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Neuropsychological Correlates of Borderline Personality Disorder

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In the current study, participants with borderline personality disorder (BPD) displayed deficits in neuropsychological functioning when compared with healthy controls. Participants with BPD performed worse on all measures of cognitive functioning: attention, verbal memory, processing speed and a measure of general neuropsychological functioning. The study found that depression was significantly more prevalent in the BPD sample compared with the control sample and that there was a significant inverse correlation between level of depression and scores on a general index of neuropsychological functioning. Results from ANCOVA analyses revealed significant differences existed in neuropsychological performance on all four measures of cognitive functioning between the two groups after controlling for depression. The role of effort in testing with persons with BPD was explored, with results indicating that participants with BPD provided good effort. Lastly, the study's findings showed that those participants with a BPD diagnosis and a neurological disease performed worse on the measure of general neuropsychological functioning compared with individuals with BPD who had no history of a diagnosed neurological disease. Results from ANCOVA analyses revealed that significant differences in neuropsychological performance on all measures of cognitive functioning existed between the two groups after controlling for presence of a diagnosed neurological disorder.

Implications of the study findings have been presented and discussed. Also, possible confounds to the study's findings were identified and discussed in the hope that future replications of the current study will control for such variables and result in robust research findings. Suggestions for future research in this area have been provided to assist in the construction of a more complete neuropsychological profile of BPD.

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Introduction

Borderline Personality Disorder (BPD) is frequently diagnosed in clinical settings and continues to be one of the most researched personality disorders in terms of its phenomenology, biological markers, treatment response, family history, and outcome (Kavoussi, Coccaro, Klar, Bernstein, & Siever, 1990). BPD tends to be a chronic and debilitating syndrome, and is considered a complex disorder largely due to the multiple variations in presentation. There are nine criteria for BPD according to the American Psychiatric Association's (APA) *Diagnostic and Statistical Manual, 4th Edition, Text Revision (DSM-IV-TR,* American Psychiatric Association, 2000), but only five of these criteria need to be met to receive a diagnosis. Therefore, the numerous combinations of symptoms that are possible to satisfy diagnostic criteria for a BPD diagnosis allows for varied presentations of the disorder.

In addition to the multitude of ways the disorder can present itself, BPD is further complicated by the high degree of co-occurrence with other disorders, such as substance abuse disorders (Casillas & Clark, 2002; Grilo et al., 1997; Joyce et al., 2003; Ross, Dermatis, Levounis, & Galanter, 2003), depression and other mood disturbances (Comtois, Cowley, Dunner, & Roy-Byrne, 1999; Joyce et al., 2003), social phobia (Comtois et al., 2003), and posttraumatic stress disorder (Comtois et al., 2003; McGlashan et al., 2000; Zlotnick, Franklin, & Zimmerman, 2002). Because BPD rarely presents in "pure" form, but instead presents with co-occurring Axis I or Axis II psychopathology, conducting research with individuals diagnosed only with BPD is extremely difficult (Donegan et al., 2003). The high degree of complexity associated

with BPD underscores the need for additional research in this area to help improve understanding of the etiology, phenomenology and effective treatments of BPD.

A wide body of neuropsychological research provides empirical evidence to support the idea that individuals with BPD suffer from various cognitive impairments (e.g., Bazanis et al., 2002; Beblo, Saavedra, Mensebach, & Driessen, 2005; Carpenter, Gold, & Fenton, 1993; Dinn et al., 2004; Judd & Ruff, 1993; O'Leary, Brouwers, Gardner, & Cowdry 1991; Ruocco, 2005; Stevens, Burkhardt, Hautizinger, Schwarz, & Unckel, 2004; Swirsky-Sacchetti et al., 1993). However, the extent to which cognitive impairments are the result of psychological distress versus physiological abnormalities remains uncertain.

Sprock, Rader, Kendall, and Yoder (2000) have suggested that the emotional and behavioral dysregulation commonly experienced by individuals with BPD are due to neurological dysfunction. For example, problems with attention, memory, processing complex information and impulsivity are all difficulties displayed by individuals with BPD and it is likely these deficits lead to psychological distress in the form of disrupted interpersonal relationships (Sprock et al., 2000). Neuropsychological studies have shown performance deficits for individuals with BPD, relative to controls, suggestive of temporal and/or frontal lobe dysfunction (Sprock et al., 2000; Swirsky-Sacchetti et al., 1993). Travers and King (2005) have hypothesized that the cognitive deficits found in persons with BPD may be secondary to such brain insults as trauma, encephalitis, or epilepsy. Examining the extent to which neuropsychological deficits in persons with BPD can be attributed to brain dysfunction remains an important research question.

Other physiological research investigating the neuroanatomical aspect of BPD suggest abnormalities in the amygdala (Donegan et al., 2003) which is an important

structure for emotion regulation, and malfunction of the medial prefrontal cortex which is associated with poor inhibition of the amygdala (Bremner et al., 2004). As a result of such findings, these deficits have been hypothesized to be contributing factors to the development and presentation of the disorder. While physiological testing and neuropsychological testing provide evidence that perhaps a subset of individuals with BPD have cerebral dysfunction that may predispose persons to BPD pathology, overall research in this area is limited with variable and assorted findings. Researchers have yet to define a specific neurological profile to fit BPD.

What follows is a review of various aspects of neuropsychological functioning in persons with BPD. The author will begin with an overview of BPD diagnostic features organized around the construct of dysregulation. Etiological theories for the development of the disorder will also be provided. Next, the paper will review the literature related to different areas of neuropsychological functioning in persons with BPD. Explanations for the inconsistent neuropsychological findings within the literature will be explored and the validity of assessment data and neuropsychological tests will be addressed. Finally, the direction, methodology, and results of the current study will be presented and discussed.

Diagnostic Features of Borderline Personality Disorder

A hallmark feature of BPD is dysregulation in a number of areas of functioning. Criteria for BPD in the *DSM-IV-TR* (APA, 2000) are associated with patterns of behavioral, emotional, and cognitive instability and dysregulation. Behavioral dysregulation may manifest for individuals with BPD through a variety of interpersonal problems. These difficulties may take the form of intense, conflict-ridden relationships with deep feelings that are not shared by the other person in the relationship. Sometimes

individuals with BPD share intimate details with others they don't know well, demand a great deal of time from the other person, and idealize others within the first or second meeting. This pattern of behavior is reflective of one BPD criterion, namely frantic efforts to avoid real or imagined abandonment (APA, 2000). Relationship boundaries can be violated and challenged by individuals with BPD. The *DSM-IV-TR* (APA, 2000) states that although individuals with BPD display short tempers, with outbursts of anger within relationships, they continue to remain in relationships because they fear abandonment. Regardless of the validity of this fear of abandonment, persons with BPD may take drastic measures to avoid being left and experiencing the potential associated feeling of being a "bad" person.

Impulsivity is another example of behavioral dysregulation that is a prominent characteristic of BPD. Impulsivity can manifest in gambling, reckless driving, promiscuity, substance abuse, and reckless spending of money (APA, 2000). While such behaviors are damaging, a more dangerous display of impulsivity by BPD sufferers is recurrent self-mutilating or self-harming behaviors. Frequently, fear of separation serves as the impetus for suicide attempts. Actual suicide attempts and self-injurious acts, such as self-mutilation and self-inflicted burns, with little or no intent to cause death are defined as parasuicidal behaviors (Kreitman, 1977). These behaviors frequently occur during moments of disassociation. Such maladaptive behaviors may serve to reaffirm the individual's capacity to feel, or to dispel notions of being an "evil" person.

Emotional dysregulation affects individuals with BPD in the form of intense mood swings in and out of very depressed, anxious, and irritable states that can last a few days or more (APA, 2000). They live in a world that appears always to be in conflict

with their emotions. Some persons with BPD express anger in the form of violent or physically aggressive behaviors. Anxiety, irritability, and dysphoria are also among the mood states that persons with BPD experience with great intensity. However, these extreme episodes typically only last a few hours, and on rare occasions persist for more than a few days (APA, 2000). Interruptions to these episodes are characterized by displays of anger, panic, or despair without the reprieve from such feelings as gratification or contentment. It is common for stress to incite these mood episodes, particularly stress in the interpersonal facets of the sufferer's life.

A chronic feeling of emptiness is another example of how people with BPD experience emotional dysregulation. This can lead them to engage in the habitual pattern of thrill seeking behaviors and dangerously impulsive decision-making (APA, 2000). Along with the feelings of emptiness, individuals with BPD commonly convey inappropriate anger and struggle to manage their expressions of sarcasm, resentment, or derision. These episodes are oftentimes connected with the sufferer perceiving a caregiver as being neglectful. These expressions of anger may result in the individual feeling guilty or embarrassed, strengthening maladaptive thoughts that they are inherently bad persons.

Cognitive dysregulation as a domain of BPD includes depersonalization, dissociative symptoms or paranoid ideation. These forms of thought dysregulation sometimes appear during periods of extreme stress and dissipate once the stress is ameliorated. Some persons with BPD have radical changes in beliefs, values, and career choices. These changes also involve the individual's sexual identity and the type of friends they choose. Frequently, people with BPD have a feeling that they do not exist at

all. In contrast, they can alter their relationship role from a dependent, clinging individual to a zealous advocate out to aid those in need. Another area of impairment under the cognitive dysregulation rubric includes cognitive distortions which are commonly part of the cluster of displayed symptoms. Examples of these cognitive distortions include odd experiences, superstitious beliefs, and magical thinking (Gunderson & Zanarini, 1987).

Prevalence

BPD is estimated to be present in roughly 2% of the general population, approximately 10% of individuals in outpatient mental health centers and about 20% of psychiatric inpatients (APA, 2000). Of individuals with a personality disorder, 30% to 60% are diagnosed with BPD, according to the APA (2000). An estimated 75% of people diagnosed with BPD are females (APA, 2000).

Etiology of BPD

Object Relations Theory

Several theories of etiology attempt to explicate the complexities of BPD. One prominent theory that proposes a disturbed caregiver-child relationship as a pathogenesis of BPD is Object Relations Theory (Westen & Gabbard, 1999). This theory represents a major development in psychodynamic theory within the past 30 years by providing an etiological explanation for BPD. The term "object relations" has several meanings, yet, most broadly, the term refers to enduring patterns of interpersonal functioning in intimate relationships along with the cognitive and affective processes mediating those patterns (Westen & Gabbard, 1999). Object Relations Theory emphasizes the effects that deprivation of healthy human contact has during infancy and early childhood, the

importance of self-representations and representations of others (called "object representations") as factors that influence interpersonal functioning, and the basic need for human connectedness that begins in infancy.

Under healthy developmental conditions, it is theorized that the caregiver allows the child to explore and separate in an effort to prompt the child to synthesize the different "good" and "bad" selves into one whole. By providing this experience for the child, "object constancy" is created. If the caregiver prevents the child from separating, the "pleasure ego" does not convert into the "healthy" or "reality ego" (Gibson, 1990).

Although the child's desire to separate from the caregiver is a healthy biological drive, the caregiver might interpret such behavior as a personal threat (Gibson, 1990). The caregiver may not be able to tolerate separation or abandonment and communicate to the child that he or she must remain attached to the caregiver or die (Bersin, 1994). The caregiver may respond by withdrawing affection and thereby creating a feeling of abandonment and anxiety in the child. This response establishes a pattern of dependence in the child. Because the child never learns to assimilate or integrate the bad object and the good object, he or she develops a dichotomous thinking pattern as a defense against reality. Under these circumstances, the child's representations of self and object can become polarized, that is, all good or all bad. This form of dysfunctional development is labeled splitting, and "from a psychodynamic point of view is a product of the irresolvable conflict between intense negative and positive emotions" (Linehan, 1993, p. 35). The cognitive preconditions for splitting are thought to establish themselves between 12 and 18 months of infancy (Gergely, 1992).

Object Relations Theory posits that once the child moves into adolescence, the healthy developmental process requires transference of satisfied libido needs by the parent to fulfilling of such needs by a mate that will eventually lead to assuming the independence necessary in adulthood (Gibson, 1990). Adolescents sometimes struggle with feelings of growing autonomy that can be equated with abandonment and depression and that conflict with the desire for feelings of closeness from his or her parents. In a failed effort to protect against depression and anxiety that result from these feelings of dissonance, adolescents may employ defense mechanisms such as denial and projection, as well as adopting polarized thinking methods. Consequently, he or she may resort to behaviors that are characteristic of individuals with BPD including affective instability as a result of discernible reactive mood and an unstable sense of self-image or sense of self (APA, 2000).

The preceding description establishes an etiological basis of BPD due to a caregiver's overinvolvement with the child to fulfill his or her own needs. There is also a second theoretical explanation that exists for the development of BPD. Several psychodynamic theorists (e.g., Adler & Buie, 1979; Kohut, 1971; Winnicott, 1991) assert that a mother or other caregiver serves as an external validator and regulator of a young child's needs and impulses. If a child has a caregiver that models a stable, nurturing environment, then the child is able to develop internal monitoring and satisfaction of impulses and an internal sense of worth. However, if the caregiver fails to provide a validating, nurturing and reinforcing environment, the result can be an undeveloped sense of self worth that typifies the individual with BPD. Consequently, the individual continually seeks involvement in relationships in order to feel validated, but maintains a

confused state regarding his or her own identity, and resorts to scanning the environment for cues on how to act and what to think and feel (Linehan, 1993). Therefore, within the psychodynamic theory of Object Relations a potential second pathogenic element of a mother's interaction with her child (in addition to overinvolvement) is the lack of appropriate responsiveness to the child's impulses and needs (Bezirganian, Cohen, & Brook, 1993). Such inappropriate responsiveness may be conceptualized as a caregiver's inconsistent parenting of the child.

Psychodynamic theories (e.g., Adler & Buie, 1979; Masterson, 1978) converge on the assumption that significant deficits exist for individuals with BPD in the area of interpersonal relationships, particularly in regard to separation-individuation. The individual with BPD experiences feelings of emptiness, anxiety, isolation, and a loss of sense of self as a result of an inability to internalize primary mother-child caring. Empirical results from a study conducted by Bezirganian and colleagues (1993) showed "the combined effect of maternal inconsistency and maternal overinvolvement accounted for the effect of poor maternal ego integration on the development of Borderline Personality Disorder in the child" (p. 1841). Such findings suggest that it is the pattern of caregiver-child interaction as an environmental factor that may be responsible in transmitting the disorder from one generation to the next.

More contemporary attachment theorists have advanced etiological explanations for BPD beyond that of Object Relations. Kernberg (1984) has postulated that disruptions in childhood attachments contribute to the development of BPD. As a result neuropsychological problems develop that are associated with difficulties in social interactions and ineffectiveness in coping with stress caused by abusive environments,

disruptive parenting, developmental deficits, and maladaptive coping techniques that contribute to one's pathology.

Also, Gronstein (1990) asserted that the neurobiological impairment seen in persons with BPD can be traced back to a preverbal developmental period where disruptive parenting results in a lack of secure attachment and bonding for the child. The theory supposes that a mother experiencing stress and pathology, including the borderline trait of emotion regulation difficulty, will engage in a pattern of interaction characterized by affective and self-regulatory limitations with her child by pulling away from her distressed child. This emotional withdrawal by the mother is experienced with great intensity by the child. Consequently, the child has a sense of being bonded to a nonnurturing family environment and forms an unstable sense of self. In more extreme conditions involving caretaker neglect and abuse, research indicates that children exposed to parental maltreatment, child physical and sexual abuse being the most dramatic example, exhibit core features associated with a disorganized style of attachment (Barnett, Ganiban, & Cicchetti, 1999; Carlson, 1998; Lyons-Ruth & Jacobvitz, 1999).

Children with a disorganized attachment style do not form an adequate bond with their caregivers, do not acquire the skills from their caregivers to help them modulate affective experiences, and develop a lifelong experience of heightened vulnerability to shame and inability to regulate their emotional experiences. According to Berntson, Cacioppo and Quiqley (1991), over time these types of experiences are imprinted onto the child's limbic system and interfere with child's ability for autonomic control. Neuroscience research supports the idea that the childhood experiences of learning to regulate emotions and successful navigate fearful experiences are necessary for

development of brain structures that help regulate anxiety and develop organized responses in frightening situations (Siegel, 2001).

Biosocial Theory

Another etiological theory, the biosocial theory proposed by Linehan (1993), posits that BPD is a result of both biological irregularities and dysfunctional environments, and in a synergistic fashion ultimately results in a dysfunction in the emotion regulation system. Similar to the psychodynamic view discussed earlier that views the mother as an external validator for the child, the biosocial perspective postulates that invalidating environments prevent a child from learning how to label and regulate arousal, tolerate emotional distress, and learn when to believe in his or her own emotional responses to events as valid interpretations of events (Linehan, 1993).

Within the biosocial model, individuals with BPD are seen as influenced during adulthood by their childhood invalidating environment, and consequently they invalidate their own emotional experiences, look to others for cues regarding correct reflections of reality, and have a tendency to oversimplify problems (Linehan, 1993). Because these individuals oversimplify their problems, they typically set unrealistic goals, have difficulty using reward rather than punishment for small accomplishments, and engage in self-hate when failing to achieve their goals. Individuals with BPD have this shame response engrained in them by their invalidating environment that censures them from expressing emotional vulnerability.

The learning conditions that interact with the biological predispositions are characterized, according to Linehan (1993), by an invalidating environment that includes rejection, minimizing, and punishing internal experiences thereby thwarting development

of effective emotion regulation. Biological underpinnings for the biosocial theory of BPD are unclear, but are believed to include disruptions in the limbic system, the brain system responsible for emotion regulation and attention control (Linehan, 1993). The emotion regulation system is complex, making it difficult to identify confidently a common variable associated with it as the cause for BPD. Biological causes could potentially include genetic heritability, with studies finding that first-degree relatives of persons with BPD have higher prevalence rates affective disorders (Akiskal, 1981, Andrulonis et al., 1981), harmful intrauterine factors that later influence behavioral patterns in individuals like fetal alcohol syndrome and its sequelae of hyperactivity, impulsiveness, distractibility, and irritability (Abel, 1981, 1982), or early childhood environmental invalidating events, abuse or trauma that affect the brain and nervous system development. Biological predispositions are reinforced through learning and thought to contribute to the development of BPD (Linehan, 1993). More specifically, biological irregularities in the limbic system interfere with effective regulation of affect, creating high sensitivity to emotional situations with a delayed return to base line for individuals with BPD.

Beck and Freemen (1990) provide a similar etiological explanation for maladaptive cognitive processes that develop early as a child and manifest as distorted cognitions such as “The world is a dangerous and hostile place” or “I am worthless and vulnerable.” These distortions influence the individual’s perception of the world, themselves, the future, as well as behavioral and emotional responses.

Diathesis-Stress Model

Another etiological theory is the Diathesis-Stress Model that states environmental risks interact with genetic vulnerabilities, including neurobiological and neuropsychological vulnerabilities, to contribute to the clinical syndrome of BPD. Diatheses have been identified and include neurobiological markers such as deficits in frontal lobe functioning that manifest in neuropsychological difficulties including impulsivity, cognitive inflexibility, and perseveration (Judd & Ruff, 1993; O'Leary et al., 1991; Swirsky-Sacchetti et al., 1993; van Reekum, Conway, Gansler, White, & Bachman, 1993; van Reekum et al., 1996). Stressors established as common features associated with BPD that also potentially contribute to the development of the disorder include a history of psychological trauma, emotional neglect, physical and/or sexual abuse during childhood and adolescence, as well as witnessing violence, with trauma being related to psychophysiological and neuropsychological changes. (Goldman, D'Angelo, DeMaso, & Mezzacappa, 1992; Guzder, Paris, Zelkowitz, & Feldman, 1999; Guzder, Paris, Zelkowitz, & Marchessault, 1996; Yen et al., 2002).

Using a Diathesis-Stress Model, Beblo et al., (2005) have proposed the existence of an unspecific genetic disposition of individuals with BPD that includes cerebral lesions and/or inherent personality traits (e.g., impulsivity). As a result of this genetic disposition, the coping abilities of children may be compromised, leading to an increased likelihood of learned negative responses within their disrupted social environment. Additionally, this genetic disposition interacting with previously mentioned common environmental stressors could contribute further to the development of BPD. Therefore, Beblo et al., (2005) propose that in addition to recurring traumatic experiences in

childhood and adolescence as a risk factor, a genetic disposition along with certain liabilities in personality may further increase the chances of developing BPD.

Neurobiological Theory

Abnormalities in an individual's biology provide yet another etiological theory for the development of BPD. The notion of an organic subtype of BPD, as defined by neurological deficits, began with a series of prevalence studies in the early 1980's that indicated up to 81% of individuals with BPD had a history some type of neurological insult (Andrulonis, Glueck, Stroebel, & Vogel., 1982; van Reekum et al., 1993). The first to note the possible existence of an organic subtype of BPD, Andrulonis and colleagues (1982) identified a pattern in the developmental history of a higher prevalence of seizures, learning disabilities, head trauma, and attention deficit disorder with or without hyperactivity, and acquired brain injuries compared with psychiatric controls. In an attempt to replicate these findings Andrulonis (1990) found that the developmental factors were present in 50% of male BPD subjects, but only 20% of the female subjects presented with such histories. As a result, females with BPD were hypothesized to display a constellation of symptoms that placed them on the affective disorders spectrum, while males with BPD were conceptualized as existing on a spectrum of organic brain dysfunction that included episodic dyscontrol syndrome and adult minimal brain dysfunction. van Reekum and colleagues (1993) also found a positive correlation between the severity of brain disturbance and severity of behavior disturbance.

Travers and King (2005) investigated the degree to which cognitive deficits observed in persons with BPD can be attributed to the result of organic insult, either developmental or acquired. Findings from a study by Travers and King (2005) provided

partial support for their hypothesis that cognitive deficits in BPD are largely the consequence of organic insult. Subjects with both BPD and a history of organic insult were significantly more impaired on measures of attention compared with subjects with only a BPD diagnosis (Travers & King, 2005).

Schore (1994) has suggested that the emotional dysregulation experience of rage that is characteristic of BPD is the result of an inability of the orbitofrontal system to modulate the excitatory ventral tegmental limbic circuit, while the depressive states associated with feelings of abandonment are due to an inability to regulate lateral tegmental circuit inhibition. As a result of an underdeveloped orbitofrontal system, individuals with BPD cannot access symbolic representations that allow for important self-soothing, reparative functions encoded in evocative memory. In addition, their autonomic nervous system responsible for emotional regulation and mediating emotional responses becomes impaired.

The amygdala has been identified as a brain structure associated with physiological and behavioral reactions to objects and situations that have particular biological significance, including painful stimuli. While the amygdala is activated in the presence of emotionally relevant stimuli, the hippocampus is associated with the process of consolidation of information in memory. These two structures have been the primary focus of several neuroimaging studies that have attempted to explicate the neurobiological features associated with BPD (e.g., Driessen et al., 2004; Lucas, Gardner, Cowdry, Pickar, 1989; Schulz, 1983; Snyder & Pitts, 1984).

