domains and found that higher-order cognitive functions, such as working memory, were *not* affected by controlling for tension. Attention is a necessary functional component of working memory, but working memory is a more complex process that also requires short-term manipulation and retrieval of information to produce a desired outcome.

It is noteworthy that in the current study, testing participants' working memory performance under stressful conditions – a study design in which distress was 'built in' to the working memory task – was not sufficient to account for the impact of distress in its entirety. Statistical controls were required to equalize levels of distress across groups, thereby allowing neurocognitive differences to emerge. In light of Fertuck et al.'s (2006b) finding that controlling for anxiety revealed superior performance in BPD, it is more likely that simple differences in working memory performance would have been found in this sample had distress levels been comparable across clinical groups. Had levels of distress been roughly equivalent across diagnostic groups, one would not have needed to control for them statistically. Therefore, one can infer that levels of distress must have differed significantly across diagnostic groups. Given that baseline distress was greater among BPD and MDD participants compared to controls, but not significantly different between BPD and MDD groups, it is reasonable to surmise that differences in distress between the two clinical groups emerged post-TSST, indicating unique

stress responses in BPD compared to MDD. Comparing post-TSST levels of distress across the diagnostic groups was beyond the scope of this study, but future research should aim to clarify the degree and type of distress experienced by BPD participants compared to MDD participants and controls.

In light of the fact that major depression is associated with impairments in attention (Gorlyn et al., 2006) – a constituent component of working memory – it is noteworthy that the BPD group in the current sample had higher levels of clinician-rated depression than the MDD group, yet outperformed the MDD group when all participants were equally distressed. Fertuck et al. (2006b) previously commented on this phenomenon and suggested that "depression-like deficits in BPD participants may be more closely related to characteristic mood fluctuations than to the effects of depression per se" (p. 65). The findings of the current study may indicate that emotional reactivity or mood fluctuations are more important than inherent deficits in accounting for neurocognitive performance in BPD, particularly in light of the fact that clear neurocognitive impairments have not emerged in the literature.

The poorer working memory performance observed in the MDD group could also be accounted for by differing presentations of depressive symptoms between the two groups. A number of researchers have noted qualitative differences in the nature of depressive symptoms experienced by individuals with BPD and MDD. For instance, depression in BPD is more likely to be characterized by mood reactivity and interpersonal sensitivity than in MDD (Fertuck et al., 2006b; Stanley & Wilson, 2006). In contrast, depressed individuals with MDD are more likely than those with BPD to have melancholic features (Bellodi, Battaglia, Gasperni, Scherillo, & Brancato, 1992), a symptom cluster which includes anhedonia and *lack* of mood reactivity.

Melancholic features of depression are also commonly associated with cognitive deficits (Austin et al., 1999), which may help to explain the MDD group's poorer performance.

In addition, psychologists have long speculated that the relationship between anxiety and performance is curvilinear, wherein increasing anxiety improves performance until a tipping point occurs and continued elevations in anxiety result in deteriorated performance (Yerkes & Dodson, 1908). This relationship has been empirically supported through the examination of cortisol. Mildly elevated glucocorticoid levels are associated with optimal consolidation of long-term memories, but excessive glucocorticoid levels impair this process (Diamond, Campbell, Park, Halonen, & Zoladz, 2007). Lack of mood reactivity in the MDD group, along with characteristic features of anhedonia and amotivation, could mean that these individuals are less likely to experience the beneficial effects of mild stress on performance. Thus, depressive symptoms – particularly the melancholic type – may have a more disabling impact in uncomplicated MDD than in BPD. The MDD participants in this sample could also have had more difficulty inhibiting their attention to negative emotions experienced during the stress procedure. Individuals with MDD have been shown to orient their attention excessively toward negative emotional cues (Elliot et al., 2011; Rinck & Becker, 2005) and to show better recall of information related to negative emotions (Bradley, Mogg, & Millar, 1996; Bradley, Mogg, & Williams, 1995; Direnfeld & Roberts, 2006; Gotlib et al., 2004).

It is also possible that the superior performance observed for BPD participants does not represent a deficit in the MDD group, but rather a relative strength in the BPD group. While BPD is a disorder associated with a wide range of functional impairments, individuals with the disorder have been shown to demonstrate particular strengths compared to controls in certain key areas. Most notable among these findings is research demonstrating that individuals with BPD

may have superior theory of mind or empathic capacities (Franzen et al., 2011; Gardner et al., 2010; Lynch et al., 2006; Merkl et al., 2010; Wagner & Linehan, 1999) despite their high degree of interpersonal dysfunction (Stanley & Siever, 2010). This has been termed the "borderline empathy" paradox (Dinsdale & Crespi, 2013; Krohn, 1974). Researchers have only recently begun to account for this paradox conceptually (Fertuck et al., 2009). BPD individuals' enhanced sensitivity to the mental states of others may be related to their hypervigilance for threats and signs of danger (Sieswarda, Arntz, Mertens, & Vertommen, 2007; Arntz et al., 2000), particularly in terms of anticipated social rejection and abandonment (Ayduk et al., 2008). This hypervigilance may be associated with heightened activity in the amygdala of individuals with BPD, resulting in attribution of negative or hostile intent when processing neutral facial stimuli (Donegan et al., 2003; Minzenberg et al., 2007).

In light of the current study's finding that BPD participants outperformed MDD participants when equivalently distressed, it may be that BPD individuals' greater attentiveness to their environments confers an advantage when a performance-based task coincides with neutral interpersonal stimuli (i.e., study confederates' neutral affect), mobilizing even greater attentiveness and vigilance. This could also help to explain why vigor was the best predictor of working memory performance in the current sample: BPD participants' attention – and therefore working memory – may have been activated and improved by the interpersonally stressful nature of the task. In addition, sex differences in theory of mind have been observed, with females being noted to hold an advantage in this domain (i.e., Geary, 2010). Given the preponderance of female participants in this sample, it is possible that participants in the current study were particularly attentive to social cues in the testing environment.

Superior working memory performance alongside heightened distress among BPD individuals may also pertain to a phenomenon known as 'apparent competence' (Linehan, 1993). This is a clinical presentation in which individuals with BPD may appear outwardly to be competent, while experiencing intense inner distress and turmoil. It also refers to the ability of BPD individuals to show effective coping skills in circumscribed and structured situations, but to 'fall apart' when structure is diminished or when circumstances change.

Of course, greater attunement and sensitivity to the mental states of others may also play a key role in impaired interpersonal functioning in BPD. Individuals with BPD may be more accurate in categorizing others' mental states, but also show biased attributions of hostile intent based on their anticipation of abandonment and rejection (Ayduk et al., 2008). Further, they may show greater rigidity in clinging to these negative interpretations of others' intentions and are unable to alter or adjust these appraisals by incorporating new information, resulting in distorted perceptions of others (Fertuck et al., 2009). This complex interpersonal process may help to account for the 'borderline empathy paradox.'

In addition, given their greater emotional sensitivity in a variety of situations, BPD participants may be more accustomed to the routine experience of stress and may be more habituated or acclimated to the experience of stress than individuals with uncomplicated depression. In situations where the degree of distress is equivalent across both groups (as in our statistical control of this variable), the impact of distress on cognitive performance may therefore be more disabling for individuals with MDD compared to those with BPD because it is more novel.

**Strengths and limitations**

This study used an experimentally manipulated, in-vivo social stressor to measure working memory capacities under stress. Experimental manipulations allow for a high degree of precision and internal validity, making it possible to pinpoint ostensible causes for participants' rise in distress after the stress procedure – the TSST is known to elevate stress because it raises concerns about negative social evaluation, and because it is experienced as uncontrollable by participants (Dickerson & Kemeny, 2004; Gruenewald et al., 2004). In the existing literature, moreover, there has been insufficient attention devoted to the impact of comorbid depression on neuropsychological performance in BPD. By assessing for the prevalence of depression and by including groups of BPD and MDD-only participants, the current study was able to identify key differences in these populations.