It has been proposed that the amygdala is the brain structure responsible for detecting and responding first to new stimuli in the environment, particularly those

stimuli that are potentially dangerous (LeDoux, 1992). Physiological studies reveal a sensory/processing loop where the amygdala connects to the sensory thalamus, which connects to the sensory cortex and then back to the amygdala (LeDoux, 1995). In this way, the amygdala serves a critical function in response to urgent situations that allow for rapid activation of emotions and interpretation of feedback from the cortex following the initial alarm (LeDoux, 1995, 1996). While the amygdala appears to be active particularly during periods of stress or threat, the hippocampal functioning seems to be somewhat impaired when exposed to the elevated levels of cortisol commonly produced in threatening situations (Nadel & Jacobs, 1998).

Neuroimaging studies evaluating the volumes of brain structures revealed 8% smaller amygdala and 16% smaller hippocampi in subjects with BPD compared to control subjects (Driessen et al., 2004). Findings from animal research support the idea of a correlation between stressful conditions and structural abnormalities in the hippocampus and subsequently memory functioning (Bremmer, 1999; Uno, Tarara, Else, Suleman, & Sapolsky, 1989). Neuroimaging research with human subjects has demonstrated smaller hippocampal volumes in persons suffering from PTSD (Bremmer et al., 1995) and in female survivors of sexual and physical abuse in childhood (Stein, Koverola, Hanna, & Torchia, 1997). Such findings are informative and particularly relevant because of the high prevalence of PTSD and trauma history in persons with BPD (McGlashan et al., 2000; Sar et al., 2003; Zlotnick et al., 2002).

Despite these neuroimaging findings, the association of reduced hippocampus and amygdala volume and human traumatic experiences remains unclear. For example, in a study by Driessen and colleagues (2004), the correlations between the reduced structural

volumes and trauma existed only with regard to the entire sample but were not present when the BPD and healthy samples were analyzed separately. Other researchers have found the reduced volumes of the hippocampus varied considerably from left to right hippocampus (Gurvits et al., 1996). Also, smaller volumes of the amygdala appear in some studies (e.g., Bremmer, 1999) while other studies found increased amygdala volumes (e.g., Gurvits et al., 1996).

Overall, the research evidence that does demonstrate the presence of reduced brain volumes in structures like the hippocampus and amygdala supports the hypothesis that there exists a subtype of BPD that is organic in nature. However, because individuals with the disorder commonly present with complicated histories of abuse and comorbid PTSD diagnosis, it is difficult to determine if BPD symptoms like emotional dysregulation have an organic etiology or if the symptoms are the result of exposure to certain environmental factors that manifest in the type of psychological distress displayed by persons with BPD.

Neuropsychology of BPD

Several characteristics associated with BPD contain pathological features with cognitive components including odd reasoning, poor concept formation, dichotomous thinking, projection and splitting, selective memory for past experiences, difficulty differentiating current experiences from past expectations, and associating anxiety with thought processes instead of ameliorating it through action (Kernberg, 1975; Knight, 1953; O'Leary & Cowdry, 1994; Stone, 1980). From a clinical perspective, these client characteristics manifest through missed appointments, misperceiving situations,

explosive responses to innocuous events, poor insight into consequences of one's actions, slow progress in therapy, and general difficulty learning from past experiences.

There are several possible explanations for why these psychological cognitive components have not been empirically studied until recently. One reason may be related to the psychodynamic conceptualization of these deficits. That is, splitting, memory lapses, misperceptions are conceptualized as defense mechanisms that protect the individual from experiencing painful emotions, memories, or conflicts. Also, in an effort to distinguish BPD from schizotypal personality disorder, the cognitive style features associated with BPD such as poor insight and cognitive distortions have been deemphasized (Kroll, 1988).

Another reason noted by O'Leary (2000) that these characteristics of BPD have historically not been the focus of neuropsychological studies is because of the often held belief that persons with BPD present as "normal" on structured psychological tests such as the Wechsler Adult Intelligence Scale-III (WAIS-III; Wechsler, 1997a). Much of the literature concerned with psychological test performance of individuals with BPD is characterized by the statement "intact WAIS, disturbed Rorschach" meaning the within normal limits profile of structured psychological tests like the WAIS contrasts with "psychotic" thinking on unstructured projective tests like the Rorschach (Berg, 1983; Carr, Goldstein, Hunt, & Kernberg, 1979; Rapaport, Gill, & Schafer, 1968; Singer, 1977). Clinical impressions corroborate research findings in the sense that the BPD population demonstrates a normal distribution of intelligence with many functioning well in structured work settings (O'Leary, 2000).

Neuropsychological Literature on BPD

Empirical evidence indicates that individuals with BPD have deficits in several areas of neurocognitive functioning (Swirsky-Sacchetti et al., 1993) suggestive of a possible underlying brain pathology. However, the research in this area is limited with variable and assorted findings that preclude a clear characterization of the neurocognitive features associated with BPD. Early neurobehavioral studies that seemed to identify a relationship between acquired or developmental brain dysfunction and borderline pathology failed to provide a consistent picture of neuropsychological deficits (Andrulonis, 1990; Andrulonis et al., 1982; Andrulonis et al., 1981; van Reekum et al., 1993; van Reekum et al., 1996). More recent neuropsychological investigations have used improved methodology and more comprehensive test batteries to identify specific neurocognitive impairments among persons with BPD (see Ruocco, 2005 for a review).

Although a wide body of literature supports the notion of neurocognitive deficits in this population, other studies have not found any significant differences in cognitive functioning between persons with BPD and healthy controls. While the relationship between neurocognition and borderline pathology remains unclear, the most consistent significant findings appear to be in the areas of attention, verbal memory, visual memory, processing speed, and visuospatial perception. Following is a review of the existing literature on neuropsychological testing involving subjects with BPD with particular emphasis on those areas of cognitive functioning that have revealed the most compelling findings.

Attention

Travers and King (2005) used the Digit Span subtest and the Digit Symbol subtest from the WAIS- R (Wechsler, 1981) and the Visual Memory Span subtest from the Wechsler Memory Scale-Revised (WMS-R; Wechsler, 1987) to measure attention. The Digit Span subtest contains a forward and backward trial, each consisting of seven pairs of random-ordered digit sequences that the examiner reads aloud and requires the participant to repeat the sequence either forward or backward. For the Digit Symbol subtest, participants are timed and asked to view a list of nonsense symbols paired with numbers and use the list as a key to fill in the correct corresponding symbol that is missing below a series of numbers that follow. Besides being a test of attention, visual motor coordination, and perceptual organization, it is the one subtest from the WAIS-R (Wechsler, 1981) that is considered the most sensitive to brain damage (Lezak, Howieson, Loring, Hannany, & Fischer, 2004). The Visual Memory Span subtest is a non-verbal analog of Digit Span and requires participants to reproduce forward and backward a pattern of tapping sequences using a set of randomly-ordered blocks beginning with 2 and going up to 8 blocks.

Travers and King (2005) compared two samples of individuals with BPD, one group without any documented history of organic brain injury and a second group with evidence of past significant head injury/encephalitis or epilepsy, and/or a current or past documented history of learning disability/ADHD. The results indicated that the BPD group with a history of organic brain injury displayed a significant deficit in attention relative to the BPD group without any brain injury. Travers and King (2005) suggest that

the attention impairments experienced by persons with BPD may be partly attributable to organic factors.

O'Leary et al. (1991) also found significant attention impairments in the group of BPD individuals compared with healthy controls as measured by the Digit Span subtest and the Digit Symbol subtest from the WAIS-R (Wechsler, 1981). Similarly, Judd and Ruff (1993) found significant differences in performance on the Digit Symbol subtest in a BPD sample compared with controls. Posner et al. (2002) used the Attentional Network Test (ANT), a reaction-time task that measures the efficiency of several aspects of attention (Fan, McCandliss, Sommer, Raz, & Posner, 2002) with a group of individuals with BPD and a control group. The ANT requires participants to press certain keys depending upon the type of stimuli that are presented on a computer monitor. The researchers found that there appears to be a specific attentional deficit in individuals with BPD concerned with conflict resolution or the ability to identify incongruent stimuli during the task.

Monarch, Saykin, and Flashman (2004) evaluated attention in persons with BPD using several measures including a Continuous Performance Test (CPT), Trail-Making Test A and B (Reitan, 1958), and the Digit Symbol and the Digit Span subtests from the WAIS-R (Wechsler, 1981). CPTs actually contain a group of attention measures that typically present stimuli briefly and provide reaction times as well as accuracy data. The Trail-Making Test A and B contains two parts. First, it requires individuals to draw lines to connect consecutively numbered circles on one work sheet (Trail A) and then connect the same number of consecutively numbered and lettered circles on a second work sheet (Trail B). Results from Monarch et al. (2004) showed that relative to comparison groups,

individuals with BPD demonstrated a significant impairment in attention as indicated by all measures of attention used in the study. Monarch and colleagues (2004) concluded that "it appears that attention skills in inpatients with BPD are severely compromised" (p. 77). The authors added that it is critical to adequately asses for attentional problems because such deficits may directly and indirectly affect other areas of functioning by, for example, interfering with a person's ability to organize his or her test-taking behaviors, solve problems, and encode and retrieve new information.

However, not all research findings support the idea of persons with BPD suffering from attention deficits. For example, Lenzenweger, Clarkin, Fertuck, and Kernberg (2004) used another type of CPT, the Continuous Performance Test-Identical Pairs Version (CPT-IP; Cornblatt, Risch, Faris, Friedman, & Frienmeyer-Kimling, 1988), to assess for sustained attention in a sample of persons with BPD. The authors found no difference in attention ability between individuals with BPD and controls as measured by the CPT-IP. Additionally, Swirsky-Sacchetti et al. (1993) found no significant differences between individuals with BPD and controls using two attention measures: the Symbol-Digit Modalities Test (Smith, 1968), a test similar to the Digit Symbol subtest from the WAIS-R (Wechsler, 1987) and the Trail-Making Test A and B (Reitan, 1958).

Verbal Memory

The WMS-III (Wechsler, 1997b) contains several subtests that are frequently used in neuropsychological assessments to assess verbal memory ability. For example, the Paired Associates subtest requires subjects to recall immediately and at delay lists of words read to them by the examiner. The more complex Logical Memory subtest asks subjects to recall immediately and again after a 30-minute delay, details of two stories

that were read aloud to them by the examiner. O'Leary et al. (1991) did not find any deficits in verbal memory using the Paired Associates subtest for the BPD group, but results did reveal they scored significantly lower at immediate and delay recall relative to the comparison group on the Logical Memory subtest. In an effort to further examine the nature of memory impairment, O'Leary et al. (1991) provided the BPD subjects with cues about the story (e.g., "Do you remember where the robbery took place?"). Interestingly, cueing allowed the individuals with BPD to remember additional information, eliminating the significant difference in performance between the two groups. The authors proposed that the improvement in performance on this task after cueing suggests that the deficits in memory exhibited by the BPD group may be the product of difficulty retrieving learned material and not a problem with the original encoding process.

Similar to findings by O'Leary and colleagues (1991), Dinn et al. (2004) found BPD subjects obtained significantly lower scores on the Logical Memory subtest compared with controls, but did not find a significant difference in scores between groups on the Associate Learning subtest. Judd and Ruff (1993) also found significant performance differences between the BPD group and controls on the delayed recall portion of the Logical Memory subtest. Score differences between groups on the immediate recall of the first Logical Memory story approached significance.

Swirsky-Sacchetti et al. (1993) conducted research to asses for evidence of impaired neuropsychological functioning in BPD and also to determine if there is any pattern of deficits. Results showed that, overall, the BPD group performed more poorly than controls across the entire battery of tests, including memory test performance. The

results showed a trend of BPD subjects to be more likely to perform more poorly than controls on a verbal-learning task when emotional interference was involved. However, the authors failed to find a significant difference in verbal memory using scores from the Logical Memory subtest. Carpenter et al. (1993) found BPD subjects performed worse than controls on the Logical Memory subtest compared to controls but this difference was not statistically significant.

Sprock et al. (2000) conducted a study examining memory in a sample of individuals with BPD, a sample of depressed individuals, and a nonpsychiatric control group. The authors used the Logical Memory, Figural Memory, and the Digit Span subtests from the WMS-R (Wechsler, 1987). The Figural Memory subtest is actually a visual memory test that presents subjects with designs for 10 seconds and then asks them to reproduce what they remember of each design. Results showed no significant differences between the BPD group, the group of depressed individuals and the control group on any of the WMS-R subtests.

In an attempt to explore Swirsky-Sancchetti et al.'s (1993) findings of an apparent trend for individuals with BPD to be more susceptible to emotional interference than controls on verbal-learning tasks, Sprock et al. (2000) included a story recall task (immediate and 15-minute delay) to assess memory for complex verbal material and the influence of emotional themes on recall. The story about a woman consisted of 10 negative, 10 positive and 10 neutral components. A verbal recall task consisting of two word lists was also completed by subjects at immediate recall and after a 1-minute period of neutral or emotional interference. Results revealed a significant difference in scores between the BPD group and the control group for neutral words on the delayed story

recall task, but the use of emotional interference on the verbal recall task did not affect performance of the BPD group or the other groups.

Kurtz and Morey (1999) studied memory functions with emotional words among a group of individuals with a co-occurring diagnosis of major depressive disorder (MDD) with BPD, a group of individuals with MDD without BPD, and a control group. The researchers used four lists of words: three lists containing words with strong emotional valences, and one list containing neutral words. Participants were instructed to judge each word using the descriptors "like" or "dislike." A free recall trial took place 5 minutes after the judgment task and a recognition trial took place 50 minutes following the judgment task. Results revealed that MDD participants with BPD performed significantly worse on the recall and recognition tasks relative to controls. While the MDD participants with BPD scored lower on these tasks compared with MDD without BPD, these differences were not significant. Both controls and participants with MDD and BPD recalled significantly more positive words than negative words. Participants with MDD without BPD recalled more positive words than negative words, but this difference was not statistically significant. Performance in the recognition task mirrored that of the recall data: control participants discriminated best across all word valences, and MDD participants with BPD scored the lowest of the three groups on the recognition task for all word valences.

Kurtz and Morey (1999) state that their data support the hypothesis that relative to controls, individuals with BPD displayed difficulties with verbal memory functions in both recall and recognition. These data corroborate a growing body of research findings that indicate memory disturbances in persons with BPD. The authors note that the study

findings suggest that the presence of BPD in depressed individuals may account for a portion of the memory deficits often attributed to depression. The authors note that although their data support the idea of greater specificity of memory deficits in individuals with MDD and BPD compared with persons diagnosed only with depression, the results are inconclusive regarding several possible causal relationships between BPD and memory deficits (see Garber & Hollon, 1991). That is, the observed memory dysfunction in the BPD group may be due to an additive affect of BPD and depression.

Renneberg et al. (2005) conducted a study that looked at the ability of a group of participants with BPD, a group of participants with depression, and a control group to retrieve autobiographical memories, or memories that relate to one's personal life. The autobiographical memory test (AMT) is comprised of five positive words, five negative words, and five neutral words. Words were presented to participants one at a time and then asked "What event does this word remind you of?" Participants' response times for each cue word were recorded. A memory was classified as specific if it referred to an occasion that did not span more than one day. The authors hypothesized that BPD participants would have more overgeneralized memories with more omissions and they would show similar retrieval as depressed persons for negatively valenced memories.

Study results from Renneberg et al. (2005) show that participants with depression, but not those with BPD, showed significant impairment in terms of number of specific retrieved memories compared to the control group. However, both depressed participants and BPD participants retrieved memories that were more negative compared with control participants' retrieved memories. BPD participants did not differ significantly in reaction time from the responses of the control participants. Renneberg and colleagues (2005)

propose that their findings suggest that individuals with BPD have easy and quick access to their autobiographical memories with negative emotional valence that may also be specific. The authors suggest that this fast and easy access to negative memories may contribute to an inability to adaptively cope with emotional turmoil.

In addition to the findings of Renneberg et al. (2005), Kurtz and Morey (1999) and Sprock et al. (2000), research has demonstrated that subjects with depression report more severe cognitive and emotional symptoms than did subjects who were not depressed (Gfeller, Chibnall, & Duckro, 1994). Subjects with depression also suffer from impaired immediate recall of new information and amount of acquisition, and when depression and anxiety presented together subjects not only displayed deficits in immediate recall and amount of acquisition, but also demonstrated deficits in retrieval of new information (Kizilbash, Vanderploeg, & Curtiss, 2002). These findings are particularly relevant when conducting research with a BPD sample because of the high prevalence of depression co-occurring with BPD (Comtois et al., 1999; Joyce et al., 2003).

The findings from Renneberg et al. (2005) are in contrast with Jones et al. (1999) who compared responses in an AMT by a sample of BPD individuals to those of controls. Jones et al. (1999) found that individuals with BPD recalled more overgeneralized memories and had more omissions (retrieval time that exceeded the allotted time limit) compared with controls. While these data suggest deficits in retrieval of specific autobiographical memories in BPD persons, Renneberg et al. (2005) and others (Arntz, Meeren, & Wessel, 2002; Kremers, Spinhoven, & vander Does, 2004) have not found evidence of overgeneralized retrieval in BPD persons indicating inconsistent findings in this area of memory functioning with this population.

Visual Memory

Several neuropsychological studies provide evidence for visual memory dysfunction in persons with BPD. The congruent findings across several comprehensive studies in the domain of visual memory offer the most compelling evidence to support the hypothesis of cognitive deficits in individuals with BPD. While it can be difficult to distinguish whether a certain test is measuring visual perception or visual memory, the Rey-Osterrieth Complex Figure Test (CFT) has been identified as being capable of measuring both domains of functioning (Corwin & Bylsma, 1993; Osterrieth, 1944). The CFT presents participants with a visual complex geometric drawing and then asks them to first copy the figure and then draw it from memory immediately and after a delay.

O'Leary et al. (1991) asked a sample of individuals with BPD to complete the CFT and found that the copy score of BPD participants was nearly identical to that of controls; however, the BPD participants' scores were significantly lower than control scores at 5 minute recall and at a 45 minute delayed recall. The authors assert that these findings suggest intact visuosperceptual and visuomotor skills but impaired complex visual memory. Additional support for deficits in this area comes from Carpenter et al. (1993) who found BPD subjects scored significantly lower compared with controls on the CFT. Judd and Ruff (1993) also found the BPD group performed significantly worse than control on the recall portion of the CFT. Additionally, Judd and Ruff (1993) analyzed the CFT copy score and discovered that 92% (n=23) of normals had perfect or near perfect scores compared with 20% (n=5) of the individuals with BPD. The results show that the subjects with BPD were unable to copy the figure accurately, and they produced either distorted drawings or drawings with missing parts.

Impaired performance on the CFT in a sample of persons with BPD was also found in a study conducted by Swirsky-Sacchetti et al. (1993), corroborating the findings by other researchers regarding visual memory deficits with this population. Swirsky-Sacchetti et al. (1993) used an older version of the Visual Reproduction subtest, the Figural Memory subtest from the WMS-R (Wechsler, 1987), to assess the ability to reproduce from memory designs that were presented for 10 seconds. The reproduction ability was tested immediately and following a 30 minute delay. Results show the BPD group produced significantly lower scores compared with controls at immediate and delayed recall. The authors propose that their data indicate visual memory deficits in participants with BPD which is consistent with their clinical reports of BPD patients who have difficulty with visual recall in daily living and in treatment.

Dinn et al. (2004) compared a sample of BPD participants with a sample of nonclinical controls. The authors used the CFT and required participants to copy the figure immediately and also at a 1 minute delay. Results show that the BPD participants obtained significantly lower scores on copy organization, copy accuracy, and recall accuracy of the CFT. Inspection of the drawings revealed that they were poorly organized and often displayed gross distortions of the geometric figure.

Not all findings concerning deficits in visual memory ability in BPD participants are as convincing or consistent. Sprock et al. (2000) included the CFT in their battery and although they found the BPD group scored lower than controls for the copy task, immediate recall, and at delayed recall, these differences in scores were not statistically significant. Using the WMS-R Visual Reproduction subtest (Wechsler, 1987), Carpenter et al. (1993) found individuals with BPD scored significantly lower than controls

indicating deficits in visual memory; however, these findings were not replicated in O'Leary et al.'s (1991) study. O'Leary et al. (2000) noted that contrasting findings often exist when comparing findings from complex memory tasks (e.g., the CFT) with results from simpler visual memory tasks like the WMS-R Visual Reproduction subtest (Wechsler, 1987).

Visual Perception

Research investigating visual perception impairment in persons with BPD appears to offer consistent neuropsychological findings. As with other domains, the tests used to assess for visual perception vary from study to study. O'Leary et al. (2000) noted that the strongest findings were found when using the Digit Symbol subtest of the WAIS-R (Wechsler, 1981) and the copy portion of the Rey Osterrieth CFT as measures of visual perception. As previously mentioned in the visual memory discussion, the Rey Osterrieth CFT is capable of measuring both visual memory and visual perception functioning (Corwin & Bylsma, 1993; Osterrieth, 1944). The copy aspect of the Rey Osterrieth CFT is considered to tap into visual perception and motor coordination. Using the Digit Symbol subtest and the copy portion of the Rey Osterrieth CFT, several studies have found impairments in visual perception (Carpenter et al., 1993; Judd & Ruff, 1993; Swirkey-Sacchetti, 1993).

More complex measures that have a strong visual perception component include the Embedded Figures Test (Witkin, Oltman, Raskin, & Karp, 1971) and the Corsi Blocks, also known as the Block Span test (Milner, 1971). The Embedded Figures Test entails presenting participants with a single geometric design and requiring them to use visual discrimination and filtering to identify the shape within a more complex one. The

Corsi Blocks test requires participants to remember and point out increasingly long patterns of three-dimensional blocks on a board with forward and backward trials. In a study by O'Leary et al. (1991) performances by BPD participants on both the Corsi Blocks Test and the Embedded Figures Test were significantly impaired on all trials. Judd and Ruff (1993) delivered the Block Span test and found on the delayed portion of the test participants with BPD produced lower mean scores that approached significance relative to controls.

Other tests have been used by researchers to assess for visual perception deficits in persons with BPD and have produced significant findings. Carpenter et al. (1993) found significant impairment in visual perception using Trail-Making Test A and B (Reitan, & Davison, 1974; Spreen & Strauss, 1991) and the Block Design subtest of the WAIS-R (Wechsler, 1981). Judd and Ruff (1993) used the Ruff Figural Fluency Test (RFFT; Ruff, Light, & Evans, 1987) and found significant deficits in BPD subjects. All of the visual perception tests mentioned include perceiving complex visual arrays that require participants to process new information, filter out extraneous stimuli, and select relevant visual details from a complex field (O'Leary, 2000).