This study had several limitations. First, sample sizes in the MDD and control groups were relatively small, limiting statistical power and increasing the likelihood of making a Type II error. Moreover, the sample size of the BPD group was approximately three times larger than the MDD or control groups. Unequal sample sizes in regression analyses with categorical predictors can reduce the likelihood of independence of predictors (Keppel & Wickens, 2004), violating one of the traditional assumptions of linear regression. However, the independence of variances among the predictor variables in this study helps to alleviate this concern. Second, laboratory findings are often limited in generalizability. The TSST was a highly controlled procedure in which the stressor was circumscribed and time-limited, and the demands of the task were relatively known and quantifiable. In addition, the fact that the sample in the current study was highly educated, predominantly female, and predominantly white may limit the generalizability of these findings. Generalizability is limited further by the fact that participants were excluded

from analysis if they stopped performing the working memory task. Analyses revealed that these participants had higher levels of baseline anxiety than those who completed the task, suggesting that this was a more severely ill group. Thus, the results of the current study may generalize more appropriately to clinical populations that are mildly to moderately ill.

Third, interpreting results of the PASAT administered under stressful conditions is made difficult by the fact that the PASAT was not normed in this manner. As discussed in Chapter II, the PASAT was originally designed as a neuropsychological measure of working memory and was only later developed for use as a laboratory stressor. This was the first study to combine both uses of the PASAT and to treat the measure as a dependent variable in order to examine working memory performance under stress. In addition, the PASAT was not normed in clinical populations and is actually contraindicated for use as a neuropsychological assessment in clinical populations, due to its difficult and highly aversive nature (Tombaugh, 2006). Although it was inferred that emotional distress impacted working memory, moreover, the fact that working memory was not tested before and after the stress procedure limits our ability to draw this conclusion. Additionally, no other measures of working memory were used in this study to assess this ability.

The PASAT has also been subjected to a number of revisions in different studies, such that the number of items and length of ISIs have varied. For these reasons, it is difficult to interpret the high degree of variance found in PASAT scores in this study, although previous studies have found wider variances in neuropsychological measures among BPD participants than among controls (Beblo et al., 2006). The wide range of PASAT scores could also have obscured additional meaningful differences across diagnostic groups, and could have washed out a statistical interaction between diagnosis and distress in predicting working memory. Large

variances in PASAT scores could be due to great individual differences in general working memory ability. Given working memory's association with IQ (e.g., Salthouse & Pink, 2008), it would have been helpful to know whether there was a wide range of intellectual abilities in this sample, and whether intelligence varied systematically across diagnoses.

**Conclusions**

This study was the first to directly measure BPD individuals' working memory capacities under stressful conditions using an experimentally manipulated, in-vivo social stressor. Findings revealed no neurocognitive deficits for BPD participants, and instead found that controlling for participants' distress revealed superior performance for BPD participants and poorer performance for MDD participants. These findings underscore the need to account for emotional distress when examining working memory in BPD and MDD groups. Mood fluctuations and emotional reactivity may play a larger role than pathophysiological factors in characterizing neurocognitive performance in these groups. These findings also underscore potential phenomenological differences in the presentation of depression in BPD compared to MDD, even when both groups self-report equivalent severity of symptoms (as was found in the current study). In addition, BPD individuals' greater attunement and sensitivity to others' emotional states may paradoxically confer an advantage when pure attentiveness and concentration are called for.

**Future directions**

Although it was beyond the scope of the current study to compare the magnitude of change in distress scores across diagnostic groups, future studies should seek to determine whether changes in distress are more strongly associated with working memory performance in BPD compared to MDD or healthy controls. Research in this area should also examine the

magnitude of change in working memory before and after stressful procedures, as well as changes across working memory trials of varying difficulty. Biological features of stress response, such as cortisol and heart rate, should also be examined for their ability to predict neurocognitive performance under stress. In addition, future research should aim to clarify the degree and type of distress experienced by BPD participants compared to MDD participants and controls. Different types of triggers may result in differing patterns of dysregulation among individuals with BPD, and future research should aim to identify such patterns. Future research should identify other circumstances in which heightened sensitivity is advantageous to individuals with BPD, and should continue to develop theoretical models (e.g., Fertuck et al., 2009) of the complex ways in which interpersonal sensitivity both helps and hinders relational functioning in this population.

Identifying correlates and predictors of treatment outcome among individuals with BPD is of critical importance, given the traditionally high dropout rate in this population (McMurran, Huband, & Overton, 2010) and the development of psychosocial treatment models like DBT which were specifically designed to target attrition, as well as therapist burnout (Linehan, 1993). Interestingly, greater executive control and visual memory have been noted to predict treatment completion in BPD (Fertuck et al., 2011). This finding points to the critical role of patients' executive functioning in enabling them to make non-impulsive treatment decisions that are guided by sound reasoning rather than transient emotional states. Future research should also aim to identify psychological and neurocognitive strengths among individuals with BPD. Given the equivocal and complex findings on neurocognitive performance in BPD to date, more research is needed to develop a clear profile.

REFERENCES

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions.* Newbury Park, CA: Sage.

American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Anderson, A. K., Christoff, K., Panitz, D., De Rosa, E., & Gabrieli, J. D. E. (2003). Neural correlates of the automatic processing of threat facial signals. *The Journal of Neuroscience, 23,* 5627–5633.

Arntz, A., Appels, C., & Sieswerda, S. (2000). Hypervigilance in borderline disorder: A test with the emotional Stroop paradigm. *Journal of Personality Disorders, 14*, 366-373. doi:10.1521/pedi.2000.14.4.366

Austin, M. P., Mitchell, P., Wilheim, K., Parker, G., Hickie, I., Brodaty, H., . . . Hadzi-Pavlovic, D. (1999). Cognitive function in depression: A distinct pattern of frontal impairment in melancholia? *Psychological Medicine, 29,* 73-85. doi:10.1017/S0033291798007788

Ayduk, O., Zayas, V., Downey, G., Cole, A. B., Shoda, Y., & Mischel, W. (2008). Rejection sensitivity and executive control: joint predictors of borderline personality features. *Journal of Research in Personality, 42,* 151-168. doi:10.1016/j.jrp.2007.04.002

Baddeley, A. (1992). Working memory. *Science, 255,* 556–559. doi:10.1126/science.1736359

Bagby, R. M., Ryder, A. G., Schuller, D. R., & Marshall, M. B. (2004). The Hamilton Depression Rating Scale: Has the gold standard become a lead weight? *American Journal of Psychiatry, 161,* 2163–2177. doi:10.1176/appi.ajp.161.12.2163

Bandura, A., Caprara, G. V., Barbaranelli, C., Gerbino, M., & Pastorelli, C. (2003). Role of affective self-regulatory efficacy in diverse spheres of psychosocial functioning. *Child Development, 74,* 769–782. doi:10.1111/1467-8624.00567

Baron, R. M., & Kenny, D. A. (1986). The moderator-mediator variable distinction in social psychological research: Conceptual, strategic, and statistical considerations. *Journal of Personality and Social Psychology, 51,* 1173–1182. doi:10.1037/0022-3514.51.6.1173

Bate, A. J., Mathias, J. L., & Crawford, J. R. (2001). Performance on the Test of Everyday Attention and standard tests of attention following severe traumatic brain injury. *The Clinical Neuropsychologist, 15,* 405-422. doi:10.1076/clin.15.3.405.10279

Bazanis, E., Rogers, R. D., Dowson, J. H., Taylor, P., Meux, C., Staley, C., . . . Sahakian, B. J. (2002). Neurocognitive deficits in decision-making and planning of patients with DSM-

III-R borderline personality disorder. *Psychological Medicine*, *32*, 1395–1405. doi:10.1017/S0033291702006657

Beblo, T., Mensebach, C., Wingenfeld, K., Rullkoetter, N., & Schlosser, N. (2011). Patients with borderline personality disorder and major depressive disorder are not distinguishable by their neuropsychological performance: a case-control study. *The primary care companion to CNS disorders*, *13*. doi:10.4088/PCC.10m00982blu

Beblo, T., Saavedra, A. S., Mensebach, C., Lange, W., Markowitsch, H. J., Rau, H., . . . Driessen, M. (2006). Deficits in visual functions and neuropsychological inconsistency in Borderline Personality Disorder. *Psychiatry Research*, *145*, 127-135. doi:10.1016/j.psychres.2006.01.017

Beck, A. T. (2006). Hopelessness as a predictor of eventual suicide. *Annals of the New York Academy of Sciences, 487,* 90-96. doi:10.1111/j.1749-6632.1986.tb27888.x

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). Manual for Beck Depression Inventory II (BDI-II). San Antonio, TX: Psychology Corporation.