Stevens et al. (2004) examined speed of visual perception via a backward masking paradigm. Participants were presented with a highly visible target (i.e., "A," "T") for a brief amount of time. Then, after a variable time interval, a low-contrast mask appeared at the same screen location. Participants were asked to press a button under the right index finger when a target was recognized (i.e., "A," "T") and press a button under the left index finger for non-targets ("X," "O"). Results show that BPD subjects required significantly more time to recognize the target stimulus compared with controls.

Processing Speed

Persons with BPD generally display poorer performance on tasks of processing speed compared to control groups (Ruocco, 2005). O'Leary et al. (1991) used the Digit Symbol subtest from the WAIS-R (Wechsler, 1981) as a measure of processing speed and found significant differences in BPD scores relative to control scores. These findings of processing speed deficits in persons with BPD were replicated by Judd and Ruff (1993) using the Digit Symbol subtest and the RFFT (Ruff et al., 1987), which besides being a measure of visual perception is also considered a measure of processing speed. Using a German version of the classic Stroop test, Kunert, Druecke, Sass, and Herpertz (2003) found differences in speeded processing performance between BPD subjects and controls on two subtests. Dinn and colleagues (2004) found deficits in BPD persons' processing speed performance relative to controls using both trials of the Trail-Making Test A and B (Reitan, 1958) as did Travers and King (2005) using only the Trails A portion of the test. Lastly, using a backward masking paradigm, Stevens et al. (2004) found significant visual perception processing speed deficits in persons with BPD compared with controls.

Not all research findings support the idea of processing speed deficits in persons with BPD. Swirsky-Sachhetti et al. (1993) were unable to find significant processing speed deficits in persons with BPD using the Stroop Color and Word Test (Golden, 1978) and the Trail-Making Test A and B (Reitan, 1958).

Meta-analysis and Review of Neuropsychological Functioning in Persons with BPD

Perhaps the most efficient and pragmatic approach to synthesize and evaluate the literature was a meta-analysis of neuropsychological studies involving persons with BPD conducted by Ruocco (2005). An advantage of using a meta-analysis is that it allows for

an amalgamation of archival data that provide a more comprehensive analysis of existent findings than would be possible in any one study. Ruocco (2005) chose studies to include in his meta-analysis that satisfied certain criteria: reported sufficient data allowing for calculation of effect sizes; a clinical sample of BPD persons; neuropsychological tests that were standardized, valid and produced reliable scores; BPD diagnosis obtained using DSM or ICD-10 classification; and publication in a peer review journal. The meta-analysis categorized each of the neuropsychological tests used in the studies into one of six domains: attention, cognitive flexibility, learning and memory, planning, speeded processing, and visuospatial skills.

Ruocco (2005) expected that amalgamating the individual findings across studies would produce a coherent characterization of the neurocognitive features of the disorder. The results from the meta-analysis show that significant differences exist between persons with BPD and healthy comparison controls in several areas of neuropsychological functioning. Specifically, individuals with BPD performed more poorly than healthy control persons in all areas of functioning assessed with effect sizes for these domains ranging from medium to large (Ruocco, 2005).

Several interesting interpretations can be made based on the findings from Ruocco's (2005) meta-analysis. First, the significant effect sizes in the areas of attention, cognitive flexibility, and speeded processing are possibly indicative of dysfunction in frontal lobe functioning in individuals with BPD (Mitchell, Johnson, Raye, & Greene, 2004; Monchi, Petrides, Petre, Worsley, & Dagher, 2001; Stuss, Floden, Alexander, Levine, & Katz, 2001). These findings are corroborated by other studies that have found evidence of frontal lobe pathology in persons with BPD symptomotology in a sample of

normal young adults (Ruocco & Trobbst, 2003) and in a head-injury sample (Ruocco & Swirsky-Sachetti, 2005). Additionally, the large effect size in planning and medium-to-large effects size for the visuospatial domain also support the idea of dysfunction in frontal lobe and potentially parietal lobe (Aleman, et al., 2002; Fincham, Carter, van Veen, Stenger, & Anderson, 2002; Jacobs & Anderson, 2002; Newman, Carpenter, Varma, & Just, 2003). Lastly, the effect sizes for learning and memory indicate deficits that possibly denote dysfunction in frontotemporal areas (Johnson, Saykin, Flashman, McAllister, & Sparling, 2004; Kelley et al., 1998).

Results from Ruocco's (2005) study provide support for the use of the Jacksonian model as an etiological explanation for BPD symptomotology (Meares, Stevenson, & Gordon, 1999). The Jacksonian model posits that many of the symptoms associated with BPD (e.g., emotional dysregulation, identity disturbance, dissociation) are the result of disrupted neural connections between the prefrontal cortex and brain regions responsible for higher cognitive functioning. The model suggests that neurocognitive impairment is diffuse and global rather than isolated to localized areas of the brain and proposes that BPD is the result of a cascade of neuropsychological impairments. Ruocco's (2005) findings of neuropsychological dysfunction in numerous domains associated with frontal lobe operations are congruent with the Jacksonian model.

The evidence of a broad range of neurocognitive dysfunction in persons with BPD that is based on averaged scores from numerous neuropsychological measures makes it difficult to draw conclusions regarding specific areas of brain pathology (Ruocco, 2005). Nonetheless, according to Ruocco (2005) it is valuable to acknowledge the localizing abilities of certain neuropsychological tests. For example, greater deficits in nonverbal

abilities were found compared with verbal abilities in the meta-analysis, suggestive of frontotemporal dysfunction that is more strongly lateralized to the right hemisphere. These findings from Ruocco (2005) are consistent with other studies that have indicated specific dysfunction in the right hemisphere of persons with BPD (Dinn et al., 2004; Niederhofer, 2004). To make more conclusions about specific areas of dysfunction, additional research is needed that utilizes both neuropsychological tests and neuroimaging techniques. Studies utilizing functional magnetic resonance imaging (fMRI) techniques with persons diagnosed with BPD revealed evidence of possible deficits in frontal and temporal lobes (Driessen et al., 2004; Tebartz van Elst et al., 2003), and deficits in amygdala functioning (Donegan et al., 2003). Additionally, Ruocco (2005) notes that fMRI studies demonstrate the presence of a more diffuse characterization of brain dysfunction in persons with BPD.

While Ruocco's (2005) meta-analysis generates a clearer picture of neuro-psychological dysfunction in persons with BPD, it is important to note that there was considerable heterogeneity in the effect sizes for several domains. Factors that likely contributed to the variability in effect sizes include the broad range of tests used across studies, the variety of diagnostic systems used to define and assess for BPD pathology and differences in subject medication regimens. External validity of the findings is limited due to the fact that most of the amalgamated sample was female and many samples included subjects with varied co-occurring Axis I and Axis II diagnosis. The inclusion of BPD subjects with co-occurring psychiatric disorders not only adds to heterogeneity of effect sizes but also limits the extent to which findings can be

generalized to persons with BPD that have other concurrent Axis I and/or Axis II disorders (Ruocco, 2005).

The existing evidence that demonstrates performance deficits for individuals with BPD in one or more area of cognitive functioning raises interesting questions. For example, how are such persons affected by these cognitive impairments? How do impairments in memory, attention, and processing speed affect persons with BPD in classroom settings, in therapy sessions, in social scenes, or during complex interpersonal interaction? Research that attempts to answer these questions is important clinically because it provides practitioners with vital information that can be incorporated into treatment planning and help inform therapists about particular client needs or necessary areas of focus.

Ruocco (2005) states that the finding of deficits in attention, memory and learning skills in persons with BPD suggests that clinicians be aware that insufficiencies in these areas of cognitive functioning may interfere with clients' ability to communicate effectively and engage in therapy. Interestingly, research by Burgess (1991) involving persons with BPD found a significant correlation between suicide and neuro-psychological deficits but not between suicide and level of depression. Another important clinical consideration is the question of whether it is prudent to prescribe medications for persons with BPD that have cognitive side effects, or if it is advisable to prescribe medications with side effects that do not interfere as much with cognitive functioning. Ruocco (2005) also advises clinicians be aware of the extent to which neuropsychological dysfunction in BPD clients act as risk factors for suicide and/or parasuicidal behaviors.

Validity and Effort in Assessment Data

A critical component to interpreting data from a neuropsychological assessment is evaluating the validity of the information provided by the examinee. Bush et al. (2005) note that several factors have the potential to compromise the validity of test data including secondary gains, resistance to the assessment and/or examiner, a client's confusion regarding the purpose of the evaluation, or existing client factors that could interfere with giving optimal effort on a neuropsychological evaluation (e.g., poor client sleep, chronic pain). Travin and Protter (1984) and Slick, Sherman, and Iverson (1999) note that the motivation to provide invalid responses may be due to a number of reasons including both conscious and unconscious factors. While the potential for diminished effort on neuropsychological tests is typically highest in forensic settings, invalid performance resulting from a client's conscious or unconscious exaggeration or fabrication is also possible in clinical settings (Bush et al., 2005). In addition to the factors identified above that have the potential to compromise test data validity, clinical factors may also interfere with a person's ability to successfully participate in neuropsychological testing (Bush et al., 2005).

To determine whether or not test data are valid requires establishing if the examinee gave accurate and comprehensive information regarding his or her symptoms and history, and if the examinee provided appropriate effort on the tests (Bush et al., 2005). Several terms have been provided by Bush and colleagues (2005) that relate to the validity of test information or test data: 1) symptom validity; 2) response bias; 3) effort; 4) malingering; 5) dissimulation. The authors define symptom validity as the accuracy or honesty of the examinee's signs, symptoms, or performance on neuropsychological tests,

and response bias is an attempt to deceive the examiner by providing inaccurate or incomplete responses or effort. Bush et al. (2005) define effort as the investment to perform at capacity levels or to perform well, malingering is the deliberate production of false or exaggerated symptoms motivated by external sources, and dissimulation is defined as intentionally misrepresenting or falsifying symptoms by over-representing or under-representing the true status of symptoms.

Historically, the detection of invalid testing data has been informed by qualitative analysis of responses offered by the examinee (Tombaugh, 1997). Under such circumstances, assessing for patterns of inconsistencies in responses was the principal technique used to identify invalid test data. However, as noted by Tombaugh (1997) qualitative analysis of patterns was not always successful in detecting invalid test data. Meehl (1954; 1997) and others (e.g., Dawes, Faust, & Meehl, 1989; Heaton, Smith, Lehman, & Vogt, 1978) have argued that clinical judgment without supporting psychometric data is largely and oftentimes inaccurate when compared with actuarial judgment based on empirical evidence from objective measures. For this reason, researchers and clinicians have proposed that neuropsychologists supplant basic clinical judgment with scientific evidence produced by psychometrically sound validity-detection techniques.

Several standard neuropsychological instruments including the WMS-III (Mittenberg, Azrin, Millsaps, & Heilbronner, 1993; Wechsler, 1997b), the Category Test (DiCarlo, Gfeller, & Oliveri, 2000), and the WAIS III – Digit Span (Heinly, Greve, Love, Brennan, & Bianchini, 2004; Wechsler, 1997a) contain validity markers that may provide evidence of invalid performance on select cognitive measures (Greve, Bianchini,

Mathias, Houston, & Crouch, 2002). However, oftentimes these validity indicators fail to provide sufficient sensitivity or specificity (Bernard, McGrath, & Houston, 1996; DenBoer & Hall, 2004; Greve & Bianchini, 2002; Hiscock, Branham, & Hiscock, 1994; Suhr & Boyer, 1999).

To correct for the relatively poor ability of standard instruments to accurately detect level of effort in testing, several specialized tests of effort have been designed. Most tests of effort utilize symptom validity testing which is based on a forced choice paradigm (Pankratz & Binder, 1997; Slick, Hopp, Strauss, Hunter, & Pinch, 1994; Slick, Hopp, Strauss, & Spellacy, 1996). Using this type of test format involves presenting a subject with an initial stimulus, or target item, followed by asking the subject to select the target item when it is paired with a foil. Due to the fact that subjects have a minimum 50% chance of correctly guessing the answer with this type of testing paradigm, symptom validity tests detect low effort partly by identifying those individuals who respond below chance accuracy (Loring, 1995). Because individuals intending to give poor effort rarely perform below chance-level, researchers have established cut off scores for several tests of effort that are set above chance level of responding and below scores generated by neurological patients (Binder, 1993; Binder & Willis, 1991; Tombaugh, 1997). Therefore, when a subject scores lower than an individual with diagnosed brain injury would typically score there is a strong possibility that the subject is not giving full effort.

Contemporary neuropsychological research has emphasized the effect of decreased performance on neuropsychological tests completed by individuals with depression, head injury, and chronic pain (Gervais et al., 2001; Green & Iverson, 2001; Green, Rohling, Lees-Haley, & Allen, 2001; Rohling, Green, Allen, & Iverson, 2002;

Suhr, 2003). In contrast to the observed decreased performance on neuropsychological tests with depressed individuals, research involving clinically depressed individuals' performance on a measure of effort, the Test of Mental Malingering (TOMM; Tombaugh, 1997), revealed that level of depression does not influence an individual's performance on this test (Ashendorf, Costantinou, & McCaffrey, 2004).

Given the recent call to include validity tests in neuropsychological testing, it seems worthwhile to explore in more depth the possible relationships between BPD and effort. The lack of research concerning effort with BPD populations stands as a noteworthy omission within empirical research in light of recommendations made by Bush et al. (2005) and others whom maintain that using measures to establish evidence of sufficient effort is a valuable and necessary practice to ensure valid data in neuropsychological testing. Because neuropsychologists are expected to account for the validity of test results, the inclusion of symptom validity tests should be considered a necessary component in any neuropsychological battery.

Limitations to the Existing Literature

There are several possible reasons for the inconsistent findings in neuropsychological literature involving persons with BPD including differences in choice of diagnostic assessment techniques, low sensitivity of cognitive measures, inadequate sample size, subject heterogeneity, and selection criteria. Ruocco (2005) identified what he believes to be major limitations within the existing research that contribute to the inconsistent findings. The first limitation regards the different ways in which BPD has been operationally defined from one study to another study. Some studies have used subjects' self-report to determine BPD diagnoses, other studies have used semi-structured

interviews, and some have used unstructured interviews. A second limitation concerns the broad assortment of neuropsychological measures used from one study to the next. The use of a wide range of different neuropsychological tests complicates the ability to make clear statements about deficits because of varying degrees of construct validity for individual tests. Ruocco (2005) argues that these sources of variability have muddied the waters and made it difficult to form a clear picture of the possible brain pathology that may underlie BPD.

Ruocco (2005) noted that insufficient sample size is yet another limitation and likely partly responsible for the inconsistencies and lack of significant findings observed in the literature:

> In light of the result of the present meta-analysis, the seeming inconsistencies observed across past neuropsychological investigations of BPD appear to be artificial. Based on the effect sizes obtained across the six neurocognitive domains, it is apparent that most prior investigations lacked sufficient power to detect potential differences between BPD and healthy comparison groups on common neuropsychological tasks (p. 199).

Ruocco (2005) adds that the mean sample size of existing studies is less than half of what is necessary to detect the effect with sufficient statistical power. One solution offered by Haase and McCaffrey (2004) that is capable of producing more consistent findings within and across neuropsychological studies includes aggregating scores from several measures that tap into specific neuropsychological constructs and conducting between-group analyses using the amalgamated test scores. Such a method was utilized

by Monarch et al. (2004) who found significant dysfunction in their sample of persons with BPD in seven out of nine cognitive domains investigated.

Monarch et al. (2004) also noted several limitations to existent research that preclude a clear neuropsychological characterization of BPD. The authors stated that almost no neuropsychological studies involving persons with BPD assessed attention and concentration adequately. That is, those studies that assessed attention neglected to implement a broad range of attention measures and/or utilized attention measures with debatable construct validity. Attention is an important ability to assess because it can affect all areas of cognitive functioning. Failure to include adequate measures of attention may limit accurate interpretation of meaningful results and serve as an impediment to constructing an accurate neuropsychological profile of BPD.

Another limitation identified by Monarch et al. (2004) includes the fact that most studies focused on assessing one area of cognitive functioning such as executive functioning rather than evaluating multiple cognitive domains. The authors report that those studies that did assess multiple domains used a single test, subtest or item, to evaluate an area of cognitive functioning. The argument against using a single measure to assess cognitive functioning in a given area is that it does not produce ample data with which to make sound, reliable statements and stands as a marked methodological deficiency in the literature.

The Current Study

The current study assessed for the presence of cognitive deficits in persons with BPD in the areas of verbal memory, processing speed, and attention. The study intended to improve on methodological shortcomings present in existent neuropsychological

literature as identified by Ruocco (2005) and Monarch et al (2004). The limitation of inconsistent operational definition of BPD in the existing research was addressed in this study by using the most common screening measure of BPD, the Structured Clinical Interview for *DSM-IV* Personality Disorders (SCID-II, First, Gibbon, Spitzer, Williams, & Benjamin, 1997).

A second limitation, the use of varied measures to assess cognitive functioning in neuropsychological research, presents a challenge for one study to address because researchers often choose measures not necessarily based on their psychometric properties, but rather because of personal preferences and allegiances to certain tests with which they are most familiar and comfortable (Rabin, Barr, & Burton, 2005). However, in an effort to address this limitation, the selection of neuropsychological measures used in this study was informed by research regarding their sound psychometric properties (e.g., score reliability and construct validity) and effect sizes of measures as reported in the meta-analytic review by Ruocco (2005). Hopefully, future research will include similar measures to assist in increasing standardization of neuropsychological assessment batteries.

A third proposed improvement concerned the limitation of insufficient number of tests devoted to assess a single cognitive domain. To correct for this shortcoming participants in this study completed several neuropsychological measures for each domain being evaluated and a composite score was calculated for each domain. Specifically, the test battery included two measures of verbal memory that yielded five scores, three measures of processing speed that yielded six scores, and three measures of attention that yielded six scores. The current study used a method of analyzing data that

entailed calculating a composite score by aggregating subtest scores from multiple measures within each cognitive domain. Use of a composite score allowed for between-group analyses based on amalgamated scores. The study also evaluated subject performance using a general index of neuropsychological functioning that contained scores from all three domains assessed. In this way an overall measure of neuropsychological performance for BPD persons and healthy control was obtained and analyzed.

The inclusion of several measures of attention constituted a fourth methodological improvement. The inadequate assessment of attention that has been noted in the literature was addressed by including three measures that assessed this domain. One last methodological flaw of other studies was insufficient sample size. This issue was addressed by conducting a power analysis to determine how many subjects were needed to maximize the likelihood of significant findings.

It has been well documented that individuals with depression suffer from more severe cognitive deficits relative to persons without depression (Gfeller, Chibnall & Duckro, 1994; Kizilbash et al., 2002; Kurtz & Morey, 1999) and that depression often co-occurs with BPD (Comtois et al., 1999; Joyce et al., 2003). The co-occurring presentation of the two disorders makes it difficult to determine which disorder contributes more to the cognitive deficits. The strength of the relationship between depression and BPD was investigated as was the influence of co-occurring depression and BPD on neuropsychological test performance.

A review of the literature reveals that no one has yet looked at the role of effort in testing performance with this population. Therefore, the current investigation included a

measure of effort to explore possible relationships between test effort and BPD. Lastly, the study also explored the extent to which a neurological disorder diagnosis in BPD persons affects performance on neuropsychological tests.

Hypotheses

1. Individuals with BPD will demonstrate significant deficits on a general index of neuropsychological performance relative to control subjects.

2. Individuals with BPD will demonstrate significant deficits in attention relative to control subjects.

3. Individuals with BPD will demonstrate significant deficits in verbal memory relative to control subjects.

4. Individuals with BPD will demonstrate significant deficits in processing speed relative to control subjects.

5a. Individuals with BPD will have higher levels of depression relative to control subjects.

5b. Individuals with BPD will have depression scores that correlate significantly with scores on the general index of neuropsychological performance.

Exploratory Areas

In addition to the hypotheses identified above, the current study included two exploratory areas:

A. One exploratory area for the current study was a measure of effort that filled in a gap in the literature by evaluating the relationship of effort in testing to BPD. Also, assessing for level of effort helped determine the validity of test data from this study.

B. A second exploratory area for the current study included evaluating the extent to which neurological test performance is affected by the presence of both BPD and a neurological disorder diagnosis (e.g., trauma, encephalitis, or epilepsy) compared with test performance by individuals diagnosed only with BPD.

Method

Participant Recruitment

Experimental participants were recruited from mental health centers and private practitioners in Missoula, MT and from Montana State Hospital in Warm Springs, MT. Experimental participants from Missoula were recruited via referrals from clinicians in private practice or working at Western Montana Mental Health Center (WMMHC).

Prior to beginning recruitment, the researcher met with directors, clinicians and psychiatrists from WMMHC and local clinicians to describe the study and ask for their assistance in making appropriate referrals. Written materials were provided with information about the study and who might be eligible (see Appendix A), as well as flyers advertising and briefly describing the details of the study with contact information for the researcher (see Appendix B). Clinicians and psychiatrists were asked to provide a flyer to those clients with a BPD diagnosis, or BPD features, who may be interested in participating in the study. Mental health professionals were instructed to refer only those clients who are aware of their BPD diagnosis, or that they have BPD features and may have BPD, to avoid participants inadvertently learning about this diagnosis/symptoms via participation in the study. Prior to running any BPD participants, the researcher gained permission from the CPC director, Jen Robohm, Ph.D., to use the clinic facilities to administer the assessments.

Experimental participants were also recruited from Montana State Hospital in Warm Springs, MT. The procedure for recruiting participants at this site was based on prior research protocols used by a UM clinical psychology graduate student and approved by the hospital's Institutional Review Board (IRB). Participant recruitment began with the researcher meeting with the hospital director, Polly Peterson, Ph.D., to gain permission to provide a presentation on the study to the hospital IRB committee. To determine which patients were likely study participants (e.g., met BPD criteria) the researcher consulted with the hospital director who reviewed patients' files. Patients were excluded if their symptoms were not sufficiently stabilized, if they were unable to consent to participation, or if they exhibited behavioral problems that would make their participation problematic.

Those patients that were determined appropriate for the study were approached by the hospital director and given a brief explanation of the study and the extent of their involvement should they choose to participate (i.e., completing questionnaires and tests for approximately 2 hours). Participants were informed that they would be financially compensated for their time and effort. The director answered the patients' questions about the study and then asked if they would be interested in participating in the study. Patients who expressed interest in participating were scheduled for a testing appointment with the researcher.

Control participants consisted of undergraduate students enrolled in an Introduction to Psychology course at UM who received 6 credits towards their experimental participation requirement for the course. All control participants were at least 18 years of age. Control participants signed up to participate in the study by

recording their names and contact information on a sign-up sheet posted in the psychology department at UM. The researcher contacted the students, explained to them what their participation in the study would entail and answered their questions. Those students that were interested in participating were scheduled for an appointment at the neuropsychology research lab on The University of Montana campus to complete the assessment. Because BPD is a diagnosis found predominately in females (about 75%; American Psychiatric Association, 2000), and this percentage was reflected in the number of females in the clinical sample, control subjects were matched to the clinical sample on the dimension of gender.

All aspects of participant recruitment and participation complied with all policies and procedures as outlined and approved by the IRB committee at The University of Montana. Additionally, participant recruitment and participation at the state hospital complied with all policies and procedures as outlined by the hospital's internal IRB. All experimental participants were informed that they would be paid $10 for their participation in the 2-hour study. Control participants were informed that they would receive 6 credits toward their course requirements.

Informed Consent Form

All participants completed an informed consent form that outlined their involvement in the study, explained confidentiality and the limits to it, as well as notified them of their rights as a research participant. Because participants were recruited from several different sites, three tailored informed consent forms were used (see Appendices C, D, and E). Each form included specific information relevant to the recruitment site

such as where confidential data would be stored, who would have access to them, and the type of compensation offered to the participants (e.g., course credit, monetary).

Human Subjects Protections

The current study recruited participants from mental health clinics in Missoula, MT and also from Montana State Hospital. Patients at the hospital are presumed by the hospital physicians to have the ability to consent to treatment unless they demonstrate that they cannot do so. The researcher consulted with the hospital director about new admissions and all patients who participated in the current study were determined to have the ability to consent to treatment and participation in the research study.

Upon determining that a patient was able to consent to research participation and met the study criteria, the hospital director briefly explained the research opportunity and asked if he or she was interested in participating. The director then scheduled the patient for the next available assessment time with the researcher. Prior to beginning the assessment, the researcher explicitly informed each patient that their participation was completely voluntary and they were permitted to stop participating at any time. Each participant was informed that he or she would be compensated $10 for completing the study and $2 if he or she did not meet study criteria. The researcher verbally described the study in more detail, discussed the consent process, and obtained informed consent from the participant. Additionally, a HIPAA-required authorization for Montana State Hospital to release protected health information to The University of Montana was signed by the participant (See Appendix F). Copies of these documents were made available to the participant. To protect the participant's identity and ensure confidentiality, the originals of these forms were held in a file cabinet separate from the participant's raw

data. Only the participant, the researcher and the hospital staff had access to these forms which were destroyed one year after the completion of the study.

All raw data for this study was identified with a 3-digit participant number. The researcher stored all raw data, original informed consents, and the identification key in locked file cabinets. In accordance with HIPAA standards, only non-identifiable raw data and electronic data were taken from the hospital campus for data analysis at The University of Montana's Department of Psychology. All data were reviewed by the hospital director before being removed from the hospital campus. The informed consents and other data will remain in locked cabinets at the hospital until one year following the completion of the study, at which time all confidential study materials were shredded.

Data gathered from Missoula participants were stored in locked file cabinets in the neuropsychology research lab at The University of Montana's Department of Psychology. Forms with identifying information (e.g., informed consent forms,) and file cabinet key were kept separate from the other data. After one year of the study's completion the researcher had all raw data destroyed. The researcher maintained each participant's data electronically, identified only with the participant number.

Several precautionary measures were taken to properly care for participants that express significant distress. The primary investigator who administered all measures was a master's level clinician with thorough training in working with individuals with BPD and in risk assessment and debriefing procedures as well as having training in neuropsychological assessment. All experimental participants were participating in on-going mental health treatment. Clinical and control participants underwent a debriefing following the study which included inquiring about their level of distress, including

thoughts of suicide or harming others, the primary investigator had a standard protocol in place that he could follow. No participants in the current study reported feeling distressed as a result of completing the assessment.

Demographic Questionnaire

A demographics questionnaire was used to gather information regarding participants' gender, age, ethnic background, marital status, and level of education. The questionnaire also assessed for any diagnosis of a neurological disorder (e.g., seizure disorder) or history of head trauma (see Appendix G).

Measures

General WMS-III and WAIS-III Normative Data Information

Several subtests from the WMS-III (Wechsler, 1997b) and the WAIS-III (Wechsler, 1997a) were used in the current study (i.e., Logical Memory, Digit Symbol, Symbol Search, Spatial Span). The norms for the WMS-III were developed based on scores from a sample of 1,250 persons spanning 13 age groups between 16 and 89 years old, with 100 people in the first 11 age groups, plus 75 people ages 80-84 and 75 people ages 85-89 (Wechsler, 1997b). The authors indicated that the normative sample contained adequate distribution of ethnic variability, with data gathered at 28 different U.S. sites. The authors note that the average subtest internal consistency reliability coefficient ranges in the .80s and .90s.

A strength of the WAIS-III subtests is their relatively complete and representative standardizations with 2,450 participants between the ages of 16 and 89 years of age comprising the standardization sample (Wechsler, 1997a). The normative group was divided into 13 age groups and stratified on major demographic variables such as age,

sex, education level, and geographic location based on the U.S. census data. The authors stated that extensive testing of the subscales was conducted to establish acceptable score reliability and construct validity, but provided no reliability coefficients.

Attention Tests

Spatial Span.

The Spatial Span subtest from the WMS-III (Wechsler, 1997b) is considered a measure of visuospatial attention (Lezak et al., 2004). The internal reliability coefficient for the Spatial Span subtest has an average of .79. The Spatial Span subtest consists of 10 cubes mounted on a board in an irregular arrangement. The task requires participants to first observe the examiner tapping the cubes in prearranged sequences. The participant must then reproduce the tapping pattern in the exact order that the sequence was presented. The subtest contains a series of both forward sequences as well as backward sequences of increasing length and complexity. Scoring involves summing the number of sequences correctly reproduced by the participant. The current study used the scores from the forward sequence, backward sequence, and total score of Spatial Span for statistical analysis.

Seashore Rhythm Test.

The Seashore Rhythm Test (SRT; Seashore, Lewis, & Saetveit, 1960) has been categorized as a sensitive measure of attention and concentration (Lezak et al., 2004). The test requires participants to discriminate between 30 like and unlike pairs of brief musical beats. Scoring for the SRT is completed by summing the number of correct responses. The SRT is part of the Halstead-Reitan Battery that includes a normative

sample of 196 individuals with a mean age of 45.3 years (SD=19.0), and containing 67.9% males (Heaton, Grant, & Mathews, 1991). Internal reliability coefficients of .77 using split-half and .78 using odd-even have been reported for the test (Bornstein, 1983). The current study used the total correct scores from the SRT for statistical analysis.

The Ruff 2 & 7 Selective Attention Test.

The Ruff 2 & 7 Selective Attention Test was designed to measure aspects of sustained and selective visual attention (Ruff & Allen, 1996). The test is constructed based on the notion that attention consists of two proposed mechanisms: Controlled Search and Automatic Detection. As such, the measure assesses differences between automatic (obvious distracters) and controlled (less obvious distracters) visual search. The Ruff 2 & 7 test measures selective attention through comparison of response accuracy and speed in Automatic Detection versus Controlled Search processing tasks.

The normative group for the Ruff 2 & 7 Test contained 360 healthy volunteers sampled from California, Michigan, and the Eastern seaboard and roughly represented the 1980 U.S. census proportions with regard to race (Ruff & Allen, 1996). The normative sample was stratified according to age (16-24 years, 25-39 years, 40-54 years, and 55-70 years) and gender (180 woman and 180 men). Test-retest reliability for the measure has been reported to be in the .84 to .97 range (Ruff & Allen, 1996).

The automatic condition consists of lines of randomly mixed capital letters with the targets 2 and 7 interspersed. The controlled condition contains lines of random single digits with the targets 2's and 7's randomly interspersed. The test includes 20 randomly ordered sets of automatic and controlled conditions. Each set contains three lines with 20 randomly ordered characters on each line for a total of 60 characters, 10 of which are 2's

an 7's. The time allowed to complete the measure is 5 minutes. The participant is instructed to cross out all the 2's or 7's in each line moving from left to right. Scoring involves summing the number of hits (correctly identified 2's and 7's), omission errors (number of times a 2 or 7 was not marked), and commission errors (number of times a distracter is marked as a target). The number of hits and errors are summed and used to calculate an automatic search score and a controlled search score. The current study used the Automatic Detection Accuracy score and the Controlled Search Accuracy score for statistical analyses. The present study's inclusion of the Ruff 2 & 7 Test as a measure of sustained attention was a welcomed addition to the literature concerning attention functioning in persons with BPD because other than Lenzenweger et al. (2004), no research has measured this aspect of attention.

Verbal Memory Tests

Logical Memory.

The Logical Memory subtest from the WMS-III is considered a measure of verbal memory (Wechsler, 1997b). The examiner reads two stories (Story A and Story B) and instructs the examinee to try to remember as many details about each of the stories. An immediate free recall score is obtained after reading each story once and is added to a second free recall score obtained for Story B after the examiner reads that story a second time to yield a recall total score (Logical Memory I). A 30-minute delayed recall score (Logical Memory II) is obtained for Stories A and B.

The Logical Memory subtest contains a story unit score that is obtained by calculating the number of story details included on the scoring protocol for Stories A and B that are correctly recalled by the examinee. Each story has 25 details with accurate

recall responses awarded a "1" and omitted or incorrect recall responses assigned a "0." This scoring system allows for a maximum possible score of 25 points for each story, or a maximum possible first recall score of 50 points for both story A and B. A second recall unit score is also calculated for story B with a maximum possible score of 25 points. When the second recall unit score is added to the first recall unit score, the maximum total recall score is 75 points for Logical Memory I. A similar scoring system is used for Story A and B at 30-minute delay recall, except that neither story is read again, and only one recall unit score is calculated for Story B at delay. The maximum possible score at delayed recall is 50 points for the unit score.

Scores for the Logical Memory I (i.e. immediate recall) portion of the WMS-III have reported reliability between .81 and .91 across the 13 age groups within the normative sample (Wechsler, 1997b). Logical Memory II (i.e., delayed recall) score reliabilities ranged from .71 to .87 in the normative sample (Wechsler, 1997b). The current study used the recall total scores for Logical Memory I and Logical Memory II for statistical analysis.

California Verbal Learning Test-II.

The California Verbal Learning Test-II (CVLT-II; Delis, Kramer, Kaplan, & Ober, 2000) is designed to use semantic associations as a strategy of learning words, and is therefore a test of verbal learning. The CVLT-II contains a List A that consists of 16 words in four categories: vegetables, animals, ways of traveling, and furniture. The test contains a List B, an interference task that also contains 16 names of vegetables, and animals along with musical instruments and parts of buildings. As such, the CVLT-II is a measure of the interaction between verbal memory and conceptual ability (Lezak, 2004).

The examinee reads each word at a rate of about one word per second and the examinee is instructed to try and recall as many words from the list as he or she is able to recall in any order. List A has five trials with the examiner re-reading the list aloud after each of the first four trials. After the fifth trial, List B is read to the examinee and he or she is asked to recall as many of the items only from List B that he or she is able to recall. Then, two "short-term delay" recall tasks of List A are administered. The first short-term delayed free recall task asks the examinee to name as many words from List A in any order. The second short-term delayed cued recall task asks the examinee to recall as many words from List A that fit into each of the four categories, one category at a time. Finally, a 20-minute long-term delayed free recall trial, similar to the short-term delay free recall, is administered.

Scoring the CVLT-II produces scores for total correct, total repetitions, total intrusions, short-term delay and long-term delay. The CVLT-II has a normative sample of 1,087 adults across seven age groups ranging from 16 to 89 years old and stratified according to the U.S. consensus by age, sex, ethnicity, educational level, and area of the country (Lezak, 2004). The authors of the test reported split-half reliability coefficients of .77 to .86 (Delis, Kramer, Fridlund, & Kaplan, 1990). The current study used total correct, short-term delay free recall, and long-term delay free recall scores from the CVLT-II for statistical analysis.

Processing Speed Tests

Digit Symbol.

The Digit Symbol subtest from the WAIS-III is a measure of processing speed (Wechsler, 1997a). The task consists of a number of rows containing small blank

squares, each paired with a randomly assigned number from one to nine. A key is provided above the rows that indicate which number corresponds with each symbol. The examinee is given a practice trial of seven symbols to ensure he or she understands the task. Then, the examinee is asked to fill in as many of the blank squares as possible with the symbol that is paired with the number above the blank space during a timed trial of 120 seconds. The examinee's score is the number of squares he or she correctly filled in with the symbol. Test-retest reliability coefficients for the Digit Symbol subtest have been reported as high in the .82 to .88 range (Wechsler, 1981). The current study used total correct score from the Digit Symbol for statistical analysis.

Symbol Search

The Symbol Search subtest from the WAIS-III is also considered a measure of processing speed (Wechsler, 1997a) with an average internal consistency coefficient of .77. The test entails showing the examinee a series of lines on which there are two cues of nonsense figures followed by a string of other nonsense figures. The examinee is asked to mark "yes" or "no" for each line if either of the cue figures is replicated in the subsequent string of nonsense figures. Following a sample trial and a practice trial, the examinee is encouraged to complete as many of the items as he or she can in 120 seconds. The score is calculated by summing the number of items the examinee completed correctly. The current study used total correct score from the Symbol Search for statistical analysis.

Paced Auditory Serial Addition Test.

The Paced Auditory Serial Addition Test (PASAT; Gronwall, 1977; Gronwall & Sampson, 1974) has demonstrated high internal consistency with correlations ranging between .76 to .95 (Sherman, Strauss, & Spellacy, 1997). Stuss, Stethem, and Pelchat (1988) examined a normative group of 90 participants equally divided among three age groups (16-29, 30-49, and 50-69). The PASAT is capable of detecting deficits in processing speed in patients with a wide variety of neuropsychological syndromes (Tombaugh, 2006). The test consists of presenting the examinee with 61 randomized numbers that require him or her to sum each number to the one that immediately preceded it. For example, if the numbers follow the order "3, 7, 2, 1, 4," then the examinee's correct responses, beginning immediately after the second number, would be "10, 9, 3, 5." There are four trials and with each trial the numbers are presented with increasing speed such that numbers in the first trail are presented one every 2.4 seconds, every 2 seconds for the second trial, every 1.6 seconds for the third trial, and every 1.2 seconds for the fourth trial. The examinee is provided first with a 10-digit practice series presented at 2.4 seconds. Scoring is most commonly reported by calculating the number of correct responses for each trial or the mean score for all four trials. The current study used participant scores from the four trials in the statistical analyses.

Test of Effort

The Test of Memory Malingering.

The *Test of Memory Malingering* (TOMM; Tombaugh, 1996; 1997) is the most popular symptom validity measures employed by private practitioners as well as by

neuropsychologists with expertise in malingering research (Shandera, Hall, DenBoer, & Crouse, 2004; Slick, Tan, Strauss, & Hultsch, 2004). The test includes 50 line-drawings of common objects that are presented to the client each for 3 seconds with a 1 second inter-stimulus interval. The TOMM has been shown to demonstrate adequate validity when used with various psychiatric populations, including individuals diagnosed with moderate to severe depression and anxiety (Ashendorf et al., 2004). Additionally, the TOMM has been shown to be an effective validity measure when used for a wide spectrum of neurological populations, including litigating and non-litigating patients with traumatic brain injuries (TBI) and patients with dementia, aphasia, psychiatric outpatients with depression and anxiety, and individuals experiencing acute pain (Ashendorf et al., 2004; Etherton, Bianchini, Greve, & Ciota, 2005; Gansler, Tombaugh, Moczynski, & Rees, 1995; Rees, Tombaugh, Gansler, & Moczynski, 1998). The current study used total correct scores from the TOMM Trial 2 for statistical analysis.

Beck Depression Inventory

The Beck Depression Inventory, Second Edition (BDI-II; Beck, Steer, & Brown 1996) is a widely used measure of depressive symptomotology that produces a continuous score. All participants completed the BDI-II and information from it was used to assess for differences in the level of depression between groups and to help explore possible relationships between level of depression in persons with BPD and performance on neuropsychological tests.

Structured Clinical Interview for the DSM-IV Personality Disorders

The BPD items from the SCID-II (First et al., 1997) was used to determine presence of the BPD among participants. The SCID-II is a semistructured interview that parallels the *DSM-IV-TR* personality disorders (Rogers, 2001). The measure uses a 3-point rating system: 1=absent or false, 2=subthreshold, 3=threshold or true. Each BPD diagnostic criterion has about two questions on the SCID-II and it is estimated to take approximately 25 minutes to complete. Rogers (2001) notes that the SCID-II is a measure that has shown to produce scores that demonstrate acceptable score reliability and validity as a measure of personality disorders. SCID-II interviews were recorded.

Procedure

The researcher spoke with all potential participants from Missoula and provided each with a standardized description of the nature and length of the study. Potential participants had an opportunity to ask the researcher any questions they might have had. Those persons interested in participating completed a brief phone screening to gather information concerning the participants' contact information, brief demographic information (e.g., age) and whether or not BPD participants were currently involved with a mental health professional. Only participants who were seeing a mental health professional were included in the study.

All participants were assigned a 3-digit participant number and all identifying information was kept separate from other data. Participant numbers were specially coded to denote whether participants were from the state hospital, a community mental health center, or the control condition. Missoula participants with a BPD diagnosis were

scheduled for an appointment at the CPC to complete the assessment. Control participants were scheduled for an appointment at the neuropsychology research lab.

Upon arriving for their appointment, all Missoula participants completed the informed consent form, the demographic questionnaire and were then screened for BPD using the BPD items from the SCID-II (First et al., 1997). The SCID-II was used to ensure that experimental participants meet criteria for BPD and to screen out control participants who meet BPD criteria. All clinical referrals satisfied the SCID-II BPD criteria. In two cases, control participants met the BPD criteria and their data were used as part of the clinical sample and compensated by providing them with their course experimental credits. Control participant recruitment was stratified based on gender to match the experimental group.

A standardized order of test administration was used for all participants (see Appendix H). Because the verbal memory measures CVLT-II and Logical Memory require a 20 minute delay period between immediate and delay trials, during these interval periods participants completed approximately 20 minutes of measures that assess processing speed and attention. It is standard practice in neuropsychological testing to have participants complete measures during interval periods that assess different cognitive functioning domains than that being assessed by the measure with the delay trail as it allows for the most economic use of testing time (Lezak et al., 2004).

After administering all the measures, experimental participants were paid and control participants were awarded their experimental credits and thanked for their participation. Participants were debriefed by providing them with a copy of the informed

consent form and encouraged to contact the researcher or his supervisor at the numbers provided on the form should they have any questions following the study.

Participants recruited from Montana State Hospital completed the same procedure used for the Missoula participants except for the fact that they were contacted at the hospital, completed a HIPPA Authorization for Disclosure of Protected Health Information form and the assessments was conducted on hospital grounds.

Statistical Analyses

Because the study used multiple comparisons in the analysis, there was the potential for an increased risk of a Type I error (rejecting the null when it is true or erroneously finding significance when it could have occurred by chance). To protect against the probability of a Type I error, a Bonferroni adjustment was applied by dividing the desired alpha level (.05) by the number of *t*-tests (4) conducted on the dependent variables (general neuropsychological functioning, attention, verbal memory, and processing speed) to determine that a p-value < .0125 (rounded to .01) would be needed to establish significance while maintaining the Type I error rate at .05 for the group of *t*-tests.

Using Statistical Package for the Social Sciences (SPSS), a composite score was calculated for each domain of functioning (i.e., general neuropsychological functioning, attention, verbal memory, and processing speed) and utilized in the statistical analysis to compare scores from participants with BPD with scores from control participants. The process of calculating composite scores included first converting participants' raw scores into standardized *t*-scores. For some subtest scores the conversion from raw score to *t*-score was achieved through the use of normative tables provided by the test developers,

while conversion of other raw data required the use of equations for converting raw scores to *z*-scores, and then *z*-scores to *t*-scores. The decision to use *t*-scores as a standard measurement allowed for corrections such as age, gender, and other demographic information to be made.

Next, a factor analysis, specifically a principle component analysis, was conducted using subtest scores from each domain to obtain beta weights. Although a larger sample than that being collected in the current study would be required in a factor analysis to identify underlying factor structures of the neuropsychological tests, beta weights could be calculated with a factor analysis and used in the composite score calculations with the sample size utilized in the current study. Beta weights were used in the calculation of the composite scores to determine how much to weight scores from each subtest in each domain. R squared values were obtained from the factor analysis to indicate how much variance was explained in each domain by the subtests. After calculating one composite score for each participant for each domain, hypotheses were tested using a series of independent samples *t*-tests to compare mean scores on each domain from the BPD participants with mean scores from the control participants.

Results

Participants

A sample size of 56 participants was obtained, consisting of 49 women and 7 men. The control group consisted of 30 undergraduate students from the University of Montana, Missoula, MT. The clinical group included persons diagnosed with BPD and contained 21 inpatient participants recruited from Montana State Hospital in Warm Springs, MT, and 5 outpatient participants referred by private practitioners and

psychiatrists from Western Montana Mental Health Center, Missoula, MT. All but one clinical participant indicated having at lease one other psychological disorder in addition to a BPD diagnosis. Frequencies of co-occurring diagnoses for the clinical and control groups are presented in Table 1.

Table 1. Frequency of Diagnoses by Sample

Frequency of Diagnoses by Sample

Diagnosis	Clinical Group	Control Group
Major Depressive Disorder	13	1
Bipolar Disorder (I and II)	13	--
Posttraumatic Stress Disorder	10	
Generalized Anxiety Disorder	6	--
Attention Deficit/Hyperactivity Disorder	5	--
Obsessive Compulsive Disorder	4	--
Schizoaffective Disorder	2	--
Alcohol Abuse	2	--
Polysubstance Abuse	2	
Oppositional Defiant Disorder	2	--
Intermittent Explosive Disorder	2	--
Dissociative Identity Disorder	2	--
Personality Disorder, NOS	2	--
Eating Disorder (Bulimia Nervosa/Anorexia Nervosa)	1	1
Social Anxiety Disorder	1	--
Marijuana Abuse	1	--
Dysthymic Disorder	1	--
Schizophrenia	1	

Table 2 presents demographic information for the clinical and control groups. A Chi-square test for independence showed no significant gender differences between the control group and the clinical group, [χ^2 (1, n = 56) = .04, p = .69]. A Chi-square test for independence was conducted using education level but results demonstrated that the

analysis violated the assumption of minimum cases per cell. To correct for this problem and because education level was ordinal data, a Mann-Whitney U Test was used to assess for significant differences between the control group and the clinical group in level of education. Results demonstrated no significant differences in level of education between the two groups [Z (1, n = 56) = -.90, p = .93]. A Chi Square test was used to assess for significant differences in ethnicity between the control group and the clinical group. Because a small number of participants endorsed an ethnicity other than Caucasian, the ethnicity variable was dichotomized as "Caucasian" or "other races" to avoid violating the Chi Square assumption of minimum cases per cell. The results showed no significant differences in between the two groups [χ^2 (1, n = 56) = .17, p = .68]. Group differences for age were analyzed using an independent samples t-test. A significant difference between the control and clinical group was found for age [t(54) = -3.95, p < .01].

Table 2.