Beck, A. T., Steer, R. A., & Carbin, M. G. (1988). Psychometric properties of the Beck Depression Inventory: Twenty-five years of evaluation. *Clinical Psychology Review, 8,* 77–100. doi:10.1016/0272-7358(88)90050-5

Beck, A. T., Steer, R. A., Kovacs, M., & Garrison, B. Hopelessness and eventual suicide: a 10-year prospective study of patients hospitalized with suicidal ideation. *American Journal of Psychiatry, 142*, 559-563.

Beck, A. T., Weissman, A., Lester, D., & Trexler, L. (1974). The measurement of pessimism: the hopelessness scale. *Journal of consulting and clinical psychology, 42,* 861-865. doi:10.1037/h0037562

Beer, J. S., Shimamura, A. P., & Knight, R. T. (2004). Frontal lobe contributions to executive control of cognitive and social behavior. In M. S. Gazzaniga (Ed.), *The Newest Cognitive Neurosciences* (3rd ed., pp. 1091–1104). Cambridge, MA: MIT Press.

Bellodi, L., Battaglia, M., Gasperni, M., Scherillo, P., & Brancato, V. (1992). The nature of depression in borderline personality disorder. *Comprehensive Psychiatry, 33,* 128-133. doi:10.1016/0010-440X(92)90010-N

Benedict, R. H. B., Fischer, J. S., Archibald, C. J., Arnett, P. A., Beatty,W.W., Bobholz, J. B., . . . Munschauer, F. (2002). Minimal neuropsychological assessment of MS patients: A consensus approach. *The Clinical Neuropsychologist, 16,* 381–397. doi:10.1076/clin.16.3.381.13859

Berenson, K. R., Gyurak, A., Ayduk, O., Downey, G., Garner, M. J., Mogg, K., . . . Pine, D. S. (2009). Rejection sensitivity and disruption of attention by social threat cues. *Journal of Research in Personality, 43,* 1064-1072. doi:10.1016/j.jrp.2009.07.007

Black, D. W., Forbush, K. T., Langer, A., Shaw, M., Graeber, M. A., Moser, D. J., . . . Blum, N. (2009). The neuropsychology of borderline personality disorder: A preliminary study on the predictive variance of neuropsychological tests vs. personality trait dimensions. *Personality and Mental Health, 3,* 128-141. doi:10.1002/pmh.63

Bohnen, N., Houx, P., Nicolson, N., & Jolles, J. (1990). Cortisol reactivity and cognitive performance in a continuous mental task paradigm. *Biological Psychology, 31,* 107–116. doi:10.1016/0301-0511(90)90011-K

Bohus, M., Schmahl, C., & Lieb, K. (2004). New developments in the neurobiology of borderline personality disorder. *Current Psychiatry Reports, 6,* 43–50. doi:10.1007/s11920-004-0038-4

Bradley, B. P., Mogg, K., & Millar, N. (1996). Implicit memory bias in clinical and non-clinical depression. *Behaviour Research and Therapy, 34,* 865-879. doi:10.1016/S0005-7967(96)00074-5

Bradley, B. P., Mogg, K., & Williams, R. (1995). Implicit and explicit memory for emotion-congruent information in clinical depression and anxiety. *Behaviour Research and Therapy, 33,* 755-770. doi:10.1016/0005-7967(95)00029-W

Brittain, J. L., LaMarche, J. A., Reeder, K. P., Roth, D. L., & Boll, T. J. (1991). Effects of age and IQ on Paced Auditory Serial Addition Task (PASAT) performance. *The Clinical Neuropsychologist, 5,* 163-175. doi:10.1080/13854049108403300

Brodsky, B. S., Groves, S. A., Oquendo, M. A., Mann, J. J., & Stanley, B. (2006). Interpersonal precipitants and suicide attempts in borderline personality disorder. *Suicide and life-threatening behavior, 36,* 313-322. doi:10.1521/suli.2006.36.3.313

Brown, J. D. (1996). *Testing in language programs.* Upper Saddle River, NJ: Prentice Hall.

Brown, G. L., Goodwin, F. K., Ballenger, J. C., Goyer, P. F., & Major, L. F. (1979). Aggression in humans correlates with cerebrospinal fluid amine metabolites. *Psychiatry Research, 1,* 131-139. doi:10.1016/0165-1781(79)90053-2

de Bruijn, E. R., Grootens, K. P., Verkes, R. J., Buchholz, V., Hummelen, J. W., & Hulstijn, W. (2006). Neural correlates of impulsive responding in borderline personality disorder: ERP evidence for reduced action monitoring. *Journal of Psychiatric Research, 40,* 428-437. doi:10.1016/j.jpsychires.2005.09.004

Burke, H. M., Davis, M. C., Otte, C., & Mohr, D. C. (2005). Depression and cortisol responses to psychological stress: a meta-analysis. *Psychoneuroendocrinology, 30,* 846–856. doi:10.1016/j.psyneuen.2005.02.010

Carroll, D., Phillips, A. C., Hunt, K., & Der, G. (2007). Symptoms of depression and cardiovascular reactions to acute psychological stress: Evidence from a population study. *Biological Psychology, 75,* 68–74. doi:10.1016/j.biopsycho.2006.12.002

Chapman, A. L., Leung, D. W., & Lynch, T. R. (2008). Impulsivity and emotion dysregulation in borderline personality disorder. *Journal of Personality Disorders, 22,* 148-164. doi:10.1521/pedi.2008.22.2.148

Cicerone, K. D. (1997). Clinical sensitivity of four measures of attention to mild traumatic brain injury. *The Clinical Neuropsychologist, 11,* 266-272. doi:10.1080/13854049708400455

Comtois, K. A., Cowley, D. S., Dunner, D. L., & Roy-Burne, R. P. (1999). Relationship between borderline personality disorder and Axis I diagnosis in severity of depression and anxiety. *The Journal of Clinical Psychiatry, 60,* 752-758. doi:10.4088/JCP.v60n1106

Corbitt, E. M., Malone, K. M., Haas, G. L., & Mann, J. J. (1996). Suicidal behavior in patients with major depression and comorbid personality disorders. *Journal of Affective Disorders, 39,* 61–72. doi:10.1016/0165-0327(96)00023-7

Cornelius, J. R., George, A. W. A., Schulz, C., Schulz, P. M., Soloff, P. H., & Tarter, R. (1989). An evaluation of the significance of selected neuropsychiatric abnormalities in the etiology of borderline personality disorder. *Journal of Personality Disorders, 3,* 19-24. doi:10.1521/pedi.1989.3.1.19

Crawford, J. R., Obonsawin, M. C., & Allan, K. M. (1998). PASAT and components of WAIS-R performance: Convergent and discriminant validity. *Neuropsychological Rehabilitation, 8,* 255–272. 10.1080/713755575

Crowell, S. E., Beauchaine, T. P., & Linehan, M. M. (2009). A biosocial developmental model of borderline personality: Elaborating and extending Linehan's theory. *Psychological Bulletin, 135,* 495–510. doi:10.1037/a0015616

Davidson, R. J. (2000). Affective style, psychopathology, and resilience: Brain mechanisms and plasticity. *American Psychologist, 55,* 1196–1214. doi:10.1037/0003-066X.55.11.1196

Deary, I. J., Langan, S. J., Hepburn, D. A., & Frier, B. M. (1991). Which abilities does the PASAT test? *Personality and Individual Differences, 12,* 983–987. doi:10.1016/0191-8869(91)90027-9

Diamond, D. M., Campbell, A. M., Park, C. R., Halonen, J., & Zoladz, P. R. (2007). The temporal dynamics model of emotional memory processing: a synthesis on the neurobiological basis of stress-induced amnesia, flashbulb and traumatic memories, and

the Yerkes-Dodson law. *Neural Plasticity*, Article ID 60803, 1-33. doi:10.1155/2007/60803

Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: a theoretical integration and synthesis of laboratory research. *Psychological Bulletin, 130,* 355–391. 10.1037/0033-2909.130.3.355

Diehr, M. C., Cherner, M., Wolfson, T. J., Miller, S. W., Grant, I., Heaton, R. K., & the HIV Neurobehavioral Research Center Group. (2003). The 50 and 100-item short forms of the Paced Auditory Serial Addition Task (PASAT): Demographically corrected norms and comparisons with the full PASAT in normal and clinical samples. *Journal of Clinical and Experimental Neuropsychology, 25*, 571–585. doi:10.1076/jcen.25.4.571.13876

Diehr, M. C., Heaton, R. K., Miller, W., & Grant, I. (1998). The Paced Auditory Serial Addition Task (PASAT): Norms For age, education, and ethnicity. *Assessment, 5,* 375-387. doi:10.1177/107319119800500407