Demographic Information for Participants

Demographics	Control Group *n = 30*	Clinical Group *n = 26*	χ^2	*Z*	*t*
Gender			.04	--	--
Men	3	4			
Women	27	22			
Education			--	-.90	--
Some high school	0	6			
High school degree	20	8			
Some college	10	9			
College degree	0	3			
Ethnicity			.17	--	--
Caucasian	23	22			
Native American	4	1			
Asian	2	0			
Hispanic	1	3			
Age			--	--	-3.95*
Mean (SD)	20 (5.63)	27 (7.60)			

Note: $*p < .01$.

General Neuropsychological Index

The hypothesis that participants with BPD would demonstrate significant deficits on a general index of neuropsychological performance relative to control participants was analyzed using an independent samples *t*-test to compare the two group's general neuropsychological functioning composite scores. Results revealed a significant difference in mean composite scores on the dependent variable, general neuro-psychological functioning, between the control group and the clinical group [$t(54) = 10.99$, $p < .01$, $\eta^2 = .69$]. R squared calculation equaled .488 meaning that 48.8% of the variance was explained by the 17 subtests used to assess for general neuropsychological

functioning. This value may be considered adequate (Tabachnick & Fidell, 1996). Table 3 presents the means and standard deviations for the control group and clinical group for the each of the cognitive variables.

Table 3.

Means and Standard Deviations by Group for Cognitive Variables

Dependent Variable	Control Group *n = 30* Mean (SD)	Clinical Group *n = 26* Mean (SD)	*t*	*Effect Size* (η^2)
General Index	57.67 (4.97)	41.15 (6.27)	10.99*	.69
Attention	55.87 (7.09)	43.22 (8.51)	6.07*	.41
Verbal Memory	57.19 (4.81)	41.70 (7.72)	8.85*	.59
Processing Speed	56.76 (6.25)	42.20 (7.52)	7.91*	.54

Note: * $p < .01$

Attention

The hypothesis that participants with BPD would demonstrate significant deficits in attention relative to control participants was analyzed using an independent samples *t*-test to compare attention composite scores from the two groups of participants. Results from the analysis revealed a significant difference in mean composite scores (see Table 3) on the dependent variable, attention, between the control group and the clinical group [$t(54) = 6.07$, $p < .01$, $\eta^2 = .41$]. R squared calculation for attention equaled .462, meaning that 46.2% of the variance was explained by the six attention subtests used in the study. This value may be considered adequate (Tabachnick & Fidell, 1996).

Verbal Memory

The hypothesis that participants with BPD would demonstrate significant deficits in verbal memory relative to control participants was analyzed using an independent samples *t*-test to compare the verbal memory composite scores from the two groups of

participants. Results revealed a significant difference in mean composite scores (see Table 3) on the dependent variable, verbal memory, between the control group and the clinical group [$t(54) = 8.85$, $p < .01$, $\eta^2 = .59$]. R squared calculation for verbal memory equaled .792, meaning that 79.2% of the variance was explained by the five verbal memory subtests included in the study. This value may be considered quite good (Tabachnick & Fidell, 1996), and lends support for the researcher's choice of measures as valid instruments to assess verbal memory.

Processing Speed

The hypothesis that participants with BPD would demonstrate significant deficits in processing speed relative to control participants was analyzed using an independent samples *t*-test to compare the processing speed composite scores from the two groups of participants. Results showed a significant difference in mean composite scores (see Table 3) on the dependent variable, processing speed, between the control group and the clinical group [$t(54) = 7.91$, $p < .01$, $\eta^2 = .54$]. R squared calculation for processing speed equaled .673, meaning that 67.3% of the variance was explained by the six processing speed subtests included in the study. This value also may be considered quite good (Tabachnick & Fidell, 1996).

Education and Neuropsychological Performance

To more thoroughly examine the influence of education on the observed differences in neuropsychological scores between the control group and clinical group four one-way Analyses of Covariance (ANCOVA) were performed on each of the four cognitive dependent variables: general neuropsychological functioning, verbal memory, attention, and processing speed. The independent variable consisted of group (control or

clinical) and level of education was used as the covariate in the analyses. After controlling for level of education, results showed there were significant differences between the clinical group and control group on the dependent variables of general neuropsychological functioning [$F(1,53) = 118.49$, $p < .01$, $\eta^2 = .69$], verbal memory [$F(1,53) = 82.03$, $p < .01$, $\eta^2 = .61$], attention [$F(1,53) = 36.43$, $p < .01$, $\eta^2 = .41$], and processing speed [$F(1,53) = 61.63$, $p < .01$, $\eta^2 = .54$]. These results indicate that the significant differences in general neuropsychological functioning, verbal memory, attention, and processing speed between the control group and the clinical group were due to group differences (i.e., features associated with BPD) and not due to differences in level of education between the two groups.

Depression and Neuropsychological Performance

The hypothesis that participants with BPD would demonstrate significantly higher levels of depression relative to control participants was analyzed using an independent samples *t*-test to compare the total scores on the BDI-II from the two groups of participants. Results revealed a significant difference in BDI-II scores between the control group ($M = 6.00$, $SD = 4.40$), and the clinical group [$M = 28.08$, $SD = 10.11$; $t(54) = -10.84$, $p < .01$, $\eta^2 = .69$]. That is, the clinical group's mean score on the measure of depression was considered "moderate" and was significantly higher than the control group's mean score on the measure of depression that was considered "mild" (Beck et al., 1996).

The hypothesis that individuals with BPD would have depression scores that correlated significantly with scores on the general index of neuropsychological performance was investigated using Pearson product-moment correlation coefficient.

Preliminary analyses were performed to ensure no violation of the assumptions of normality, linearity and homoscedasticity existed. There was a strong, significant negative correlation between the two variables [$r = -.70$, n = 56, $p < .01$], with higher levels of depression associated with lower scores on the general measure of neuropsychological functioning.

To address concerns regarding the possible contribution of depression in the observed differences in cognitive functioning between the two groups, an ANCOVA was performed using the independent variable, group, and depression was specified as the covariate. Interestingly, after controlling for depression, results showed a significant difference between the clinical group and control group on the dependent variables of general neuropsychological functioning [$F(1,53) = 35.76$, $p < .01$, $\eta^2 = .21$], verbal memory [$F(1,53) = 27.02$, $p < .01$, $\eta^2 = .20$], attention [$F(1,53) = 17.09$, $p < .01$, $\eta^2 = .19$], and processing speed [$F(1,53) = 13.27$, $p < .01$, $\eta^2 = .11$]. These results indicate that the significant differences in the assessed areas of cognitive functioning between the control group and the clinical group were due to group differences (i.e., features associated with BPD) and not due to differences in level of depression between the two groups.

Participant Effort

The possible relationship between test effort and BPD was explored by using a cut off score of 45 on trial 2 for the TOMM to identify the number of participants that provided insufficient effort on the tests. All control and clinical participants scored above a 45 on the TOMM trial 1 and 2, supporting the idea that all subjects provided good effort on the neuropsychological tests.

Neurological Disorder and Neuropsychological Performance

A within group analysis of the clinical sample was conducted using an independent samples *t*-test to explore the extent to which neuropsychological test performance was affected by a co-occurring BPD diagnosis and a self-reported neurological disorder diagnosis. General neuropsychological functioning scores of BPD participants who endorsed having a diagnosed neurological disease (n = 5) were compared with general neuropsychological functioning scores of BPD participants who did not endorse having a diagnosed neurological disease (n = 21). Results revealed a significant difference in general neuropsychological functioning between BPD participants who reported having a diagnosed neurological disease (M = 34.01, SD = 3.75) and BPD participants who did not report having a diagnosed neurological disease [M = 42.85, SD = 5.53; $t(24) = -3.37$, $p < .01$, $\eta^2 = .32$]. The results may be interpreted as meaning those clinical participants who endorsed having a neurological disease performed worse on the measure of general neuropsychological functioning compared with clinical participants who did not endorse having a diagnosed neurological disease. However, while the assumption of equal variances was met for this *t*-test analysis, interpretation of this result must be made with caution given the unequal and small sample size of clinical participants who had a diagnosed neurological disorder.

Additionally, a between group analysis was conducted to evaluate the influence of a neurological disorder diagnosis on neuropsychological test performance between the control group and the clinical group. An ANCOVA was performed using the independent variable, group, and neurological disorder diagnosis was used as the covariate. After controlling for a diagnosis of a neurological disorder, results revealed a

significant difference between the control group and clinical group on the dependent variables of general neuropsychological functioning [$F(1,53) = 122.17$, $p < .01$, $\eta^2 = .60$], verbal memory [$F(1,53) = 77.23$, $p < .01$, $\eta^2 = .56$], attention [$F(1,53) = 33.00$, $p < .01$, $\eta^2 = .36$], and processing speed [$F(1,53) = 64.62$, $p < .01$; $\eta^2 = .43$]. Therefore, the observed differences in neuropsychological functioning between the control and the clinical group existed after controlling for diagnosed neurological disorder. These results suggest that the differences in test performance may be due to group differences in terms of features associated with BPD and not due to differences in diagnosed neurological disorders between the two groups. Again, caution is necessary in interpreting these results because of the unequal and small sample size of clinical participants who had a diagnosed neurological disorder that was used as the covariate in the analysis.

Discussion

General Discussion

The results of the study demonstrate that, relative to healthy controls, individuals with BPD experience cognitive deficits. Specifically, participants with BPD obtained statistically significantly lower scores compared with control group participants on measures of attention, verbal memory, processing speed and a measure of general neuropsychological functioning that was an amalgamate score of the three previously mentioned domains. These significant differences between the control group and clinical group existed even after controlling for possible confounds such as level of education, depression, and presence of a diagnosed neurological disorder.

Why might individuals with BPD score significantly lower on neuropsychological measures relative to individuals without the diagnosis? One possible explanation is that

individuals with BPD have a neurobiology that is different from other persons, caused by prenatal teratogens (Abel, 1981, 1982) or postnatal experiences in the form of abuse and trauma (Goldman, D'Angelo, DeMaso, & Mezzacappa, 1992; Guzder et al., 1999; Guzder et al., 1996; Yen et al., 2002) that have deleterious effects on neurobiological development and functioning. Neuroimaging research findings of smaller amygdala and hippocampi in persons with BPD (Driessen et al., 2004; Gurvits et al., 1996) may provide a neurobiological explanation for the deficits in cognitive functioning found in the current study.

Because the hippocampus is a brain structure associated with memory functioning, it seems plausible to hypothesize that small hippocampi observed in persons with BPD suggests damage to, or abnormal development of, the hippocampus which could impair its functioning and result in verbal memory deficits. Similarly, reduced amygdala volume, a brain structure associated with emotional response, suggests damage or abnormal development that might interfere with its healthy functioning and result in deficits in verbal memory. For example, malformations in the amygdala may contribute to difficulty regulating stress emotions associated with completing 2 hours of neuropsychological testing, and consequently, may interfere with performance. Additional research that combines neuroimaging techniques with neuropsychological testing is needed to explicate the complex relationship between neurobiological structures and cognitive functioning in persons with BPD and to help corroborate these hypotheses.

Another possible explanation for the observed deficits in cognitive functioning in persons with BPD includes the presence of co-occurring psychological disorders. The most commonly reported co-occurring disorders included major depressive disorder,

bipolar I or II disorder, posttraumatic stress disorder (PTSD), generalized anxiety disorder (GAD) and attention deficit/hyperactivity disorder (ADHD). While the study's findings existed even after controlling for level of depression, other disorders not controlled for in the study may have contributed to the poor performance by the clinical sample on the tests of cognitive functioning. Given the psychological sequelae associated with disorders like PTSD, GAD and ADHD that includes difficulty concentrating (APA, 2000), it seems reasonable to assume that the symptoms of these co-occurring disorders influenced the test performance of participants who suffered from them. Additional neuropsychological research with individuals with BPD that controls for co-occurring disorders like PTSD, GAD and ADHD is needed.

The study found that participants with BPD had significantly higher levels of depression compared with the control group. The strong, negative correlation that existed between depression and general neuropsychological functioning meant that as participants' level of depression increased, their scores on the general neuropsychological measure decreased. It was somewhat surprising to find that the control group scored within the mild depression range on the BDI-II. A possible explanation for this finding includes the frequency and degree to which control participants endorsed those BDI-II items that assessed for disturbances in sleep patterns, tiredness or fatigue, and loss of energy. While these are symptoms associated with depression, they may also be the result of a college student's busy schedule and not necessarily indicative of depression.

A particularly remarkable finding was the significant differences in neuropsychological performance between the two groups after controlling for depression. This was a surprising finding given the empirical evidence that demonstrates individuals

with depression suffer from cognitive impairments (Gfeller, Chibnall & Duckro, 1994, Kurtz & Morey, 1999, Renneberg et al., 2005, Sprock et al., 2000). Nonetheless, the significant findings with large effect sizes in the current study indicate that the notable differences in test performance between the groups can be explained by the presence of the BPD diagnosis, and not due to the differences in levels of depression between the two groups. Such a determination is important because it helps elucidates the complex relationship between suppressed neuropsychological test performance and factors such as depression and BPD.

It may be worthwhile to consider what features associated with BPD might have affected test scores. Perhaps the hallmark features of BPD, such as affective instability, impulsivity or dissociation contributed to the cognitive deficits, more so than the effect of depressive symptoms. It seems likely that these BPD symptoms would interfere with one's ability to perform well on tests of memory, attention and speed of processing. Clinical participants may have experienced frustration during testing and had difficulty regulating their emotions, which may have interfered with their ability to focus, and consequently their performance suffered. Maybe a tendency to respond in an impulsive manner led to more incorrect responses and low scores. Perhaps clinical participants that felt stressed during the assessment dissociated as a protective coping mechanism causing them to be less present and resulting in poor scores.

The study explored how the presence of a neurological disease presenting with a BPD diagnosis affected performance on neuropsychological tests. The study found that clinical participants who self-reported having a diagnosed neurological disease performed worse on the measure of general neuropsychological functioning compared with clinical

participants who had no history of a diagnosed neurological disease. These findings indicate that neurological disease contributes to poor test performance in persons who have a BPD diagnosis.

The study also found that the presence of a neurological disorder diagnosis did not significantly contribute to the observed differences in test performance between the control group and clinical group. Therefore, in the current study, differences in neuropsychological test performance between the clinical group and the control group seemed to be attributable to presence of BPD and not the presence of a neurological disorder. However, interpretation of this finding is tempered by the limitation of a small sample size of participants with a diagnosed neurological disease. It may be that given a larger sample size of participants with diagnosed neurological disorders, results would show that neurological disorders contribute significantly to poor neuropsychological test performance. The degree to which neurological disorders contribute to deficits in neuropsychological test performance in persons with BPD remains an empirical question that may be answered by future research's replication of the current study using larger and equal sample sizes of BPD participants with and without a diagnosed neurological disease.

An exploratory area in the current study included evaluating the role of effort in neuropsychological testing with persons with BPD. All participants in the study provided good effort on the neuropsychological tests as measured by the TOMM. Clinical participants provided adequate effort despite the fact that most suffered from a moderate degree of depression, had symptoms associated with co-occurring disorders that may have been distracting and often performed poorly on the tests. These findings suggest

that the presence of a BPD diagnosis does not indicate a tendency to provide poor effort in testing situations, and that the data collected in the study were valid.

Implications of the Study Findings

Overall, the current findings support a growing body of literature that indicates deficits in cognitive functioning exist in persons with BPD (Bazanis et al., 2002; Beblo et al., 2005; Carpenter et al., 1993; Dinn, et al., 2004; Judd & Ruff, 1993; Monarch et al., 2004; O'Leary et al., 1991; Ruocco, 2005; Stevens et al., 2004; Swirsky-Sacchetti et al., 1993). The study results are important for several reasons. It seems likely that deficits in cognitive functioning would contribute to difficulties in different areas of life for persons with BPD. Generally speaking, deficits in attention and verbal memory could adversely impact interpersonal relationships and compromise one's ability to regulate emotions, both of which are features of BPD. A decreased ability to process information quickly may interfere with work performance or ability to maintain employment. Additionally, cognitive deficits in any of these areas may disrupt and hinder a client's involvement and progress in therapy (Allen, 2002). More specific implications of neuropsychological deficits can be discussed by evaluating each of the cognitive domains.

The finding that individuals with BPD experience attention difficulties is noteworthy because attention is a cognitive ability that affects other areas of cognitive functioning (Monarch et al., 2004). Clinically, this is vital information for therapists to be aware of in terms of interpreting client behavior. As noted by Monarch et al. (2004), inattention, forgetfulness or distracted behavior in session may be due to neuropsychological impairments rather than perceiving such behavior as a client's resistance to treatment. Additionally, because of the ubiquitous nature that attention

deficits have on other areas of cognitive functioning, it is important for clinicians to be aware of evidence that shows individuals with BPD have attention difficulties because such evidence may help explain other cognitive limitations displayed by a client.

The finding that persons with BPD experience deficits in attention has several other clinical implications. Difficulty sustaining and focusing attention in therapy sessions would likely compromise a client's ability to fully benefit from the therapeutic process. Allen (2002) notes that flexibility on the part of the clinician is a must when working with clients who suffer from neuropsychological impairments. This includes both diffuse deficits like decreased attention and impaired processing speed, and more focal deficits such as impaired verbal memory.

Developing an awareness that attention difficulties may exist for clients with BPD may prompt a therapist to adopt certain therapeutic techniques to accommodate for such deficits. Pragmatically, this may look like the therapist asking for feedback from the client during sessions to assess if he or she has lost focus. To accomplish this, Allen (2002) offers several suggestions including having the therapist and client agree on a prompt or a cue phrase (e.g., "Let's stay focused") to use in session to help provide structure and focus and minimize tangential discussions. Also, a therapist could introduce more structure by presenting a limited number of goals to address each session and provide the client a written list of the goals to help him or her remain attentive. By presenting information at a slower rate and avoiding lengthy sentences the therapist can assist clients with cognitive deficits in successfully tracking a topic discussion and decreases their confusion or distraction. A therapist could pause for a few minutes in session to allow the client to mentally review the previous discussion, or the therapist

could check for the client's level of understanding throughout the session to help him or her manage the therapeutic information and attend to what is being discussed.

Another possible implication of attention deficits includes its impact on clients' interpersonal relationships. Perhaps because of attention deficits, an individual with BPD is unable to be attentive and focused with a friend when he or she needs the person to be present. This may cause frustration for both persons and may serve as an added stressor in the relationship leading to its dissolution. Attentional deficits may make it difficult to maintain employment, the consequences of which might include financial hardship, homelessness, strained relationships, low self-esteem, and frustration. All of these consequences could become added stressors that exacerbate BPD symptoms.

A clinical implication of poor verbal memory in individuals with BPD includes difficulty effectively communicating and engaging in psychotherapy, a process reliant on a client's ability to recall information communicated verbally. Successful psychological intervention is dependent upon a client's verbal memory ability to retain conclusions, insights, and the context of the dialogue between client and therapist in a session (Bennett, 1989). An inability to recall skills verbally communicated in therapy that teach, for example, how to regulate emotions may cause a client to become emotionally dysregulated more frequently or more severely. Given how common emotion dysregulation is in persons with BPD, it is seems important for clinicians to consider how emotional regulation is impacted by verbal memory deficits.

Therapists working with BPD clients may want to consider providing the client with a tape recording of therapy appointments to allow him or her to review the sessions' content between meetings. Another compensatory measure that therapists may employ

includes suggesting to clients that they take notes during therapy to help them remember salient points discussed in session. Lewis and Langer (1994) suggest strategies for working with clients who have verbal memory deficits that include the use of symbolism and working with the client to create visual images to assist in encoding verbal material. The authors suggest that this technique may resemble mnemonic techniques such as the therapist assigning each letter of an acronym to a therapy goal or homework assignment. Allen (2002) recommends the use of a memory notebook that allows clients to organize and recall therapy information by recording important points of sessions, changing therapy goals, scheduled appointments, and homework assignments.

Verbal memory deficits, similar to attention deficits, may contribute to relationship difficulties. For example, perhaps poor verbal memory may cause a person with BPD to forget scheduled meetings with friends, or important issues discussed with a friend about their relationship and cause frustration and stress for both persons involved. Verbal memory deficits may also make it difficult for persons with BPD to find or keep a job. Certainly, unemployment creates stressors that may exacerbate BPD symptoms and add to the psychological stress experienced by an individual.

The finding that individuals with BPD demonstrate deficits with processing speed has clinical relevance. In an age of managed care when delivery of therapeutic services is often constrained by time limitations, the speed with which a client can process information is likely to influence how beneficial therapy will be for the client. One example of this is managed care providers often require clinicians to utilize manualized treatments with a time-limited format. Manualized treatments often outline material to be presented in each psychotherapy session and require clients to be able to process

information in a timely fashion. If a therapist is required to present information at a slower pace in therapy because of a client's cognitive limitations in areas like processing speed, this would impede progress and make it difficult to adhere to the therapy schedule associated with a manualized treatment. Clinically, individuals with BPD that display a deficit in processing speed may experience this as a barrier to treatment that compromises their ability to benefit from therapy that employs manualized interventions.

Similar to attention and verbal memory deficits acting as obstacles to employment, poor processing speed may also make it difficult for a person with BPD to find work or remain employed. Certainly unemployment creates a host of problems and consequences including those previously discussed. Both impulsivity and intense anger are symptoms of BPD that may be exacerbated by poor processing speed. Difficulty processing information quickly may frustrate individuals with BPD and result in less patience when problem solving and create a more impulsive pattern of responding. In addition to poor processing speed possibly contributing to impulsivity, the frustration associated with the deficit may lead to more bouts of anger or more intense feelings of anger.

The relationship between medications and cognitive functioning is complex. Research has yet to clearly explain the extent to which the myriad of psychotropic medications contribute to deficits in cognitive functioning. However, findings such as those from the current study that indicate cognitive difficulties in persons with BPD provide important information for clinicians and physicians to consider when prescribing medications that have known cognitive side effects. Prescribing a medication with side effects that are less detrimental to cognitive functioning may be an important

consideration for clinicians and physicians when working with this population. Additionally, research by Dawson (2004) shows that involving BPD clients more in the process of selecting medications to mange their symptoms results in them making better choices for themselves, not abusing or misusing pills they have chosen themselves, and choosing a far more sensible regimen of medication than has been prescribed for them in the past.

Another important clinical implication of the study's general findings regarding limitations in cognitive functioning in individuals with BPD concerns the issue of suicide. Persons that suffer from BPD have a high rate of self-harm and suicide behaviors (Linehan, 1993), and suicide in persons with BPD has been correlated with cognitive functioning and not depression (Burgess, 1991). Therefore, evidence that demonstrates persons with BPD have difficulty with attention, verbal memory and processing speed provides clinicians with vital information that can be utilized in diagnosis, treatment planning, and suicide prevention. The study results underscore the importance that the practice of using screening measures to detect the presence of other pathologies and client limitations should include screening for neuropsychological impairments in clients with BPD.

A clinical intervention with empirical evidence supporting its efficacy in the treatment of BPD is Dialectical Behavior Therapy (DBT; Koons et al., 2001; Linehan, Armstong, Suarez, Allmon, & Heard, 1991; Linehan, Heard, & Armstrong, 1993; Verheul et al., 2003). DBT is a manual-based treatment that includes a psychoeducational skills training component, incorporates several different modes of treatment, and involves repetition of skills-based knowledge (Linehan, 1993). The

treatment seeks to apply a variety of cognitive and behavior therapy strategies to the problems faced by individuals with BPD, particularly suicidal behaviors.

By nature of its design, DBT may serve nicely as an intervention that accommodates for the cognitive limitations of persons with BPD. For example, DBT's didactic approach involves clear instruction and repetition of information and skills practice to enhance skills acquisition. DBT presents material to clients using audio-video skills training tapes, printed handouts on skills practice, and makes use of role-playing as a teaching tool. This model of therapy that incorporates clear instruction, repetition and multi-dimensional teaching approaches seems ideal for persons with cognitive difficulties in areas such as attention, verbal memory and processing speed. In light of the current findings, it is interesting to speculate that perhaps one reason DBT is effective as a treatment is because it includes techniques that are sensitive to issues related to the cognitive deficits experienced by individuals with BPD.

Strengths and Limitations

The current study contains a number of strengths including its methodology that used several measures to assess for cognitive functioning in three areas. A benefit of using multiple measures to evaluate attention, verbal memory, and processing speed was that it provided a more comprehensive battery for assessing each area of functioning than would have been afforded by using a single measure for each domain. Also, by utilizing several neuropsychological tests the researcher was able to assess for multiple aspects of the same construct (e.g., sustained and controlled attention) and test functioning in each domain using different mediums of assessment (e.g., visual test and auditory test presentations).

The study's sample size of 56 participants may also be considered a strength. Relative to other studies evaluating the neuropsychological functioning in persons with BPD, a clinical sample size of 26 can be considered large. The use of normative data tables to convert raw scores into standardized *t*-scores allowed the researcher to control for the confounding effects of variables like age, gender, and level of education for most measures used in the study. Therefore, the use of standardized scores to compare mean performance on the neuropsychological measures represented another strength of the study. While age and was accounted for by the use of normative tables for all measures, it is important to note that not all tables for the utilized neuropsychological measures provided norms that accounted for the variables of gender and level of education. This issue was addressed by conducting a Chi Square test and a Mann-Whitney U test to demonstrate that no significant differences existed in gender or level of education between the two groups. Additionally, the results from an ANCOVA that controlled for level of education showed that significant differences between the two groups existed due to group differences and not due to an education effect. The medium to large effect sizes, as interpreted using guidelines established by Cohen (1988), that were observed in the study indicate the differences in cognitive functioning that existed between the two groups were notable differences and strengthen the study's findings.

Another strength of the study included the careful screening of participants to ensure that all had met criteria for BPD. Because all participants completed the SCID-II (First et al., 1997) the researcher was confident that all participants in the clinical sample were accurately diagnosed. The use of the SCID-II was consistent with diagnostic screenings techniques used by other research studies in this area. Careful screening of

participants for the BPD diagnosis strengthened the internal validity of the current study and allows the findings to be applied to other individuals diagnosed with BPD. While the current study did not include participants with only BPD diagnoses, it is rare to find such cases, as co-occurring substance use, depression, and other Axis I diagnoses are common with this population.

Several variables often contribute notably to performance on neuropsychological tests. For example, years of education has been identified as a variable that affects test performance on measures used in the current study such as the PASAT (Stuss, Stethem, & Poirier, 1987), Logical Memory (Richardson & Marottoli, 1996; Ylikoski et al., 1998), and the Corsi Block-Tapping Test which closely resembles the Spatial Span subtest from the WAIS-III (Orsini et al., 1986). The study's analyses found significant differences in cognitive functioning continued to exist after controlling for several possible confounds, including education, depression and, with less certainty, a neurological disorder diagnosis. Confounding variables like level of education, depression and neurological disorders often affect neuropsychological test performance and limit conclusions regarding the affect of the primary variable of interest in a study (i.e., BPD) on cognitive functioning. While the study was unable to control for all confounds, the results from the analyses that controlled for those variables discussed above provided support for the hypothesis that cognitive deficits in persons with BPD were attributable to the disorder and not to certain identified extraneous variables.

One limitation of the current study included not controlling for possibly confounding variables like medications, co-occurring Axis I diagnoses, and hospitalization. It is important to consider the contribution of secondary consequences of

BPD such as medications, co-occurring disorders like substance abuse, or hospitalization when interpreting the findings. For example, all experimental participants were taking some type of psychotropic medication to help manage their mental illnesses. Several authors found individuals who are taking antiepileptic medications may score low on the Digit Symbol and Symbol Search subtests of the WAIS-III (Aldenkamp et al., 2000; Pagliaro & Pagliaro, 1999). Therefore, interpretation of the findings must take into account the possible confounding affect medications may have exerted on participants' performance.

Additionally, 11 (42%) of the clinical participants self-reported having a substance abuse or dependence diagnosis. Extensive literature demonstrates a correlation between chronic alcohol abuse and cognitive deficits in areas including memory, visuospatial and executive functioning (Adams & Victor, 1993; Fals-Stewart, Schafer, Lucente, Rustine, & Brown, 1994; Grant et al., 1978; Miller, 1991; Parsons, 1987; Reitan & Wolfson, 1993). Also, sustained use of cocaine has been associated with memory deficits (Horner, 1997; Mittenberg & Motta, 1993; O'Malley & Gawin, 1990; Rosselli & Ardila, 1996; Van Gorp et al., 1999). As a result, the current study findings must be interpreted within the context of co-occurring substance abuse found in over two-fifths of the clinical sample.

In addition to substance abuse or substance dependence disorders contributing to cognitive difficulties, other disorders can have a similar affect on an individual's neuropsychological performance including anxiety (Gass, 2002; Gass, Ansley, & Boyette, 1994; Kizilbash et al., 2002), PTSD, and ADHD. Given the prevalence of these

disorders in the clinical sample, it is important to recognize the possible adverse affect this disorder exerted on participant performance.

Beyond factors such as medications and co-occurring disorders there are other explanations for why an individual may perform poorly on a neuropsychological assessment. For example, 20 (77%) of the clinical participants were recruited and tested in a hospital setting, suggesting it may be important to consider the effect of hospitalization on neuropsychological functioning. Participants' length of stay in the hospital may have negatively impacted their neuropsychological functioning. A review of the literature reveals little research investigating the affect of hospitalization on neuropsychological performance. Additional research in this area is needed.

Another limitation includes the method used to assess for neurological disease and, as mentioned previously, the small sample of participants with a neurological disorder diagnosis that restricted interpretation of the results. Because diagnosed neurological disorders were determined based on participants' self-report, it is difficult to assess how accurate the prevalence rates for neurological diseases were in the study's sample. A more precise method for determining presence of a neurological disorder diagnosis might include referencing participants' medical records to corroborate participants' self-reports.

The clinical sample was a primarily female in-patient sample (85%), and thus constitutes another limitation. While prevalence rates for the disorder indicate roughly 75% of diagnosed persons with BPD are female (APA, 2000), the percentage of females in the current study was over-representative of this statistic. Also, the study attempted to recruit as homogeneous a sample of participants as possible. However, because of the

obvious limitations associated with data collection in a rural area, the clinical sample consisted of both in-patient and outpatient participants. Therefore, future research is necessary to determine to what extent the current study's findings have external validity and can be generalized to males or to outpatient populations with BPD.

Future Directions

Future research in this area may include assessing for deficits in other areas of cognitive functioning not addressed in the current study such as executive functioning, visuospatial, planning and motor abilities. Deficits in these areas would generate other implications and concerns that may necessitate additional clinical consideration. For example, cognitive limitations in executive functioning or planning are likely to contribute to a client's difficulty anticipating potential triggers that lead to decompensation unless effective therapeutic coping skills are used. Research investigating for the presence of other cognitive deficits in persons with BPD would provide clinicians with important information that could be used to better adapt therapeutic techniques to accommodate clients' needs. Additional research into other areas of cognitive functioning of persons with BPD, as well as replication of the current findings, will assist in the construction of a valid neuropsychological profile of BPD.

The nature of BPD makes it very difficult to gather a sample of participants who do not have co-occurring diagnoses, are not taking medications, are not hospitalized, or do not have a diagnosed neurological condition. Nonetheless, the possible confounding affects of these factors on cognitive functioning are important considerations to try and address through additional empirical research. Future research may seek to control for these possible confounds through recruitment of BPD participants with limited co-

occurring diagnoses, without a neurological condition and who are not taking medications. Lastly, future studies with individuals with BPD may want to include conducting comparison analyses of neuropsychological functioning of an inpatient sample, an outpatient sample and a healthy control sample to investigate the possible role of hospitalization on cognitive functioning.

References

Abel, E. L. (1981). Behavioral teratology of alcohol. *Psychological Bulletin, 90,* 564-581.

Abel, E. L. (1982). Consumption of alcohol during pregnancy: A review of effects on growth and development of offspring. *Human Biology, 54,* 421-453.

Adams, M. W., & Victor, M. (1993). *Principles of Neurology* (5th ed.). New York: McGraw-Hill.

Adler, G., & Buie, D. (1979). Aloneness and borderline psychopathology: The possible Relevance of child development issues. *International Journal of Psychoanalysis, 60*, 83-96.

Akiskal, H. S. (1981). Subaffective disorders: Dythymic, cyclothymic and bipolar II disorders in the "borderline" realm. *Psychiatric Clinics of North America, 4,* 25-46.

Allen, J. B. (2002). *Treating patients with neuropsychological disorders: A clinician's guide to assessment and referral.* Washington, DC: American Psychological Association.

Aleman, A., Schutter, D. J., Ramsey, N. F., van Honk, J., Kessels, R. P., Hoogduin, J. M., et al. (2002). Functional anatomy of top-down visuospatial processing in the human brain: Evidence from rTMS. *Brain Research: Cognitive Brain Research, 14,* 300-302.

Aldenkamp, A. P., Baker, G., Mulder, O. G., Chadwick, D., Cooper, P., Doelman, J., et al. (2000). A multicenter, randomized clinical study to evaluate the effect on cognitive function of topiramate compared with valproate as add-on therapy to carbamazepine in patients with partial-onset seizures. *Epilepsia, 41,* 1167-1178.

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

Andrulonis, P. A. (1990). Disruptive behavior disorder in boys and the borderline personality disorder in men. Paper presented at Einstein Medical College, Philadelphia.

Andrulonis, P. A**.,** Glueck, B. C**.,** Stroebel, C. F., & Vogel, N. G. (1982). Borderline personality subcategories. *Journal of Nervous and Mental Disease, 170,* 670-679.

Andrulonis, P. A., Glueck B. C., Stroebel, C. F., Vogel, N. G., Shapiro, A. L., & Aldridge, D. M. (1981). Organic brain dysfunction and the borderline syndrome. *Psychiatric Clinics of North America, 4,* 47-66.

Arntz, A., Meeren, M., & Wessel, I. (2002). No evidence for overgeneral memories in borderline personality disorder. *Behaviour Research and Therapy, 40*, 1063-1068.

Ashendorf, L., Costantinou, M., & McCaffrey, R. J. (2004). The effect of depression and anxiety on the TOMM in community-dwelling older adults. *Archives of Clinical Neuropsychology, 19*, 125-130.

Barnett, D., Ganiban, J., & Cicchetti, D. (1999). Maltreatment, negative expressivity, and the development of Type D attachment from 12 to 24 months of age. *Monographs of the Society for Research in Child Development, 64,* 97-118.

Bazanis, E., Rogers, R. D., Dowson, J. H., Taylor, P., Meux, C., Staley, C., et al. (2002). Neurocognitive deficits in decision-making and planning of patients with DSM-III-R borderline personality disorder. *Psychological Medicine, 32,* 1395-1405.

Beblo, T., Saavedra, A. S., Mensebach, C., & Driessen, M. (2005). Cognitive and psychophysiological functioning in borderline personality and posttraumatic stress disorder. In T. A. Corales (Ed.), *Focus on Posttraumatic Stress Disorder Research.* (pp. 51-70). Hauppauge, NY: Nova Science Publishers.

Beck, A. T., & Freeman, A. (1990) Cognitive therapy for personality disorders. New York: Guilford Press.

Beck, A. T., Steer, R. A., & Brown, G. K. (1996). *BDI-II: Beck Depression Inventory - 2nd Edition Manual*. San Antonio, Texas: The Psychological Corporation.

Bennett, T. L. (1989). Individual psychotherapy and minor head injury. *Cognitive Rehabilitation, 7(5),* 20-25.

Berg, M. (1983). Borderline psychopathology as displayed on psychological tests. *Journal of Personality Assessment. 47,* 120-133.

Bernard, L. C., McGrath, M. J., & Houston, W. (1996). The differential effects of simulating malingering, closed head injury, and other CNS pathology on the Wisconsin Card Sorting Test: Support for the "pattern of performance" hypothesis. *Archives of Clinical Neuropsychology, 11*, 231-245.

Berntson, G. G., Cacioppo, J. T., & Quigley, K. S. (1991). Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. *Psychological Review, 98,* 459-487.

Bersin, E. V. (1994). Development formulation and psychotherapy of borderline adolescents. *American Journal of Psychotherapy, 48*, 5-29.

Bezirganian, S., Cohen, P., & Brook, J. S. (1993) The impact of mother-child interaction on the development of Borderline Personality Disorder. *American Journal of Psychiatry, 150*, 1836-1842.

Binder, L. M. (1993). Assessment of malingering after mild head trauma with the Portland Digit Recognition Test. *Journal of Clinical and Experimental Neuropsychology, 15,* 170-183.

Binder, L. M., & Willis, S. C. (1991). Assessment of motivation after financially compensable minor head trauma. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 3,* 175-181.

Bornstein, R. A. (1983). Reliability and item analysis of the Seashore Rhythm Test *Perceptual and Motor Skills, 57,* 57-574.

Bremmer, J. D. (1999). Does stress damage the brain? *Biological Psychiatry, 45,* 797-805.

Bremmer, J. D., Randall, P., Scott, T. M., Bronen, R.A., Seibyl, J. P., Southwick, et al. (1995). MRI-based measurement of hippocampual volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry, 152,* 973-981.

Bremner, J. D., Vermetten, E., Vythilingam, M., Afzal, N., Schmahl, C., Elzinga, B. et al. (2004). Neural correlates of the classic color and emotional Stroop in women with abuse-related posttraumatic stress disorder. *Biological Psychiatry, 55*, 612-620.

Burgess, J. W. (1991). Relationship of depression and cognitive impairment to self-injury in borderline personality disorder, major depression, and schizophrenia. *Psychiatry Research, 38,* 77-87.

Bush, S. S., Ruff, R. M., Troster, A. I., Barth, J. T., Koffler, S. P., Pliskin, N. H. et al. (2005). Symptom validity assessment:Practice issues and medical necessity. NAN Policy and Planning Committee. *Archives of Clinical Neuropsychology, 20*, 419-426.

Carlson, E. A. (1998). A prospective longitudinal study of attachment disorganized/disorientation. *Child Development, 69,* 1107-1128.

Carpenter, C. J., Gold, J. M., & Fenton, W. (1993). Neuropsychological testing results in borderline inpatients. Poster presentation at the 146th Annual Meeting of the American Psychiatric Association. San Francisco, CA.

Carr, A. C., Goldstein, E. G., Hunt, H. F., & Kernberg, O. (1979). Psychological tests and borderline patients. *Journal of Personality Assessment, 43*, 582-590.

Casillas, A., & Clark, L. A. (2002). Dependency, impulsivity, and self-harm: Traits theorized to underlie the association between cluster B personality disorders and substance abuse disorders. *Journal of Personality Disorders, 16*, 424-436.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences.* Hillsdale, NJ: Erlbaum.

Cohen, J. (1992). A power primer. *Psychological Bulletin, 112*(1), 155-159.

Comtois, K. A., Cowley, D. S., Dunner, D. L., & Roy-Byrne, P. (1999). Relationship between borderline personality disorder and Axis I diagnosis in severity of depression and anxiety. *Journal of Clinical Psychiatry, 60*, 752-758.

Comtois, K. A., Russo, J., Snowden, M., Srebnik, D., Ries, R., & Roy-Byrne, P. (2003). Factors associated with high use of public mental health services by persons with borderline personality disorder. *Psychiatric Services, 54*, 1149-1154.

Cornblatt, B. A., Risch, N. J., Faris, G., Friedman, D., & Frienmeyer-Kimling, L. (1988). The Continuous Performance Test, Identical Pairs Version (CPT-IP): I. New findings about sustained attention in normal families. *Psychiatry Research, 26,* 223-238.

Corwin, J., & Bylsma, F. (1993). Translations of excerpts from Andre Rey's psychological examination of traumatic encephalopathy and P.A. Osterrieth's The Complex Figure Copy Test. *Clinical Neuropsychology, 7*, 3-15.

Dawes, R. M., Faust, D., & Meehl, P. E. (1989). Clinical versus actuarial judgment *Science, 243,* 1668-1675.

Dawson, D. (2004). Relationship management. *Psychiatric Annals, 34,* 482-490.

DenBoer, J. W., & Hall, S. (February, 2004). TOMM vs. WCST: TOMM Classifies Coached Malingerers and Controls More Accurately. Presentation to the International Neuropsychological Society Annual Convention: Baltimore, MY.

Delis, D. C., Kramer, J. H., Fridlund, A. J., & Kaplan, E. (1990). A cognitive science approach to neuropsychological assessment. In P. McReynolds et al. (Eds.), *Advances in psychological assessment* (Vol. 7). New York: Plenum Press.

Delis, D. C., Kramer, J. H., Kaplan, E., & Ober, B. A. (2000). *California Verbal Learning Test-Second Edition (CVLT-II).* San Antonio, TX: Psychological Corporation.

DiCarlo, M. A., Gfeller, J. D., & Oliveri, M. V. (2000). Effects of coaching on detecting feigned cognitive impairment with the Category Test. *Archives of Clinical Neuropsychology, 15*, 399-413.

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 NeuroPsychopharmacology & Biological Psychiatry, 28*, 329-341.

Donegan, N. H., Sanislow, C. A., Blumberg, H. P., Fulbright, R. K., Lacadie, C., Skudlarski, P., et al. (2003). Amygdala hyperreactivity in borderline personality disorder : Implications for emotional dysregulation. *Biological Psychiatry, 54*, 1284-1293.

Driessen, M., Beblo, T., Mertens, M., Peifke, M., Rulkoetter, N., Silva-Saavedra, A., et al. (2004). Posttraumatic stress disorder and fMRI activation patterns of traumatic memory in patients with borderline personality disorder. *Biological Psychiatry, 55,* 603-611.

Etherton, J. L., Bianchini, K. J., Greve, K. W., Ciota, M. A. (2005). Test of Memory Malingering performance is unaffected by laboratory-induced pain: Implications for clinical use. *Archives of Clinical Neuropsychology, 20*, 375-384.

Fals-Stewart, W., Schafer, J., Lucente, S., Rustine, T., & Brown, L. (1994). Neurobehavioral consequences of prolonged alcohol and substance abuse: A review of findings and treatment implications. *Clinical Psychology Review, 14,* 755-778.

Fan, J., McCandliss, B. D., Sommer, T., Raz, M., & Posner, M.I. (2002). Testing the efficiency and independence of attentional networks. *Journal of Cognitive Neuroscience, 14,* 340-347.

Fincham, J. M., Carter, C. S., van Veen, V., Stenger, V. A., & Anderson, J. R., (2002). Neural mechanisms of planning: A computational analysis using event-related fMRI. Proceedings of the National Academy of Sciences of the United States of America, 99, 3346-3351.

First, M. B., Gibbon, M., Spitzer, R. L., Williams, J. B. W., & Benjamin, L. (1997). *The Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II).* Washington, DC: American Psychiatric Press.

Gansler, D., Tombaugh, T. N., Moczynski, N. P., & Rees, L. M. (1995). *Test of memory malingering (TOMM): Initial validation in a traumatic braining injury cohort.* Poster presented at the Annual Meeting of the National Academy of Neuropsychology, San Francisco, CA.

Garber, J. & Hollon, S. D. (1991). What can specificity designs say about causality in psychopathology research? *Psychological Bulletin, 110*, 129-136.

Gass, C. S. (2002). Does test anxiety adversely affect neuropsychological test performance? *Archives of Clinical Neuropsychology, 17,* 860.

Gass, C. S., Ansley, J., & Boyette, S. (1994). Emotional correlates of fluency test and maze performance. *Journal of Clinical Psychology, 50,* 586-590.

Gergely, G. (1992). Developmental reconstructions: Infancy from the point of view of psychoanalysis and developmental psychology. *Psychoanalysis and Contemporary Thought, 15,* 3-55.

Gervais, R. O., Russell, A. S., Green, P., Allen, III, L. M., Ferrari, R., & Pieschl, S. D. (2001). Effort testing in fibromyalgia patients with disability incentives. *Journal of Rheumatolgoy, 28,* 1892-1899.

Gfeller, J. D., Chibnall, J. T., & Duckro, P. N. (1994). Postconcussion symptoms and cognitive functioning in posttraumatic headache patients. *Headache, 34,* 503-507.

Gibson, D. (1990). Borderline Personality Disorder issues of etiology and gender. *Occupational Therapy in Mental Health, 10*, 63-77.

Goldman, S. J., D'Angelo, E. J., DeMaso, D. R., & Mezzacappa, E. (1992). Physical and sexual abuse histories among children with borderline personality disorder. *American Journalof Psychiatry, 149,* 1723-1726.

Grant, I., Adams, K., Carlin, A., Rennick, P. M., Judd, L. L., & Schooff, K., (1978). The collaborative neuropsychological study of polydrug users. *Archives of General Psychiatry, 35,* 1063-1074.

Green, P., & Iverson, G. L. (2001). Effects of injury severity and cognitive exaggeration on olfactory deficits in head injury compensation claims. *Neruorehabilitation, 16,* 237-243.

Green, P., Rohling, M. L., Lees-Haely, P. R., & Allen, III, L. M. (2001). Effort has a greater effect on test scores than severe brain injury in compensation claimants, *Brain Injury, 15,* 1045-1060.

Greve, K. W., & Bianchini, K. J. (2002). Using the Wisconsin Card Sorting Test to detect malingering: An analysis of the specificity of two methods of in nonmalingering normal and patient samples. *Journal of Clinical and Experimental Neuropsychology, 24,* 48-54.

Greve, K. W., Bianchini, K. J., Mathias, C. W., Houston, R. J., & Crouch, J. A. (2002). Detecting malingered performance with the Wisconsin Card Sorting Test: A preliminary investigation in traumatic brain injury. *The Clinical Neuropsychologist, 16*, 179-191.