Dinn, W. M., Harris, C. L., Aycicegi, A., Greene, P. B., Kirkley, S. M., & Reilly, C. (2004). Neurocognitive function in borderline personality disorder. *Progress in Neuro-Psychopharmacology and Biological Psychiatry, 28,* 329-341. doi:10.1016/j.pnpbp.2003.10.012

Dinsdale, N., & Crespi, B. J. (2013). The borderline empathy paradox: Evidence and conceptual models for empathic enhancements in borderline personality disorder. *Journal of Personality Disorders, 27,* 172-195. doi:10.1521/pedi_2012_26_071

Direnfeld, D. M., & Roberts, J. E. (2006). Mood congruent memory in dysphoria: The roles of state affect and cognitive style. *Behaviour Research and Therapy, 44*, 1275-1285. doi:10.1016/j.brat.2005.03.014

Dolcos, F., & McCarthy, G. (2006). Brain systems mediating cognitive interference by emotional distraction. *The Journal of Neuroscience, 26,* 2072–9. doi:10.1523/JNEUROSCI.5042-05.2006

Domes, G., Winter, B., Schnell, K., Vohs, K., Fast, K., & Herpertz, S. C. (2006). The influence of emotions on inhibitory functioning in borderline personality disorder. *Psychological Medicine, 36,* 1163–1172. doi:10.1017/S0033291706007756

Donegan, N. H., Sanislow, C. A., Blumberg, H. P., Fulbright, R. K., Lacadie, C., Skudlarski, P., . . . Wexler, B.E. (2003). Amygdala hyperreactivity in borderline personality disorder: implications for emotional dysregulation. *Biological Psychiatry, 54,* 1284–1293. doi:10.1016/S0006-3223(03)00636-X

Driessen, M., Herrmann, J., Stahl, K., Zwaan, M., Meier, S., Hill, A., . . . Petersen, D. (2000). Magnetic resonance imaging volumes of the hippocampus and the amygdala in women

with borderline personality disorder and early traumatization. *Archives of General Psychiatry, 57*, 1115-1122. doi:10.1001/archpsyc.57.12.1115

Dyce, J. A. (1996). Factor structure of the Beck Hopelessness Scale. *Journal of Clinical Psychology, 52*, 555-558. doi:10.1002/(SICI)1097-4679(199609)52:5<555::AID-JCLP10>3.0.CO;2-D

Eisenberg, N., & Fabes, R. A. (1992). Emotion, regulation, and the development of social competence. In M. S. Clark (Ed.), *Emotion and Social Behavior* (Vol. 14, pp. 119–150). Thousand Oaks, CA: Sage Publications, Inc.

Eisenberg, N., Fabes, R. A., Guthrie, I. K., & Reiser, M. (2000). Dispositional emotionality and regulation: their role in predicting quality of social functioning. *Journal of Personality and Social Psychology, 78*, 136–157. doi:10.1037/0022-3514.78.1.136

Elliott, R., Zahn, R., Deakin, J. W., & Anderson, I. M. (2011). Affective cognition and its disruption in mood disorders. *Neuropsychopharmacology*, *36*, 153-182. doi:10.1038/npp.2010.77

Fertuck, E. A., Jekal, A., Song, I., Wyman, B., Morris, M. C., Wilson, S. T., . . . Stanley, B. (2009). Enhanced "Reading the Mind in the Eyes" in borderline personality disorder compared to healthy controls. *Psychological Medicine, 39*, 1979-1988. doi:10.1017/S003329170900600X

Fertuck, E. A., Keilp, J. K., Song, I., Morris, M. C., Wilson, S. T., Brodsky, B. S., & Stanley, B. (2011). Higher executive control and visual memory performance predict treatment completion in borderline personality disorder. *Psychotherapy and Psychosomatics, 81*, 38-43. doi:10.1159/000329700

Fertuck, E. A., Lenzenweger, M. F., Clarkin, J. F., Hoermann, S., & Stanley, B. (2006a). Executive neurocognition, memory systems, and borderline personality disorder. *Clinical Psychology Review, 26*, 346–375. doi:10.1016/j.cpr.2005.05.008

Fertuck, E. A., Marsano-Jozefowicz, S., Stanley, B., Tryon, W. W., Oquendo, M., Mann, J. J., & Keilp, J. G. (2006b). The impact of borderline personality disorder and anxiety on neuropsychological performance in major depression. *Journal of Personality Disorders, 20*, 55–70. doi:10.1521/pedi.2006.20.1.55

Feske, U., Mulsant, B. H., Pilkonis, P. A., Soloff, P., Dolata, D., Sackeim, H. A., & Haskett, R. F. (2004). Clinical outcome of ECT in patients with major depression and comorbid borderline personality disorder. *American Journal of Psychiatry. 161*, 2073-2080. doi:10.1176/appi.ajp.161.11.2073

First, M. B., Gibbon, M., Spitzer, R. L., & Williams, J. B. W. (1996). Structured Clinical Interview for DSM-IV Axis I Disorders (SCID-I). New York: Biometrics Research Department, New York State Psychiatric Institute.

First, M. B., Gibbon, M., Spitzer, R. L., Williams, J. B. W., & Benjamin, L. S. (1997). Structured Clinical Interview for DSM-IV axis II personality disorders (SCID-II). Washington, DC: American Psychiatric Press, Inc.

Fisk, J. D., & Archibald, C. J. (2001). Limitations of the Paced Auditory Serial Addition Test as a measure of working memory in patients with multiple sclerosis. *Journal of the International Neuropsychological Society, 7,* 363-372. doi:10.1017/S1355617701733103

Fos, L. A., Greve, K.W., South, M. B., Mathias, C., & Benefield, H. (2000). Paced Visual Serial Addition Test: An alternative measure of information processing speed. *Applied Neuropsychology, 7,* 140–146. doi:10.1207/S15324826AN0703_4

Franzen, N., Hagenhoff, M., Baer, N., Schmidt, A., Mier, D., Sammer, G., . . . Lis, S. (2011). Superior 'theory of mind' in borderline personality disorder: An analysis of interaction behavior in a virtual trust game. *Psychiatry Research, 187,* 224-233. doi:10.1016/j.psychres.2010.11.012

Fritz, M. S., & MacKinnon, D. P. (2007). Required sample size to detect the mediated effect. *Psychological Science, 18,* 233-239. doi:10.1111/j.1467-9280.2007.01882.x

Gardner, K. J., Qualter, P., Stylianou, M., & Robinson, A. J. (2010). Facial affect recognition in non-clinical adults with borderline personality features: The role of effortful control and rejection sensitivity. *Personality and Individual Differences, 49,* 799-804. doi:10.1016/j.paid.2010.07.018

Geary, D. C. (2010). *Male, female: The evolution of human sex differences* (2nd ed.). Washington, DC: American Psychological Association. doi:10.1037/12072-000

Gorlyn, M., Keilp, J. G., Oquendo, M. A., Burke, A. K., Sackeim, H. A., & Mann, J J. (2006). WAIS-III performance in major depression: Absence of VIQ/PIQ differences. *Journal of Clinical and Experimental Neuropsychology, 28,* 1145-1157. doi:10.1080/13803390500246944

Gotlib, I. H., Kasch, K. L., Traill, S., Joormann, J., Arnow, B. A., & Johnson, S. L. (2004). Coherence and specificity of information-processing biases in depression and social phobia. *Journal of Abnormal Psychology*, *113*, 386. doi:10.1037/0021-843X.113.3.386

Grant, B. F., Chou, S.P., Goldstein, R.B., Huang, B., Stinson, F. S., Saha, T. D., . . . Ruan, W.J. (2008). Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry, 69,* 533-545. doi:10.4088/JCP.v69n0404

Gratz, K. L., Rosenthal, M. Z., Tull, M. T., Lejuez, C. W., & Gunderson, J. G. (2006). An experimental investigation of emotion dysregulation in borderline personality disorder. *Journal of Abnormal Psychology, 115,* 850–855. doi:10.1037/0021-843X.115.4.850

Gronwall, D. M. A. (1977). Paced auditory serial-addition task: a measure of recovery from concussion. *Perceptual and Motor Skills, 44,* 367–373. doi:10.2466/pms.1977.44.2.367

Gronwall, D., & Wrightson, P. (1981). Memory and information processing capacity after closed head injury. *Journal of Neurology, Neurosurgery, and Psychiatry, 44,* 889-895. doi:10.1136/jnnp.44.10.889

Gruenewald, T. L., Kemeny, M. E., Aziz, N., & Fahey, J. L. (2004). Acute threat to the social self: shame, social self-esteem, and cortisol activity. *Psychosomatic Medicine, 66,* 915–924. doi:10.1097/01.psy.0000143639.61693.ef

Gross, J. J. (1998). The emerging field of emotion regulation: An integrative review. *Review of General Psychology, 2,* 271–299. doi:10.1037/1089-2680.2.3.271

Gross, J. J. (2002). Emotion regulation: Affective, cognitive, and social consequences. *Psychophysiology, 39,* 281–291. doi:10.1017/S0048577201393198

Gross, J. J., & John, O. P. (2003). Individual differences in two emotion regulation processes: implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85,* 348–362. doi:10.1037/0022-3514.85.2.348

Gunderson, J. G., Morey, L. C., Stout, R. L., Skodol, A. E., Shea, M. T., McGlashan, T. H., . . . Bender, D. S. (2004). Major depressive disorder and borderline personality disorder revisited: longitudinal interactions. *Journal of Clinical Psychiatry*, *65,* 1049-1056. doi:10.4088/JCP.v65n0804

Haaland, V. Ø., & Landrø, N. I. (2007). Decision making as measured with the Iowa Gambling Task in patients with borderline personality disorder. *Journal of the International Neuropsychological Society, 13,* 699-703. doi:10.1017/S1355617707070890

Hagenhoff, M., Franzen, N., Koppe, G., Baer, N., Scheibel, N., Sammer, G., . . . Lis, S. (2013). Executive functions in borderline personality disorder. *Psychiatry Research, 210,* 224-231. doi:10.1016/j.psychres.2013.05.016

Hamilton, M. (1959). The assessment of anxiety states by rating. *British Journal of Medical Psychology, 32,* 50–55. doi:10.1111/j.2044-8341.1959.tb00467.x

Hamilton, M. (1960). A rating scale for depression. *Journal of Neurology, Neurosurgery, and Psychiatry, 23,* 56-62. doi:10.1136/jnnp.23.1.56

Harris, C. L., Dinn, W. M., & Marcinkiewicz, J. A. (2002). Partial seizure-like symptoms in borderline personality disorder. *Epilepsy & Behavior*, *3*, 433-438. doi:10.1016/S1525-5050(02)00521-8

Haslam, C., Batchelor, J., Fearnside, M. R., Haslam, S. A., & Hawkins, S. (1995). Further examination of post-traumatic amnesia and post-coma disturbance as non-linear predictors of outcome after head injury. *Neuropsychology, 9,* 599-605. doi:10.1037/0894-4105.9.4.599

Hayes, A. F., & Preacher, K. J. (2013). Statistical mediation analysis with a multicategorical independent variable. *British Journal of Mathematical and Statistical Psychology*. Advance online publication. doi:10.1111/bmsp.12028

Herpertz, S. C. (2003). Emotional processing in personality disorder. *Current psychiatry reports*, *5*, 23-27. doi:10.1007/s11920-003-0005-5

Herpertz, S. C., Dietrich, T. M., Wenninga, B., Krings, T., Erberich, S. G., Willmes, K., . . . Sass, H. (2001). Evidence of abnormal amygdala functioning in borderline personality disorder: a functional MRI study. *Biological Psychiatry, 50,* 292–298. doi:10.1016/S0006-3223(01)01075-7

Hilsenroth, M. J., Ackerman, S. J., Blagys, M. D., Baumann, B. D., Baity, M. R., Smith, S. R., Price, J. L., . . . Holdwick, D. J. (2000). Reliability and validity of DSM-IV axis V. *American Journal of Psychiatry, 157,* 1858–1863. doi:10.1176/appi.ajp.157.11.1858

Holdwick, D. J., & Wingenfeld, S. A. (1999). The subjective experience of PASAT testing: Does the PASAT induce negative mood? *Archives of Clinical Neuropsychology, 14*, 273–284. doi:10.1016/S0887-6177(98)00021-3

Irle, E., Lange, C., & Sachsse, U. (2005). Reduced size and abnormal asymmetry of parietal cortex in women with borderline personality disorder. *Biological psychiatry*, *57,* 173-182. doi:10.1016/j.biopsych.2004.10.004

Iverson, G. L., Lovell, M. R., Smith, S. S. (2000). Does brief loss of consciousness affect cognitive functioning after mild head injury? Archives of Clinical Neuropsychology, 15, 643-848. doi:10.1016/S0887-6177(99)00048-7

Jogems-Kosterman, B. J. M., de Knijff, D. W. W., Kusters, R., & van Hoof, J. J. M. (2007). Basal cortisol and DHEA levels in women with borderline personality disorder. *Journal of Psychiatric Research, 41,* 1019–1026. doi:10.1016/j.jpsychires.2006.07.019

Johnson, P. A., Hurley, R. A., Benkelfat, C., Herpertz, S. C., & Taber, K. H. (2003). Understanding emotion regulation in borderline personality disorder: contributions of neuroimaging. *Journal of Neuropsychiatry and Clinical Neurosciences, 15,* 397–402. doi:10.1176/appi.neuropsych.15.4.397

Joyce, R. R., Mulder, R. T., Luty, S. E., McKenzie, J. M., Sullivan, P. F., & Cloninger, R. C. (2003). Borderline personality disorder in major depression: Symptomatology, temperament, character, differential drug response, and 6-month outcome. *Comprehensive Psychiatry, 44,* 35-43. doi:10.1053/comp.2003.50001

Judd, P. H., & Ruff, R. M. (1993). Neuropsychological dysfunction in borderline personality disorder. *Journal of Personality Disorders, 7,* 275-284. doi:10.1521/pedi.1993.7.4.275

Kabacoff, R. I., Segal, D. L., Hersen, M., & Van Hasselt, V. B. (1997). Psychometric properties and diagnostic utility of the Beck Anxiety Inventory and the State-Trait Anxiety Inventory with older adult psychiatric outpatients. *Journal of Anxiety Disorders, 11,* 33-47. doi:10.1016/S0887-6185(96)00033-3

Keilp, J. G., Gorlyn, M., Cooper, T., Oquendo, M., & Mann, J. J. (2010). A modified social stress task for high-risk populations. Poster presented at the Society of Biological Psychiatry, New Orleans, LA.

Keilp, J. G., Klain, H. M., Brodsky, B., Oquendo, M. A., Gorlyn, M., Stanley, B., & Mann, J. J. (2007). Early visual information processing deficit in depression with and without Borderline Personality Disorder. *Psychiatry Research*, *149*, 139-145. doi:10.1016/j.psychres.2006.09.014

Keppel, G., & Wickens, T. D. (2004). *Design and analysis: A researcher's handbook.* Prentice Hall.

Kirschbaum, C., Pirke, K. M., & Hellhammer, D. H. (1993). The "Trier Social Stress Test": a tool for investigating psychobiological stress responses in a laboratory setting. *Neuropsychobiology, 28*, 76–81. doi:10.1159/000119004

Kobak, K. A. (2010). Hamilton Depression Rating Scale. In I. B. Weiner & W. E. Craighead (Eds.), *Corsini Encyclopedia of Psychology* (4th ed., Vol. 1, p. 748). Hoboken, NJ: Wiley. doi:10.1002/9780470479216.corpsy0402

Koenigsberg, H. W., Siever, L. J., Lee, H., Pizzarello, S., New, A. S., Goodman, M., Cheng, H., . . . Prohovnik, I. (2009). Neural correlates of emotion processing in borderline personality disorder. Psychiatry Research. *Neuroimaging, 172,* 192–199. doi:10.1016/j.pscychresns.2008.07.010

Koerner, N., Antony, M. M., & Dugas, M. J. (2010). Limitations of the Hamilton Anxiety Rating Scale as a primary outcome measure in randomized, controlled trials of treatments for generalized anxiety disorder. *American Journal of Psychiatry, 167,* 103-104. doi:10.1176/appi.ajp.2009.09091264

Korfine, L., & Hooley, J. M. (2000). Directed forgetting of emotional stimuli in borderline personality disorder. *Journal of Abnormal Psychology, 109*, 214–221. doi:10.1037/0021-843X.109.2.214

Krause-Utz, A., Oei, N. Y. L., Niedtfeld, I., Bohus, M., Spinhoven, P., Schmahl, C., & Elzinga, B. M. (2012). Influence of emotional distraction on working memory performance in borderline personality disorder. *Psychological Medicine, 42,* 2181–2192. doi:10.1017/S0033291712000153

Krohn, A. (1974). Borderline 'empathy' and differentiation of object representations: A contribution to the psychology of object relations. *International Journal of Psychoanalytic Psychotherapy, 3,* 142-165.