Grilo, C. M., Martino, S., Walker, M. L., Becker, D. F., Edell, W. S., & McGlashan, T. H. (1997). Controlled study of psychiatric comorbidity in psychiatrically hospitalized young adults with substance use disorders. *American Journal of Psychiatry, 157*, 1305-1307.

Gronstein, J. S. (1990). Invarients in primitive emotional disorders. In L.B. Boyer & P.L. Giovacchini (Eds.), *Master clinicians on treating the regressed patient.* (pp.139-163). Northvale, NJ: Jason Aronson.

Gronwall, D. M. A. (1977). Paced Auditory Serial-Addition Task: A measure of recovery from concussion. *Perceptual and Motor Skills, 44,* 367-373.

Gronwall, D. M. A., & Sampson, H. (1974). The psychological effects of concussion. Auckland: Oxford University Press.

Gunderson, J. G., & Zanarini, M. C. (1987). Current overview of the borderline diagnosis. *Journal of Clinical Psychiatry*, *48*(5), 1-11.

Gurvits, T. V., Shenton, M. E., Hokama, H., Ohtta, H., Lasko, N. B., Gilbertson, M.W., et al. (1996). Magnetic resonance imaging-based measurement of hippocampal volume in chronic, combat related posttraumatic stress disorder. *Biological Psychiatry, 40,* 1091-1099.

Guzder, J., Paris, J., Zelkowitz, P., & Feldman, R. (1999). Psychological risk factors for borderline pathology in school-age children. *Journal of American Academy of Child and Adolescent Psychiatry,38*, 206-212.

Guzder, J., Paris, J., Zelkowitz, P., & Marchessault, K. (1996). Risk factors for borderline pathology in children. *Journal of American Academy of Child and Adolescent Psychiatry, 35*, 26-33.

Haase, R. G., & McCaffrey, R. J. (2004). Too many variables! Some strategies for analyzing multiple dependent variables in complex designs. Special Topics Workshop. Presented at the 24th Annual Conference of the National Academy of Neuropsychology, Seattle.

Harris, C. L., Dinn, W.M., & Marcinkiewicz, J. A. (2002). Partial seizure-like symptoms in borderline personality disorder. *Epilepsy and Behavior, 3,* 433-438.

Heaton, R. K., Grant, I., & Matthews, C. G. (1991). *Comprehensive norms for an expanded Halstead-Reitan Battery: Demographic corrections, research findings, and clinical applications*. Odessa, FL: Psychological Assessment Resources.

Heaton, R. K., Smith, H. H., Jr., Lehman, R. A. W., & Vogt, A. T. (1978). Prospects for faking believable deficits on neuropsychological testing. *Journal of Consulting and Clinical Psychology, 46*(5), 892-900.

Heinly, M. T., Greve, K. W., Love, J. M., Brennan, A., & Bianchini, K. J. (2004). The use of the WAIS digit span markers in the detection of malingered neurocognitive dysfunction in traumatic brain injury. *Archives of Clinical Neuropsychology, 19,* 861.

Hiscock, C. K., Branham, J. D., & Hiscock, M. (1994). Detection of feigned cognitive impairment: The two-alternative forced-choice method compared with selected conventional tests. *Journal of Psychopathology and Behavioral Assessment, 16*, 95-110.

Horner, M. D. (1997). Cognitive functioning in alcoholic patients with and without cocaine dependence. *Archives of Clinical Neuropsychology, 12,* 667-676.

Jacobs, R., & Anderson, V. (2002). Planning and problem solving skills following focal frontal brain lesions in childhood: Analysis using the Tower of London. *Neuropsychology, Development, and Cognition. Section C, Child Neuropsychology, 8,* 93-106.

Johnson, S. C., Saykin, A. J., Flashman, L. A., McAllister, T. W., & Sparling, M. B. (2004). Brain activation of fMRI and verbal memory ability: Functional neuroanatomic correlates of CVLT performance. *Journal of the International Neuropsychological Society, 7,* 55-62.

Jones, B., Heard, H., Startup, M., Swales, M., Williams, J. M. G., & Jones, R. S. W. (1999). Autobiographical memory and dissociation in borderline personality disorder. *Psychological Medicine, 29,* 1397-1404.

Joyce, P. 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*, 25-43.

Judd, P. H., & Ruff, R. M. (1993). Neuropsychological Dysfunction in borderline personality disorder. *Journal of Personality Disorders, 7*, 275-284.

Kavoussi, R. J., Coccaro, E. F., Klar, H. M., Bernstein, D., & Siever, L. J. (1990). Structured interviews for Borderline Personality Disorder. *American Journal of Psychiatry,147*, 1522-1525.

Kelley, W. M., Miezin, F. M., McDermott, K. B., Buckner, R. L., Raichle, M. E., Cohen, N. J., et al. (1998). Hemispheric specialization in human dorsal frontal cortex and medial temporal lobe for verbal and nonverbal memory encoding. *Neuron, 20,* 927-936.

Kernberg, O. (1975). *Borderline conditions and pathological narcissism*. New York: Jason Aronson.

Kernberg, P. (1984). The psychological assessment of children with borderline personality organization. Presented to the American Psychoanalytic Association, NewYork.

Kizilbash, A. H., Vanderploeg, R. D., & Curtiss, G. (2002). The effects of depression and anxiety on memory performance. *Archives of Clinical Neuropsychology, 17,* 57-67.

Knight, R. (1953). Borderline states. *Bulletin of Menninger Clinic, 17*, 1-12.

Kohut, H. (1971). *The analysis of the self*. New York: Basic Books.

Koons, C. R., Robbins, C. J., Tweed, J. L., Lynch, T. R., Gonzales, A. M., Morse, J. Q. et al., (2001). Efficacy of Dialectical Behavior Therapy in women veteran with borderline personality disorder.*Behavior Therapy, 32,* 371-390.

Kremers, I., Spinhoven, P., & vander Does, A. J. W. (2004). Autobiographical memory in depressed and non-depressed patients with borderline personality disorder. *British Journal of Clinical Psychology, 43*, 17-29.

Kreitman, N. (1977). *Parasuicide*. Chester, England: Wiley.

Kroll, J. (1988). *The challenge of the borderline patient*. New York: W.W. Norton.

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.

Kurtz, J. E., & Morey, L. C. (1999). Verbal memory dysfunction in depressed outpatients with and without borderline personality disorder. *Journal of Psychopathology and Behavioral Assessment, 21,* 141-156.

LeDoux, J. E. (1992). Emotion as memory: Anatomical systems underlying indelible neural traces. In S. A. Christianson (Ed.). *The handbook of emotion and memory: Research and theory.* (pp. 269-288). Hillsdale, NJ: Lawrence Erlbaum Associates.

LeDoux, J. E. (1995). Setting "stress" into motion: Brain mechanisms of stimulus evaluation. In M. J. Friedman, D. S. Charney & A. Y. Deutch (Eds.), *Neurobiological and clinical consequences of stress: From normal adaptation to PTSD* (pp. 125-134). Philadelphia: Lippincott-Raven Publishers.

LeDoux, J. E. (1996). *The emotional brain: The mysterious underpinnings of emotional life*. New York: Touchstone.

Lenzenweger, M. F., Clarkin, K. 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.

Lewis, L., & Langer, K. G. (1994). Symbolization in psychotherapy with patients who are disabled. *American Journal of Psychotherapy, 48,* 231-239.

Lezak, M. D., Howieson, D. B., Loring, D. W., Hannany, H. J., & Fischer, J. S. (2004). *Neuropsychological assessment* (4th ed.). London: Oxford University Press.

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

Linehan, M. M., Armstrong, H. E., Suarez, A., Allmon, D., & Heard, H. L. (1991). Cognitive-behavioral treatment of chronically parasuicidal borderline patients. *Archives of General Psychiatry, 48,* 1060-1064.

Linehan, M. M., Heard, H. L., & Armstrong, H. E. (1993). Naturalistic follow-up of a behavioral treatment for chronically parasuicidal borderline patients. *Archives of General Psychiatry, 50,* 971-974.

Loring, D. W. (1995, May). *Psychometric detection of malingering*. Paper presented at the meeting of the American Academy of Neurology, Seattle, WA.

Lucas, P. B., Gardner, D. L., Cowdry, R. W., & Pickar, D. (1989). Cerebral structure in borderline personality disorder. *Psychiatry Research, 27*, 111-115.

Lyons-Ruth, K., & Jacobvitz, D. (1999). Attachment disorganization: Unresolved loss, relationship violence, and lapses in behavioral and attentional strategies. In J. Cassidy & P. R. Shaver (Eds.). *Handbook of Attachment: Theory, research, and clinical applications* (pp. 520-693). New York: Cambridge University Press.

Masterson, J. F. (1978). The borderline adolescent: An object relations view. *Adolescent Psychiatry, 6*, 344-359.

McGlashan, T. H., Grilo, C. M., Skodol, A. E., Gunderson, J. G., Shea, M. T., Morey, L. C. et al., (2000). The collaborative longitudinal personality disorders study: Baseline Axis I/II and II/II diagnostic co-occurrence. *Acta Psychiatrica Scandinavica, 102*, 256-264.

Meares, R., Steveson, J., & Gordon, E. (1999). A Jacksonian and biopsychosocial hypothesis concerning borderline and related phenomena. *Australia and New Zealand Journal of Psychiatry, 33,* 831-840.

Meehl, P. (1954). *Clinical vs. statistical prediction.* Minneapolis: University of Minnesota Press.

Meehl, P. (1997). Credentialed persons, credentialed knowledge. *Clinical Psychology: Science and Practice, 4,* 91-98.

Miller, L. (1991). Predicting relapse and recovery in alcoholism and addiction: Neuropsychology, personality, and cognitive style. *Journal of Substance Abuse Treatment, 8,* 277-291.

Milner, B. (1971). Interhemispheric differences in the localization of psychological processes in man. *British Medical Bulletin. 27*, 272-277.

Mitchell, K. J., Johnson, M. K., Raye, C. L., & Greene, E. J. (2004). Prefrontal cortex activity associated with source monitoring in a working memory task. *Journal of Cognitive Neuroscience, 16,* 921-934.

Mittenberg, W., Azrin, R., Millsaps, C., & Heilbronner, R. (1993). Identification of malingered head injury on the Wechsler Memory Scale—Revised. *Psychological Assessment, 5*(1), 34-40.

Mittenberg, W., & Motta, S. (1993). Effects of chronic cocaine abuse on memory and learning. *Archives of Clinical Neuropsychology, 8,* 477-483.

Monarch, E. S., Saykin, A. J., & Flashman, L. A. (2004). Neuropsychological impairment in borderline personality disorder, *Psychiatric Clinics of North America, 27,* 67-82.

Monchi, O., Petrides, M., Petre, V., Worsley, K., & Dagher, A. (2001). Wisconsin Card Sorting revisited: Distinct neural circuits participating in different stages of the task identified by event-related functional magnetic resonance imaging. *Journal of Neuroscience, 21,* 7733-7741.

Nadel, L., & Jacobs, W. J. (1998). Traumatic memory is special. *Current Directions in Psychological Science, 7*, 154-157.

Newman, S. D., Carpenter, P. A., Varma, S., & Just, M. A. (2003). Frontal and parietal participation in problem solving in the Tower of London: fMRI and computational modeling of planning and high-level perception. *Neuropsychologia, 41,* 1668-1682.

Niederhofer, H. (2004). Left-handedness in a sample of nine patients with borderline personality disorder. *Perceptual and Motor Skills, 99,* 849-852.

O'Leary, K. M. (2000). Borderline personality disorder: Neuropsychological testing results. *Psychiatric Clinics of North America, 23*, 41-60.

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.

O'Leary, K. M., & Cowdry, R. W. (1994). In K. R. Silk (Ed.), *Biological and neurobehavioral studies of borderline personality disorder,* (pp.127-157). Washington: American Psychiatric Press.

O'Malley, S. S. & Gawin, F. H. (1990). Abstinence symptomatology and neuropsychological deficits in chronic cocaine abusers. In J. W. Spencer and J. J. Boren (Eds.), *Residual effects of abused drugs on behavior* (National Institute on Drug Abuse Research Monograph, 101, pp. 179-190). Washington D.C.: US Government Printing Office.

Orsini A., Chiacchio L., Cinque M., Cocchiaro C., Schiappa S. O. & Grossi D. (1986). Effects of age, education and sex on two tests of immediate memory: A study of normal subjects from 20-99 years of age. *Perceptual and Motor Skills, 63,* 727-732.

Osterrieth, P. A. (1944). Le test de copie d'une figure complexe [In French]. *Archives de Psychologie, 30*, 206-356.

Pagliaro, L. A., & Pagliaro, A. M. (1999). *Psychologists' neuropsychotropic drug reference.* Philadelphia: Brunner/Mazel.

Parsons, O. A. (1987). Do neuropsychological deficits predict alcoholics' treatment course and posttreatment recovery? In O.A. Parsons, N. Butters, & P.E. Nathan Eds.), *Neuropsychology of alcoholism: Implications for diagnosis and treatment* (pp. 273-290). New York: The Guilford Press.

Pankratz, L., & Binder, L. M. (1997). Malingering on intellectual and neuropsychological measures. In R. Rogers (Ed.), *Clinical assessment of malingering and deception* (2nd ed., pp. 223-236). New York: Guilford Press.

Posner, M.I., Rothbart, M. K., Vizueta, N., Levy, K. N., Evans, D. E., Thomas, K. M. et al. (2002). Attentional mechanisms of borderline personality disorder. *Proceedings of the National Academy of Sciences of the United States of America, 99,* 16366-16370.

Rabin, L. A., Barr, W. B., & Burton, L. A. (2005). Assessment practices of clinical neuropsychologists in the United States and Canada: A survey of INS, NAN, and APA Division 40 members. *Archives of Clinical Neuropsychology, 20,* 33-65.

Rapaport, D., Gill, M. M., & Schafer, R. (1968). *Diagnostic psychological testing* (Rev. ed., R. Holt, Ed). New York: International Universities Press.

Rees, L. M., Tombaugh, T. N., Gansler, D. A., & Moczynski, N. P. (1998). Five validation experiments of the Test of Memory Malingering (TOMM). *Psychological Assessment, 10*(1), 10-20.

Reitan, R. M. (1958). Validity of the Trail Making Test as an indicator of organic brain damage. *Perceptual Motor Skills, 8,* 271-276.

Reitan, R. M., & Davison, L. A. (1974). Clinical Neuropsychology: Current status and applications. New York: Winston-Wiley.

Reitan, R. M., & Wolfson, D. (1993). *The Halstead-Reitan Neuropsychological Test Battery:Theory and clinical applications* (2nd ed.). Tucson, AZ: Neuropsychology Press.

Renneberg, B., Theobald, E., Nobs, M., & Weisbrod, M. (2005). Autobiographical memory in borderline personality disorder and depression. *Cognitive Therapy and Research, 29,* 343-358.

Richardson E. D. & Marottoli, R. A. (1996). Education-specific normative data on common neuropsycholgical indices for individuals older than 75 years. *The Clinical Neuropsychologist, 10,* 375-381.

Rogers, R. (2001). *Handbook of Diagnostic and Structured Interviewing*. New York: The Guilford Press.

Rohling, M. L., Green, P., Allen, III, L. M., & Iverson, G. L. (2002). Depressive symptoms and neurocognitive test scores in patients passing symptom validity tests. *Archives of Clinical Neuropsychology, 17,* 205-222.

Ross, S., Dermatis, H., Levounis, P., & Galanter, M. (2003). A comparison between dually diagnosed inpatients with and without Axis II comorbidity and the relationship to treatment outcome. *American Journal of Drug and Alcohol Abuse,\ 29*, 263-280.

Rosselli, M., & Ardila, A. (1996). Cognitive effects of cocaine and polydrug abuse. *Journal of Clinical and Experimental Neuropsychology, 18,* 122-135.

Ruff, R. M., & Allen, C. C. (1996). *Ruff 2 & 7 Selective Attention Test: Professional Manual.* Lutz, FL: Psychological Assessment Resources.

Ruff, R. M., Light, R. H., & Evans, R. (1987). The Ruff figural fluency test: A normative study with adults. *Developmental Neuropsychology, 3*, 37-51.

Ruocco, A. C. (2005). The neuropsychology of borderline personality disorder: A meta-analysis and review. *Psychiatry Research, 137,* 191-202.

Ruocco, A. C., & Swirsky-Sachetti, T. (2005). Relations between MCMI-II Personality Disorder Scales and Wechsler Adult Intelligence Scale Indices in a head-injury sample. Presented at the Annual Meeting of the Society for Personality Assessment, Chicago.

Ruocco, A. C., & Trobbst, K. K. (2003). Frontal lobe functioning and personality disorder symptomatology. *Archives of Clinical Neuropsychology, 18,* 737.

Sar, V., Kundakci, T., Kiziltan, E., Kizitlan, E., Yargic, I. L., Tutkun, H., et al. (2003). The Axis-I dissociative disorder comorbidity of borderline personality disorder among psychiatric outpatients. *Journal of Trauma & Dissociation, 4*, 119-136.

Schore, A. N. (1994). *Affect Regulation and the Origin of Self: The Neurobiology of Emotional Development.* Hillsdale, New Jersey: Lawrence Erlbaum Associates.

Schulz, S. C. (1983). Ventricular enlargement in teenage patients with schizophrenia spectrum disorder. *American Journal of Psychiatry, 140,* 1592-1595.

Seashore, C. B., Lewis, C., & Saetveit, J. G. (1960). *Seashore measures of musical talent: Manual*. New York: Psychological Corporation.

Shandera, A., Hall, S., DenBoer, J., & Crouse, E. M. (2004, February). *Survey of use of malingering tests by practicing neuropsychologists: Preliminary results*. Poster session will be presented at the annual meeting of the International Neuropsychological Society, Baltimore, MD.

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.

Siegel, D. J. (2001). Toward an interpersonal neurobiology of the developing mind: Attachment relationships, "mindsight," and neural integration. *Infant Mental Health Journal, 22,* 67-94.

Singer, M. T. (1977). The borderline diagnosis and psychological tests: Review and Research. In P. Hartocollis (Ed.), *Borderline personality disorders: The concept, the syndrome, the patient*. New York: International Universities Press.

Slick, D., Hopp, G., Strauss, E., Hunter, M., & Pinch, D. (1994). Detecting dissumulation: Profiles of simulated malingerers, traumatic brain-injury patients, and normal controls on a revised version of Hiscock and Hiscock's forced-choice memory test. *Journal of Clinical and Experimental Neuropsychology, 16,* 472-481.

Slick, D. J., Hopp, G., Strauss, E., & Spellacy, F. J. (1996). Victoria Symptom Validity Test: Efficiency for detecting feigned memory impairment and relationship to neuropsychological tests and MMPI-2 validity scales. *Journal of Clinical and Experimental Neuropsychology, 18*(6), 911-922.

Slick, D. J., Sherman, E. M. S., & Iverson, G. L. (1999). Diagnostic criteria for Malingered neurocognitive dysfunction: Proposed standards for clinical practice and research. *The Clinical Neuropsychologist, 13,* 545-561.

Slick, D. J., Tan, J. E., Strauss, E. H., & Hultsch, D. F. (2004). Detecting malingering: a survey of the experts' practices. *Archives of Clinical Neuropsychology, 19*, 465-473.

Smith, A. (1968). The Symbol Digit Modalities Test: A neuropsychological test for economic screening of learning an other cerebral disorders. *Learning Disorders, 3,* 83-91.

Snyder, S., & Pitts, W. M. (1984). Electroencephalography of DSM-III borderline personality disorder. *Acta Psychiatrica Scandinavica, 69,* 129-134.

Spreen, O., & Strauss, E. (1991). A compendium of neuropsychological tests. New York: Oxford University Press.

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.

Stein, M. B., Koverola, C., Hanna, C., & Torchia, M.G. (1997). Hippocampal volume in women victimized by childhood sexual abuse. *Psychological Medicine, 27,* 951-959.

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.

Stone, M. H. (1980). *The borderline syndromes*. New York: McGraw Hill.

Stuss, D. T., Floden, D., Alexander, M. P., Levine, B., & Katz, D. (2001). Stroop performance in focal lesion patients: Dissociation of processes and frontal lobe lesion location. *Neuropsychologia, 39,* 771-786.

Stuss, D. T., Stethem, L. L., & Pelchat, G. (1988). Three tests of attention and rapid information processing: An extension. *The Clinical Neuropsychologist, 2,* 246-250.

Stuss, D. T., Stethem, L. L., & Poirier, C. A. (1987). Comparison of three tests of attention and rapid information processing across six age groups. *The Clinical Neuropsychologist, 1,* 139-152.,

Suhr, J. A. (2003). Neuropsychological impairment in fibromyalgia: The influence of pain, depression, and fatigue. *Journal of Psychosomatic Research, 55,* 521-529.

Suhr, J. A., & Boyer, D. (1999). Use of Wisconsin Card Sorting Test in the detection of malingering in student simulator and patient samples. *Journal of Clinical and Experimental Neuropsychology, 21,* 701-708.

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.

Tabachnick, B. G., & Fidell, L. S. (1996). *Using multivariate statistics* (3rd edition). New York: HarperCollins.

Tebartz van Elst, L., Hesslinger, B., Theil, T., Geiger, E., Haefele, K., Lemieux, L., et al. (2003). Frontolimbic brain abnormalities in patients with borderline personality disorder: A volumetric magnetic resonance imaging study. *Biological Psychiatry,54,* 163-171.

Tombaugh, T. N. (2006). A comprehensive review of the Paced Auditory Serial Addition Test (PASAT). *Archives of Clinical Neuropsychology, 21,* 53-76.

Tombaugh, T. N. (1996). *TOMM. Test of Memory Malingering.* North Tonawanda: NY: Multi-health systems.

Tombaugh, T. N. (1997). The Test of Memory Malingering (TOMM): Normative data from cognitively intact and cognitively impaired individuals. *Psychological Assessment, 9*(3), 260-268.

Travers, C., & King, R. (2005). An investigation or organic factors in the neuropsychological functioning of patients with borderline personality disorder. *Journal of Personality Disorders, 19,* 1-18.

Travin, S., & Protter, B. (1984). Malingering and malingering-like behavior: Some clinical and conceptual issues. *Psychiatric Quarterly, 56*(3), 189-197.

Uno, H.Tarara, R., Else, J. G., Suleman, M. A., & Sapolsky, R. M. (1989). Hippocampal damage associated with prolonged and fatal stress in primates. *Journal of Neuroscience, 9,* 1705-1711.

Van Gorp, W. G., Wilkins, J. N., Hinkin, C. H., Moore, L. H., Hull, J., Horner, M. D. et al. (1999). Declarative and procedural memory functioning in abstinent cocaine abusers. *Archives of General Psychiatry, 56,* 85-89.

van Reekum, R., Conway, C., Gansler, D., White, R., & Bachman, D. L. (1993). Neurobehavioral study of borderline personality disorder. *Journal of Psychiatry Neuroscience, 18*, 121-129.

van Reekum, R., Links, P. S., Finlayson, M. A., Boyle, M., Boiago, I., Ostrander, L. A. et al. (1996). Repeat neurobehavioral study of borderline personality disorder. *Journal of Psychiatry and Neuroscience, 21*, 13-20.

Verheul, R., Van Den Bosch, L., Koeter, M. W. J., De Ridder, M. A. J., Stijnen, T., & Van Den Brink, W. (2003). Dialectical behavior therapy for women with borderline personality disorder. *British Journal of Psychiatry, 182,* 135-140.

Wechsler, D. (1981) Wechsler Adult Intelligence Scale-Revised. New York: Psychological Corporation.

Wechsler, D. (1997a). WAIS-III: Wechsler Adult Intelligence Scale, Third Edition. San Antonio, Texas: The Psychological Corporation.

Wechsler, D. (1997b). WMS-III: Wechsler Memory Scale, Third Edition. San Antonio, Texas: The Psychological Corporation.

Wechsler, D. (1987). Wechsler Memory Scale – Revised. San Antonio, Texas: The Psychological Corporation.

Westen, D., & Gabbard, G. O., (1999). Psychoanalytic approaches to personality. In L. A. Pervin & O. P. John (Eds.), *Handbook of personality: Theory and research* (2nd ed.), (pp. 154-247). New York: Guilford Press.

Winnicott, D. W. (1991). *The maturation process and the facilitating environment*. New York: International Universities Press.

Witkin, H. A., Oltman, P. K., Raskin, E.,& Karp, S. A. (1971). *A manual for the Embedded Figures Test*. Palo Alto, CA: Consulting Psychologists Press.

Yen, S., Shea, M. T., Battle, C. L., Johnson, D. M., Zlotnick, C., Dolan-Sewell, R. et al. (2002). Traumatic exposure and posttraumatic stress disorder in borderline, schizotypal, avoidant, and obsessive- compulsive personality disorders: Findings from the collaborative longitudinal personality disorder study. *The Journal of Nervous and Mental Disease, 190,* 510-518.

Ylikoski, R., Ylikoski, A., Erkinjuntti, T., Sulkava, R., Keskivaara, P., Raininko, R. et al. (1998). Differences in neuropsychological functioning associated with age, education, neurological status, and magnetic resonance imaging findings in neurological healthy elderly individuals. *Applied Neuropsychology, 5,* 1-14.

Zlotnick, C., Franklin, C. L., & Zimmerman, M. (2002). Is comorbidity of posttraumatic stress disorder and borderline personality disorder related to greater pathology and impairment? *American Journal of Psychiatry, 159*, 1940-1943.

Appendix A

Participant Recruitment Letter

Dear Clinician,
My name is Chris Miller and I am a clinical graduate student at The University of Montana conducting my dissertation study. My research project will look at several areas of functioning in persons with borderline personality disorder (BPD). The study will require participants to complete several neuropsychological measures of memory and processing speed abilities. Individuals participating in the study will have their identity and personal information kept in strict confidentiality. Additionally, the study will be supervised by Dr. Stuart Hall, associate professor in the psychology department at The University of Montana. The study has been approved by the University's Institutional Review Board and all participants will be treated in accordance with the American Psychological Association's (2002) "Ethical Principles of Psychologists and Code of Conduct." If you have clients with a diagnosis of BPD that would be interested in earning $10 to participate in my 2-hour study, I would be very interested in having you refer them to me. Enclosed is a flyer for the study along with my contact information where interested persons may call to schedule an appointment with me. I appreciate your help with this request and I thank you for your time.

Sincerely,

Chris Miller, M.A.
Clinical Psychology Center
The University of Montana
Missoula, MT 59801
Ph: (406) 243.2367

Appendix B

Study Flyer

Are you interested in earning money to answer questions?

You could be involved in a research project at The University of Montana that offers you an opportunity to earn $10 for completing a few tests for about 2 hours.

If you have any questions or think you may be interested in participating in this study, call the person at the number listed below to set up an appointment.

Chris Miller, M.A.
Clinical Psychology Center
The University of Montana
Missoula, MT 59801
Ph: (406) 243.2367

Appendix C

Informed Consent for Warm Springs Participants

Study Title: Neuropsychological Correlates of Borderline Personality Disorder

Investigators: Christopher Miller, M.A.
Department of Psychology
University of Montana
Missoula, MT 59812
(406) 243.4522

Stuart Hall, Ph.D.
Department of Psychology
University of Montana
Missoula, MT 59812
(406) 243.5667

Polly Peterson, Ph.D.
Department of Psychology
Montana State Hospital
PO Box 300
Warm Springs, MT 59706
(406) 693-7120

Please Note

This consent form may contain words that are new to you. If you read any words that are not clear to you, please ask the person who gave you this form to explain them to you.

Purpose

The purpose of this study is to determine how well individuals with borderline personality disorder (BPD) perform on a number of different measures that assess different areas of functioning like memory and attention skills relative to individuals without the diagnosis. We are interested in studying how individuals do on these measures because we believe it will provide valuable information that can be used to improve treatment programs that will better serve clients' needs.

Procedure

If you agree to take part in this research, and complete all the measures, it will involve approximately 2 hours of your time. You will be asked to complete a form that asks about your background information (e.g., date of birth, level of education). An interviewer will ask you about your current emotional problems and your past and present psychiatric history, including questions about depression, trauma history, and drug and alcohol use. You will be asked about your mood, thinking, impulsive behavior and interpersonal relationships. This portion of the study will be recorded, but no identifying information will be included on the audiotape. Because we are looking for participants with some specific characteristics, you may or may not have the characteristics we need for this study. If you do not, you will be told so immediately and you will be paid $5 for your time and you will be finished with the study. If you do have

the characteristics needed for the study, you will be told so immediately. If you decide to continue with the study, you will be asked to complete a series of tests that will assess your attention, memory and ability to work quickly. If you complete the study you will be paid $15 for your effort and time.

Risks/Discomforts

There are no serious risks or discomforts associated with this study. However, answering some of the questions may cause you to sad or upset. If this happens, you can choose not to answer certain questions, or choose not to complete the study. The research assistant will be happy to talk to you about these problems things if they occur.

Benefits of the Research

The study is not specifically designed to benefit you directly. The primary benefit will be to add to our knowledge base about what types of difficulties in functioning persons with BPD have relative to individuals without the diagnosis. The information gathered from this study will increase clinicians' awareness of deficits with this population and improve the effectiveness of treatment.

Confidentiality

You will be assigned a study number, and that number will be used by the researchers to track your data. Information you provide will not leave the hospital with your name attached to it; instead, you will be called (for example) "participant 000" for the purposes of analyzing the research data. The people who work at Montana State Hospital will not have direct access to the answers you give on the tests or to your demographic information. Forms with your name on it (i.e., Informed Consent, HIPPA Authorization for Disclosure of Protected Health Information, Competency Assessment) will be kept separate from your test answers so as not to be identified with you. All data will be stored in locked filed cabinets and accessed only by the study investigators. Finally, if you report that you feel suicidal, feel like harming yourself, or feel like harming someone else, or if you behave as if you are going to do any of those things, the researcher will disclose that information to the hospital staff because of concerns about your safety.

Compensation for Injury

Although we do not foresee any risk in taking part in this study, the following liability statement is required in all University of Montana consent forms:

> In the event that you are injured as a result of this research, you should individually seek appropriate medical treatment. If the injury is caused by the negligence of the University or any of its employees, you may be entitled to reimbursement or compensation pursuant to the Comprehensive State Insurance Plan established by the Department of Administration under the authority of M.C.A., Title 2, Chapter 9. In the event of a claim for such injury, further information may be obtained from the University's Claims Representative or University Legal Counsel. (Reviewed by University Legal Counsel, July 6, 1993).

Voluntary Participation/Withdrawal: Your decision to take part in this research study is entirely voluntary. You may refuse to take part, or you may choose to withdraw from the study at any time without penalty or loss of benefits that you normally receive at Montana State Hospital. Also, you may be asked to leave the study if the Project Director or your treatment providers think it that participation is not in your best interest. Should you choose or be asked to withdraw from the study, the researcher will be available to you following your withdrawal if you would like to discuss your experience with the study. Again, your decision to participate or not participate in this research will in no way affect your treatment at Montana State Hospital.

Questions

If you have any questions about the research or the results of the study, either now, during or after the study, you may contact Christopher Miller (406.243.4522) or Dr. Stuart Hall (406.243.5667). If you have any questions regarding your rights as a research participant, you may contact the Research Offices at The University of Montana at (406) 243.6670.

Participant Statement of Consent

I have read the above description of this research study. I have been informed of the risks and benefits involved, and all of my questions have been answered to my satisfaction. Furthermore, I have been assured that any future questions I may have will also be answered by a member of the research team. I voluntarily agree to take part in this study and consent to be contacted in the future by the researchers to ask if I would be interested in participating in additional studies. I understand I will receive a copy of this consent form.

Printed Name of Participant	Date	Participant ID number

Participant's Signature	Date

Appendix D

Informed Consent Form for Missoula Clinical Participants

Study Title: Neuropsychological Correlates of Borderline Personality Disorder

Investigators: Christopher Miller, M.A.
Department of Psychology
University of Montana
Missoula, MT 59812
(406) 243.4522

Stuart Hall, Ph.D.
Department of Psychology
University of Montana
Missoula, MT 59812
(406) 243.5667

Please Note

This consent form may contain words that are new to you. If you read any words that are not clear to you, please ask the person who gave you this form to explain them to you.

Purpose

The purpose of this study is to determine how well individuals with borderline personality disorder (BPD) perform on a number of different measures that assess different areas of functioning like memory and attention skills relative to individuals without the disorder. We are interested in studying how individuals do on these measures because we believe it will provide valuable information that can be used to improve treatment programs that will better serve clients' needs.

Procedure

If you agree to take part in this research, and complete all the measures, it will involve approximately 2 hours of your time. You will be asked to complete a form that asks about your background information (e.g., date of birth, level of education). An interviewer will ask you about your current emotional problems and your past and present psychiatric history, including questions about depression, trauma history, and drug and alcohol use. You will be asked about your mood, thinking, impulsive behavior and interpersonal relationships. This portion of the study will be recorded, but no identifying information will be included on the audiotape. Because we are looking for participants with some specific characteristics, you may or may not have the characteristics we need for this study. If you do not, you will be told so immediately and you will be paid $5 for your time and you will be finished with the study. If you do have the characteristics needed for the study, you will be told that immediately. If you decide to continue with the study, you will be asked to complete a series of tests that will assess your attention, memory and ability to work quickly. If you complete the study you will be paid $15 for your effort and time.

Risks/Discomforts

There are no serious risks or discomforts associated with this study. However, answering some of the questions may cause you to sad or upset. If this happens, you can

choose not to answer certain questions, or choose not to complete the study. The research assistant will be happy to talk to you about these problems things if they occur.

Benefits of the Research

The study is not specifically designed to benefit you directly. The primary benefit will be to add to our knowledge base about what types of difficulties in functioning persons with BPD have relative to individuals without the disorder. The information gathered from this study will increase clinicians' awareness of deficits with this population and improve the effectiveness of treatment.

Confidentiality

You will be assigned a study number, and that number will be used by the researchers to track your data. Information you provide will not have your name attached to it; instead, you will be called (for example) "participant 000" for the purposes of analyzing the research data. Your informed consent form with your name on it will be kept separate from your test answers so as not to be identified with you. All data will be stored in locked filed cabinets and accessed only by the study investigators. If you report that you feel suicidal, feel like harming yourself, or feel like harming someone else, or if you behave as if you are going to do any of those things, the researcher will take the necessary steps to ensure your safety including consulting with a clinical supervisor.

Compensation for Injury

Although we do not foresee any risk in taking part in this study, the following liability statement is required in all University of Montana consent forms:

> In the event that you are injured as a result of this research, you should individually seek appropriate medical treatment. If the injury is caused by the negligence of the University or any of its employees, you may be entitled to reimbursement or compensation pursuant to the Comprehensive State Insurance Plan established by the Department of Administration under the authority of M.C.A., Title 2, Chapter 9. In the event of a claim for such injury, further information may be obtained from the University's Claims Representative or University Legal Counsel. (Reviewed by University Legal Counsel, July 6, 1993).

Voluntary Participation/Withdrawal

Your decision to take part in this research study is entirely voluntary. You may refuse to take part, or you may choose to withdraw from the study at any time without penalty. Should you choose or be asked to withdraw from the study, the researcher will be available to you following your withdrawal if you would like to discuss your experience with the study.

Questions

If you have any questions about the research or the results of the study, either now, during or after the study, you may contact Christopher Miller (406.243.4522) or Dr. Stuart Hall (406.243.5667). If you have any questions regarding your rights as a research

participant, you may contact the Research Offices at The University of Montana at (406) 243.6670.

Participant Statement of Consent

I have read the above description of this research study. I have been informed of the risks and benefits involved, and all of my questions have been answered to my satisfaction. Furthermore, I have been assured that any future questions I may have will also be answered by a member of the research team. I voluntarily agree to take part in this study and consent to be contacted in the future by the researchers to ask if I would be interested in participating in additional studies. I understand I will receive a copy of this consent form.

Printed Name of Participant	Date	Participant ID number

Participant's Signature	Date

Appendix E

Informed Consent Form for Control Participants

Study Title: Neuropsychological Correlates of Borderline Personality Disorder

Investigators: Christopher Miller, M.A.
Department of Psychology
University of Montana
Missoula, MT 59812
(406) 243.4522

Stuart Hall, Ph.D.
Department of Psychology
University of Montana
Missoula, MT 59812
(406) 243.5667

Please Note

This consent form may contain words that are new to you. If you read any words that are not clear to you, please ask the person who gave you this form to explain them to you.

Purpose

The purpose of this study is to determine how well individuals with borderline personality disorder (BPD) perform on a number of different measures that assess different areas of functioning like memory and attention skills. We are interested in studying how individuals do on these measures because we believe it will provide valuable information that can be used to improve treatment programs that will better serve clients' needs.

Procedure

If you agree to take part in this research, and complete all the measures, it will involve approximately 2 hours of your time. You will be asked to complete a form that asks about your background information (e.g., date of birth, level of education). An interviewer will ask you about your current emotional problems and your past and present psychiatric history, including questions about depression, trauma history, and drug and alcohol use. You will be asked about your mood, thinking, impulsive behavior and interpersonal relationships. This portion of the study will be recorded, but no identifying information will be included on the audiotape. Because we are looking for participants with some specific characteristics, you may or may not have the characteristics we need for this study. If you do not, you will be allowed to continue with the study if you choose to do so. If you decide to continue with the study, you will be asked to complete a series of tests that will assess your attention, memory and ability to work quickly. If you complete the study you will receive your 6 experimental credits.

Risks/Discomforts

There are no serious risks or discomforts associated with this study. However, answering some of the questions may cause you to sad or upset. If this happens, you can choose not to answer certain questions, or choose not to complete the study. The research assistant will be happy to talk to you about these problems things if they occur.

Benefits of the Research

The study is not specifically designed to benefit you directly. The primary benefit will be to add to our knowledge base about what types of difficulties in functioning persons with BPD have relative to individuals without the diagnosis. The information gathered from this study will increase clinicians' awareness of deficits with this population and improve the effectiveness of treatment.

Confidentiality

You will be assigned a study number, and that number will be used by the researchers to track your data. Information you provide will not have your name attached to it; instead, you will be called (for example) "participant 000" for the purposes of analyzing the research data. Your informed consent form with your name on it will be kept separate from your test answers so as not to be identified with you. All data will be stored in locked filed cabinets and accessed only by the study investigators. If you report that you feel suicidal, feel like harming yourself, or feel like harming someone else, or if you behave as if you are going to do any of those things, the researcher will take the necessary steps to ensure your safety including consulting with a clinical supervisor.

Compensation for Injury: Although we do not foresee any risk in taking part in this study, the following liability statement is required in all University of Montana consent forms:

> In the event that you are injured as a result of this research, you should individually seek appropriate medical treatment. If the injury is caused by the negligence of the University or any of its employees, you may be entitled to reimbursement or compensation pursuant to the Comprehensive State Insurance Plan established by the Department of Administration under the authority of M.C.A., Title 2, Chapter 9. In the event of a claim for such injury, further information may be obtained from the University's Claims Representative or University Legal Counsel. (Reviewed by University Legal Counsel, July 6, 1993).

Voluntary Participation/Withdrawal: Your decision to take part in this research study is entirely voluntary. You may refuse to take part, or you may choose to withdraw from the study at any time without penalty. Should you choose or be asked to withdraw from the study, the researcher will be available to you following your withdrawal if you would like to discuss your experience with the study.

Questions

If you have any questions about the research or the results of the study, either now, during or after the study, you may contact Christopher Miller (406.243.4522) or Dr. Stuart Hall (406.243.5667). If you have any questions regarding your rights as a research participant, you may contact the Research Offices at The University of Montana at (406) 243.6670.

Participant Statement of Consent

I have read the above description of this research study. I have been informed of the risks and benefits involved, and all of my questions have been answered to my satisfaction. Furthermore, I have been assured that any future questions I may have will also be answered by a member of the research team. I voluntarily agree to take part in this study and consent to be contacted in the future by the researchers to ask if I would be interested in participating in additional studies. I understand I will receive a copy of this consent form.

Printed Name of Participant	Date	Participant ID number

Participant's Signature	Date

Appendix F

HIPPA AUTHORIZATION FOR DISCLOSURE OF PROTECTED HEALTH INFORMATION

THE UNIVERSITY OF MONTANA
A HYBRID ENTITY AS DEFINED BY HIPAA

AUTHORIZATION FOR DISCLOSURE OF PROTECTED HEALTH INFORMATION TO The University of Montana, Missoula, Department of Psychology **BY ANOTHER ENTITY**

At the request of The University of Montana, Missoula, Department of Psychology, I authorize Montana State Hospital to disclose to The University of Montana, Missoula, Department of Psychology the following (hereinafter a protected health information):

Diagnostic information
Medication information
Assessment information

This protected health information is to be used by The University of Montana, Missoula, Department of Psychology only for the following purpose:

To carry out research in an effort to better characterize and define a specific neurological profile of borderline personality disorder.

I acknowledge that I understand that treatment, payment, enrollment in a health plan, or eligibility for benefits is **not** dependent on my signing of this Authorization. However, the University of Montana, Missoula, Department of Psychology may require my signing of this authorization before I may participate in this study.

I may refuse to sign this authorization if I choose.

Requestor may use or disclose existing protected health information (PHI) or PHI created within the next six (6) months for up to thirty (30) months from the date of my signing this authorization.

At all times, I retain the right to revoke this Authorization, except if the Authorization was obtained as a condition of obtaining insurance coverage. Such revocation must be submitted to The University of Montana, Missoula, Department of Psychology, in writing. The revocation shall be effective *except* to the extent that The University of Montana, Missoula, Department of Psychology, has already used or disclosed information in reliance on the Authorization. I may revoke this authorization by sending written notice to:

Chris Miller, M.A., Department of Psychology, The University of Montana, Missoula, MT, 59812

I have been informed and understand that information used or disclosed pursuant to this Authorization may be subject to redisclosure by recipient of such information, and at that point, the information may no longer be protected under the terms of this Authorization.

I HAVE READ AND UNDERSTAND THIS INFORMATION. I HAVE RECEIVED A COPY OF THE FORM AND I AM THE PATIENT OR AM AUTHORIZED TO ACT ON BEHALF OF THE PATIENT TO SIGN THIS DOCUMENT VERIFYING AUTHORIZATION FOR THE USE OR DISCLOSURE OF THE PROTECTED HEALTH INFORMATION UNDER THE ABOVE STATED TERMS.

_____ _____

Signature Date

_____

Printed Name

_____ _____

Signature of Witness Date

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Appendix G

DEMOGRAPHICS FORM

Participant ID Number__ __ __

1. What is your age? ____ Years old

2. What is your gender? (check one)
   _____ Female
   _____ Male
   _____ Transgender

3. What is your race or ethnic background? (check all that apply)
   _____ African American
   _____ White/Caucasian
   _____ Asian American
   _____ Native American
   _____ Hispanic
   _____ Alaska Native/Pacific Islander
   _____ Other (specify): _____

4. What is your marital status? (check all that apply)
   _____ Single
   _____ Living together
   _____ Married
   _____ Divorced
   _____ Separated
   _____ Widowed

5. What is your highest level of education?
   _____ Eight grade or less
   _____ Some high school
   _____ High school graduate (GED)
   _____ Some college
   _____ College graduate
   _____ Master's degree
   _____ Doctoral degree

	Yes	No
6. Are you currently in therapy?	____	____

If yes, with whom? _____

What is his/her phone number? _____

		Yes	No
7.	Are you currently seeing a psychiatrist? If yes, who? _____ What is his/her phone number? _____	____	____
8.	Are you currently seeing a case manager? If yes, who? _____ What is his/her phone number? _____	____	____
9.	Do you experience any problems with your hearing?	____	____
10.	Are you applying for social security disability income?	____	____
11.	Are you involved with any legal proceedings?	____	____
12.	Have you ever been diagnosed with any neurological condition? (e.g., seizure disorder, encephalitis), or have you ever had a blow to your head in which you were unconscious for longer than 30 minutes? If so, please describe briefly: _____ _____	____	____
13.	Have you ever been diagnosed with a learning disability and/or attention deficit disorder with or without hyperactivity? If so, please describe briefly: _____ _____	____	____
14.	Have you ever been formally diagnosed with borderline personality disorder or told that you have characteristics of BPD?	____	____
15.	Do you have a current substance abuse or dependence diagnosis?	____	____

16. Please list all medications you are currently taking.

Drug Taken	Amount Taken	Time Taken
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____
_____	_____	_____

17. Please list all current mental disorder diagnoses.

1._____
2._____
3._____
4._____
5._____

Appendix H

Order of Test Administration

Informed Consent Form
HIPPA Authorization for Disclosure of Protected Health Information Form (Warm Springs Participants only)
Demographic Information Form
Beck Depression Inventory-II
Structured Clinical Interview for the DSM-IV Personality Disorders - BPD items

California Verbal Learning Test, List A, immediate free recall trials 1-5
California Verbal Learning Test, List B, immediate free recall
California Verbal Learning Test, List A, short-delay free recall
California Verbal Learning Test, List A, short -delay cued recall

Test of Mental Malingering, Trial 1
Test of Mental Malingering, Trial 2

California Verbal Learning Test, List A, long-delay free recall
California Verbal Learning Test, List A, long-delay cued recall
California Verbal Learning Test, List A, long-delay Yes/No recognition

Wechsler Memory Scale-III - Logical Memory I

Ruff 2 & 7 Selective Attention Test

Seashore Rhythm Test

Wechsler Memory Scale-III - Logical Memory II

Wechsler Memory Scale-III - Spatial Span

Wechsler Adult Intelligent Scale-III - Digit Symbol

Wechsler Adult Intelligent Scale-III - Symbol Search

Paced Auditory Serial Test