Kunert, H. J., Druecke, H. W., Sass, H., & Herpertz, S. C. (2003). Frontal lobe dysfunctions in borderline personality disorder? Neuropsychological findings. *Journal of Personality Disorders 17,* 497-509. doi:10.1521/pedi.17.6.497.25354

Landrø, N. I., Stiles, T. C., & Sletvold, H. (2001). Neuropsychological function in nonpsychotic unipolar major depression. *Cognitive and Behavioral Neurology, 14,* 233–240.

Larrabee, G. T., & Curtiss, G. (1995). Construct validity of various verbal and visual memory tasks. *Journal of Clinical and Experimental Neuropsychology, 17,* 536–547. doi:10.1080/01688639508405144

Lazzaretti, M., Morandotti, N., Sala, M., Isola, M., Frangou, S., De Vidovich, G., . . . Brambilla, P. (2012). Impaired working memory and normal sustained attention in borderline personality disorder. *Acta Neuropsychiatrica, 24,* 349-355. doi:10.1111/j.1601-5215.2011.00630.x

LeGris, J., Links, P. S., van Reekum, R., Tannock, R., & Toplak, M. (2012). Executive function and suicidal risk in women with Borderline Personality Disorder. *Psychiatry research, 196,* 101-108. doi:10.1016/j.psychres.2011.10.008

Lejuez, C. W., Kahler, C. W., & Brown, R. A. (2003). A modified computer version of the Paced Auditory Serial Addition Task (PASAT) as a laboratory-based stressor. *The Behavior Therapist, 26,* 290–293.

Lezak, M. D. (1995). Neuropsychology assessment (3rd ed.) New York: Oxford University Press.

Lenzenweger, M. F., Clarkin, J. F., Fertuck, E. A., & Kernberg, O. F. (2004). Executive neurocognitive functioning and neurobehavioral systems indicators in borderline personality disorder: a preliminary study. *Journal of Personality Disorders, 18,* 421–438. doi:10.1521/pedi.18.5.421.51323

Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York, NY: The Guilford Press.

Linehan, M. M., Bohus, M., & Lynch, T. R. (2007). Dialectical behavior therapy for pervasive emotion dysregulation. In J. Gross (Ed.), *Handbook of emotion regulation* (pp. 581–605). New York, NY: The Guilford Press.

Linehan, M. M., Goodstein, L. J., Nielsen, S. L., & Chiles, J. A. (1983). Reasons for staying alive when you are thinking of killing yourself: The Reasons for Living Inventory. *Journal of Consulting and Clinical Psychology, 51,* 276–286. doi:10.1037/0022-006X.51.2.276

Links, P. S., Heslegrave, R., & Reekum, R. V. (1999). Impulsivity: core aspect of borderline personality disorder. *Journal of Personality Disorders, 13,* 1-9. doi:10.1521/pedi.1999.13.1.1

Lobbestael, J., Leurgans, M., & Arntz, A. (2011). Inter-rater reliability of the structured clinical interview for DSM-IV axis I disorders (SCID I) and axis II disorders (SCID II). *Clinical Psychology & Psychotherapy, 18,* 75-79. doi:10.1002/cpp.693

Lynch, T. R., Rosenthal, M. Z., Kosson, D. S., Cheavens, J. S., Lujuez, C. W., & Blair, R. J. B. (2006). Heightened sensitivity to facial expressions of emotion in borderline personality disorder. *Emotion, 6,* 647-655. 10.1037/1528-3542.6.4.647

Macleod, D., & Prior, M. (1996). Attention deficits in adolescents with ADHD and other clinical groups. *Child Neuropsychology, 2,* 1-10. doi:10.1080/09297049608401345

Madigan, N. K., DeLuca, J., Diamond, B. J., Tramontano, G., & Averill, A. (2000). Speed of information processing in traumatic brain injury: Modality specific factors. *Journal of Head Trauma Rehabilitation, 15,* 943-956. doi:10.1097/00001199-200006000-00007

Maffei, C., Fossati, A., Agostoni, I., Barraco, A., Bagnato, M., Deborah, D., . . . Namia, C. (1997). Interrater reliability and internal consistency of the Structured Clinical Interview for DSM-IV axis II personality disorders (SCID-II), version 2.0. *Journal of Personality Disorders, 11,* 279-284. doi:10.1521/pedi.1997.11.3.279

Mann, J. J. (2002). A current perspective of suicide and attempted suicide. *Annals of Internal Medicine, 136,* 302-311. doi:10.7326/0003-4819-136-4-200202190-00010

McCaffrey, R. J., Westervelt, H.J., & Haase, R. (2001). Serial neuropsychological assessment with the National Institute of Mental Health (NIMH) AIDS abbreviated neuropsychological battery. *Archives of Clinical Neuropsychology, 16*, 9–18. doi:10.1016/S0887-6177(99)00055-4

McGlashan, T. H., Grilo, C. M., Skodol, A. E., Gunderson, J. G., Shea, M. T., Morey, L. C., . . . Stout, R. L. (2000). The Collaborative Longitudinal Personality Disorders Study: baseline Axis I/II and II/II diagnostic co-occurrence. *Acta Psychiatrica Scandinavica, 102,* 256-264. doi:10.1034/j.1600-0447.2000.102004256.x

McMurran, M., Huband, N, & Overton, E. (2010). Non-completion of personality disorder treatments: A systematic review of correlates, consequences, and interventions. *Clinical Psychology Review, 30,* 277-287. doi:10.1016/j.cpr.2009.12.002

McNair, D. M., Lohr, M., & Droppelman, L. F. (1981). Manual for the Profile of Mood States. San Diego, CA: Educational and Industrial Testing Service.

Mensebach, C., Wingenfeld, K., Driessen, M., Rullkoetter, N., Schlosser, N., Steil, C., . . . Beblo, T. (2009). Emotion-induced memory dysfunction in borderline personality disorder. *Cognitive Neuropsychiatry, 14,* 524–541. doi:10.1080/13546800903049853

Merkl, A., Ammelburg, N., Aust, S., Roepke, S., Reinecker, H., Trahms. L., . . . Sander, T. (2010). Processing of visual stimuli in borderline personality disorder: A combined behavioural and magnetoencephalographic study. *International Journal of Psychophysiology, 78,* 257-264. doi:10.1016/j.ijpsycho.2010.08.007

Minzenberg, M. J., Fan, J., New, A. S., Tang, C. Y., & Siever, L. J. (2007). Frontolimbic dysfunction in response to facial emotion in borderline personality disorder: an event-related fMRI study. *Psychiatry Research, 155,* 231-243. doi:10.1016/j.pscychresns.2007.03.006

Morfeld, M., Petersen, C., Krüger-Bödeker, A., von Mackensen, S., & Bullinger, M. (2007). The assessment of mood at workplace: psychometric analyses of the revised Profile of Mood States (POMS) questionnaire. *Psychosocial Medicine, 4,* 6-15.

Nater, U. M., Bohus, M., Abbruzzese, E., Ditzen, B., Gaab, J., Kleindienst, N., . . . Ehlert, U. (2010). Increased psychological and attenuated cortisol and alpha-amylase responses to acute psychosocial stress in female patients with borderline personality disorder. *Psychoneuroendocrinology, 35,* 1565–1572. doi:10.1016/j.psyneuen.2010.06.002,

O'Donnell, J. P., MacGregor, L. A., Dabrowski, J. J., Oestreicher, J. M., & Romero, J. J. (1994). Construct validity of neuropsychological tests of conceptual and attentional abilities. *Journal of Clinical Psychology, 50,* 596-600. doi:10.1002/1097-4679(199407)50:4<596::AID-JCLP2270500416>3.0.CO;2-S

O'Leary, K. M., Brouwers, P., Gardner, D. L., & Cowdry, R. W. (1991). Neuropsychological testing of patients with borderline personality disorder. *American Journal of Psychiatry, 148,* 106-111.

Ochsner, K. N., & Gross, J. J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9,* 242–249. doi:10.1016/j.tics.2005.03.010

Öhman, A. (2005). The role of the amygdala in human fear: Automatic detection of threat. *Psychoneuroendocrinology, 30,* 953-958. doi:10.1016/j.psyneuen.2005.03.019

Osman, A., Gifford, J., Jones, T., Lickiss, L., Osman, J., & Wenzel, R. (1993). Psychometric evaluation of the Reasons for Living Inventory. *Psychological Assessment, 5,* 154-158. doi:10.1037/1040-3590.5.2.154

Overall, J. E., & Gorham, D. R. (1962) The Brief Psychiatric Rating Scale. *Psychological Reports, 10,* 790-812. doi:10.2466/pr0.1962.10.3.799

Paelecke-Habermann, Y., Pohl, J., & Leplow, B. (2005). Attention and executive functions in remitted major depression patients. *Journal of Affective Disorders, 89*, 125-135. doi:10.1016/j.jad.2005.09.006

Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt Impulsiveness Scale. *Journal of Clinical Psychology, 51,* 768-774. doi:10.1002/1097-4679(199511)51:6<768::AID-JCLP2270510607>3.0.CO;2-1

Ponsford, J., & Kinsella, G. (1992). Attentional deficits following closed-head injury. *Journal of Clinical and Experimental Neuropsychology, 14,* 822-838. doi:10.1080/01688639208402865

Posner, M. I., Rothbart, M. K., Vizueta, N., Levy, K. N., Evans, D. E., Thomas, K. M., & Clarkin, J. F. (2002). Attentional mechanisms of borderline personality disorder. *Proceedings of the National Academy of Sciences, 99,* 16366-16370. doi:10.1073/pnas.252644699

Putnam, F. W., & Trickett, P. K. (1997). Psychobiological effects of sexual abuse. *Annals of the New York Academy of Sciences, 821,* 150–159. doi:10.1111/j.1749-6632.1997.tb48276.x

Rentrop, M., Backenstrass, M., Jaentsch, B., Kaiser, S., Roth, A., Unger, J., . . . Renneberg, B. (2007). Response inhibition in borderline personality disorder: performance in a Go/Nogo task. *Psychopathology, 41,* 50-57. doi:10.1159/000110626

Rinck, M., & Becker, E. S. (2005). A comparison of attentional biases and memory biases in women with social phobia and major depression. *Journal of Abnormal Psychology, 114*, 62. 10.1037/0021-843X.114.1.62

Rinne, T., De Kloet, E. R., Wouters, L., Goekoop, J. G., DeRijk, R. H., & van den Brink, W. (2002). Hyperresponsiveness of hypothalamic-pituitary-adrenal axis to combined dexamethasone/corticotropin-releasing hormone challenge in female borderline personality disorder participants with a history of sustained childhood abuse. *Biological Psychiatry, 52,* 1102-1112. doi:10.1016/S0006-3223(02)01395-1

Roman, D. D., Edwall, G. E., Buchanan, R. J., & Patton, J. H. (1991). Extended norms for the Paced Auditory Serial Addition Task. *The Clinical Neuropsychologist, 5*, 33–40. doi:10.1080/13854049108401840

Roemer, L. (2001). Measures for anxiety and related constructs. In M. M. Antony, S. M. Orsillo, & L. Roemer (Eds.), *Practitioner's guide to empirically based measures of anxiety* (49-83). New York, NY: Springer.

Royan, J., Tombaugh, T. N., Rees, L., & Francis, M. (2004). The Adjusting-Paced Serial Addition Test (Adjusting-PSAT): Thresholds for speed of information processing as a function of stimulus modality and problem complexity. *Archives of Clinical Neuropsychology, 19,* 131–143. doi:10.1093/arclin/19.1.131

Salthouse, T. A., & Pink, J. E. (2008). Why is working memory related to fluid intelligence? *Psychonomic Bulletin and Review, 15,* 384-371. doi:10.3758/PBR.15.2.364

Sansone, R. A., & Sansone, L. A. (2011). Gender patterns in borderline personality disorder. *Innovations in Clinical Neuroscience, 8,* 16-20.

Schnell, K., Dietrich, T., Schnitker, R., Daumann, J., & Herpertz, S. C. (2007). Processing of autobiographical memory retrieval cues in borderline personality disorder. *Journal of Affective Disorders, 97,* 253–259. doi:10.1016/j.jad.2006.05.035

Sebastian, A., Jacob, G., Lieb, K., & Tüscher, O. (2013). Impulsivity in borderline personality disorder: a matter of disturbed impulse control or a facet of emotional dysregulation? *Current Psychiatry Reports, 15,* 1-8. doi:10.1007/s11920-012-0339-y

Shear, M. K., Vander Bilt, J., Rucci, P., Endicott, J., Lydiard, B., Otto, M. W., Pollack, M. H., . . . Frank, D. M. (2001). Reliability and validity of a structured interview guide for the Hamilton Anxiety Rating Scale (SIGH-A). *Depression and anxiety, 13,* 166–178. doi: 10.1002/da.1033

Sherman, E. M. S., Strauss, E., & Spellacy, F. (1997). Validity of the Paced Auditory Serial Addition Test (PASAT) in adults referred for neuropsychological assessment after head injury. *The Clinical Neuropsychologist, 11,* 34-45. doi:10.1080/13854049708407027

Sherman, E. M. S., Strauss, E., & Spellacy, F., & Hunter, M. (1995). Construct validity of WAIS-R factors: Neuropsychological test correlates in adults referred for evaluation of possible head injury. *Psychological Assessment, 7,* 440-444. doi:10.1037/1040-3590.7.4.440

Shimamura, A. P. (2000). The role of the prefrontal cortex in dynamic filtering. *Psychobiology, 28,* 207–218.

Shoum, K. (2011). *Grief and depression following miscarriage: Examining unique and shared correlates and patterns of change at six months post-loss.* (Doctoral dissertation). Retrieved from ProQuest Dissertations & Theses Full Text. 3451524.

Shucard, J. L., Parrish, J., Shucard, D.W., McCabe, D. C., Benedict, R. H. B., & Ambrus, J. (2004).Working memory and processing speed deficits in systemic lupus erythematosus as measured by the paced auditory serial addition test. *Journal of the International Neuropsychological Society, 10,* 35–45. doi:10.1017/S1355617704101057

Skodol, A. E., & Bender, D. S. (2003). Why are women diagnosed borderline more than men? Psychiatric Quarterly, 74, 349-360. doi:10.1023/A:1026087410516

Sieswerda, S., Arntz, A., Mertens, I., & Vertommen, S. (2007). Hypervigilance in patients with borderline personality disorder: Specificity, automaticity, and predictors. *Behaviour research and therapy, 45,* 1011-1024. doi:10.1016/j.brat.2006.07.012

Silbersweig, D., Clarkin, J., Goldstein, M., Kernberg, O., Tuescher, O., Levy, K., . . . Stern, E. (2007). Failure of frontolimbic inhibitory function in the context of negative emotion in borderline personality disorder. *American Journal of Psychiatry, 164*, 1832–1841. doi:10.1176/appi.ajp.2007.06010126

Simeon, D., Knutelska, M., Smith, L., Baker, B. R., & Hollander, E. (2007). A preliminary study of cortisol and norepinephrine reactivity to psychosocial stress in borderline personality disorder with high and low dissociation. *Psychiatry Research, 149*, 177–184. doi:10.1016/j.psychres.2005.11.014

Smith, E. E., & Jonides, J. (1999). Storage and executive processes in the frontal lobes. *Science, 283*, 1657–1661. doi:10.1126/science.283.5408.1657

Söderberg, P., Tungström, S., & Armelius, B. Å. (2005). Special section on the GAF: reliability of Global Assessment of Functioning ratings made by clinical psychiatric staff. *Psychiatric Services, 56,* 434–438. doi:10.1176/appi.ps.56.4.434

Soloff, P. H., Lynch, K. G., Kelly, T. M., Malone, K. M., & Mann, J. J. (2000). Characteristics of suicide attempts of patients with major depressive episode and borderline personality disorder: a comparative study. *American Journal of Psychiatry, 157*, 601-608. doi:10.1176/appi.ajp.157.4.601

Spielberger, C. D. (1983). Manual for the State-Trait Anxiety Inventory (STAI, Form Y). Palo Alto, CA: Mind Garden.

Sprock, J., Rader, T. J., Kendall, J. P., & Yoder, C. Y. (2000). Neuropsychological functioning in patients with borderline personality disorder. *Journal of Clinical Psychology, 56*, 1587-1600. doi:10.1002/1097-4679(200012)56:12<1587::AID-9>3.0.CO;2-G

Staebler, K., Helbing, E., Rosenbach, C., & Renneberg, B. (2011). Rejection sensitivity and borderline personality disorder. *Clinical Psychology & Psychotherapy, 18*, 275-283. doi:10.1002/cpp.705

Stanley, B., & Brodsky, B. S. (2005). Suicidal and self-injurious behavior in borderline personality disorder: a self-regulation model. *Understanding and Treating Borderline Personality Disorder: A Guide for Professionals and Families. Edited by Gunderson JG, Hoffman PD.* Washington, DC, American Psychiatric Publishing, 43-63.

Stanley, B., & Siever, L. J. (2010). The interpersonal dimension of borderline personality disorder: Toward a neuropeptide model. *American Journal of Psychiatry, 167,* 24-39. doi:10.1176/appi.ajp.2009.09050744

Steed, L. (2001). Further validity and reliability evidence for Beck Hopelessness Scale scores in a nonclinical sample. *Educational and Psychological Measurement, 61,* 303–316. doi:10.1177/00131640121971121

Stevens, A., Burkhardt, M., Hautzinger, M., Schwarz, J., & Unckel, C. (2004). Borderline personality disorder: impaired visual perception and working memory. *Psychiatry Research, 125,* 257-267. doi:10.1016/j.psychres.2003.12.011

Stiglmayr, C. E., Shapiro, D. A., Stieglitz, R. D., Limberger, M. F., & Bohus, M. (2001). Experience of aversive tension and dissociation in female patients with borderline personality disorder: a controlled study. *Journal of Psychiatric Research, 35,* 111–18. doi:10.1016/S0022-3956(01)00012-7

Stuss, D. T., Stethem, L. L., Hugenholtz, H., & Richard, M. T. (1989). Traumatic brain injury: A comparison of three clinical tests and analysis of recovery. *The Clinical Neuropsychologist, 3,* 145-156. doi:10.1080/13854048908403287

Swartz, M., Blazer, D., George, L., & Winfield, I. (1990). Estimating the prevalence of borderline personality disorder in the community. *Journal of Personality Disorders, 4,* 257-272. doi:10.1521/pedi.1990.4.3.257

Swirsky-Sacchetti, T., Gorton, G., Samuel, S., Sobel, R., Genetta-Wadley, A., & Burleigh, B. (1993). Neuropsychological function in borderline personality disorder. *Journal of Clinical Psychology, 49,* 385-396. doi:10.1002/1097-4679(199305)49:3<385::AID-JCLP2270490313>3.0.CO;2-4

Takahashi, T., Ikeda, K., Ishikawa, M., Tsukasaki, T., Nakama, D., Tanida, S., & Kameda, T. (2004). Social stress-induced cortisol elevation acutely impairs social memory in humans. *Neuroscience Letters, 363,* 125–130. doi:10.1016/j.neulet.2004.03.062

Tebartz van Elst, L., Hesslinger, B., Thiel, T., Geiger, E., Haegele, K., Lemieux, L., . . . Ebert, D. (2003). Frontolimbic brain abnormalities in patients with borderline personality disorder: a volumetric magnetic resonance imaging study. *Biological Psychiatry, 54,* 163-171. doi:10.1016/S0006-3223(02)01743-2

Tombaugh, T. N. (2006). A comprehensive review of the Paced Auditory Serial Addition Test (PASAT). *Archives of Clinical Neuropsychology, 21,* 53–76. doi:10.1016/j.acn.2005.07.006

Tombaugh, T. N., Rees, L., Baird, B., & Kost, J. (2004). The effects of list difficulty and modality of presentation on a computerized version of the Paced Serial Addition Test (PSAT). *Journal of Clinical and Experimental Neuropsychology, 26*, 257–265. doi:10.1076/jcen.26.2.257.28080

Torgersen, S., Kringlen, E., & Cramer, V. (2001). The prevalence of personality disorders in a community sample. *Archives of General Psychiatry*, *58*, 590-596. doi:10.1001/archpsyc.58.6.590

Veiel, H. O. F. (1997). A preliminary profile of neuropsychological deficits associated with major depression. *Journal of Clinical and Experimental Neuropsychology, 19,* 587–603. doi:10.1080/01688639708403745

Wagner, A. W., & Linehan, M. M. (1999). Facial expression recognition ability among women with borderline personality disorder: Implications for emotion regulation? *Journal of Personality Disorders, 13,* 329-244. doi:10.1521/pedi.1999.13.4.329

Walter, M., Bureau, J. F., Holmes, B. M., Bertha, E. A., Hollander, M., Wheelis, J., Brooks, N. H., & Lyons-Ruth, K. (2008). Cortisol response to interpersonal stress in young adults with borderline personality disorder: a pilot study. *European Psychiatry, 23*, 201–204. doi:10.1016/j.curpsy.2007.12.003

Ward, T. (1997). A note of caution for clinicians using the Paced Auditory Serial Addition Task. *British Journal of Clinical Psychology, 36,* 303-307. doi:10.1111/j.2044-8260.1997.tb01417.x

Warner, R. M. (2013). *Applied statistics from bivariate through multivariate techniques* (2nd ed.). SAGE Publications, Inc.

Welch, S. S., & Linehan, M. M. (2002). High-risk situations associated with parasuicide and drug use in borderline personality disorder. *Journal of Personality Disorders*, *16*, 561-569. doi:10.1521/pedi.16.6.561.22141

Widiger, T. A., & Weissman, M. M. (1991). Epidemiology of borderline personality disorder. *Hospital and Community Psychiatry*, *42*, 1015-1021.

Wiens, A.N., Fuller, K. H., & Crossen, J. R. (1997). Paced Auditory Serial Addition Test: Adult norms and moderator variables. *Journal of Clinical and Experimental Neuropsychology, 19,* 473-483. doi:10.1080/01688639708403737

Wingenfeld, K., Spitzer, C., Rullkötter, N., & Löwe, B. (2010). Borderline personality disorder: Hypothalamus pituitary adrenal axis and findings from neuroimaging studies. *Psychoneuroendocrinology, 35,* 154–170. doi:10.1016/j.psyneuen.2009.09.014

Wingenfeld, S. A., Holdwick, D. J., Davis, J. L., & Hunter, B. B. (1999). Normative data on Computerized Paced Auditory Serial Addition Task performance. *The Clinical Neuropsychologist, 13,* 268–273. doi:10.1076/clin.13.3.268.1736

Winter, D., Elzinga, B., & Schmahl, C. (2014). Emotions and memory in borderline personality disorder. *Psychopathology, 47,* 71-85. doi:10.1159/000356360

Vuilleumier, P. (2005). How brains beware: neural mechanisms of emotional attention. *Trends in Cognitive Sciences, 9,* 585–594. doi:10.1016/j.tics.2005.10.011

Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit-formation. *Journal of Comparative Neurology and Psychology, 18,* 459-482. doi:10.1002/cne.920180503

Zakzanis, K., Leach, L., & Kaplan, E. (1998). On the nature and pattern of neurocognitive function in major depressive disorder. *Cognitive and Behavioral Neurology, 11,* 111–119.

Zanarini M. C., Frankenburg, F. R., DeLuca, C. J., Hennen ,J., Khera, G.S ., & Gunderson, J. G. (1998). The pain of being borderline: dysphoric states specific to borderline personality disorder. *Harvard Review of Psychiatry, 6,* 201–07. doi:10.3109/10673229809000330

Zanarini, M. C., Frankenburg, F. R., Dubo, E. D., Sickel, A. E., Trikha, A., Levin, A., & Reynolds, V. (1998). Axis I comorbidity of borderline personality disorder. *American Journal of Psychiatry, 155,* 1733-1739.

Zanarini, M. C., Skodol, A. E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., . . . Gunderson, J. G. (2000). The collaborative longitudinal personality disorders study: Reliability of axis I and II diagnoses. *Journal of personality disorders, 14,* 291-299. doi:10.1521/pedi.2000.14.4.291

Zimmerman, M., & Mattia, J. I. (1999). Axis I diagnostic comorbidity and borderline personality disorder. *Comprehensive Psychiatry, 40,* 245-252. doi:10.1016/S0010-440X(99)90123-2

van Zomeran, A. H., & Brouwer, W. H. (1994). *Clinical neuropsychology of attention.* New York: Ocford University Press.