Journal of Personality Disorders, 18(5), 421-438, 2004

EXECUTIVE NEUROCOGNITIVE FUNCTIONING AND NEUROBEHAVIORAL SYSTEMS INDICATORS IN BORDERLINE PERSONALITY DISORDER: A PRELIMINARY STUDY

Mark F. Lenzenweger, PhD, John F. Clarkin, PhD,
Eric A. Fertuck, PhD, and Otto F. Kernberg, MD

It is argued that borderline personality disorder (BPD) represents the interaction of underlying neurobehavioral systems that are reflected principally in the phenotypic constructs of positive emotion, negative emotion, and nonaffective constraint (Depue & Lenzenweger, 2001). This preliminary and exploratory study sought to examine predictions made from the Depue–Lenzenweger model with respect to controlled (effortful) information processing in BPD. It was hypothesized that (a) BPD subjects may display deficits on tasks that require controlled information processing (sustained attention, spatial working memory, and executive functioning), (b) they may reveal elevated negative emotion as well as decreased positive emotion and nonaffective constraint, and (c) nonaffective constraint should be substantially inversely associated with accurate performance on controlled information processing tasks. The results of this study, which examined 24 BPD diagnosed individuals and 68 normal adults, found support for each of these predictions in relation to performance on the Wisconsin Card Sorting Test. The implications of these results for further experimental psychopathology investigations of BPD as well as further refinement of theoretical models of the disorder are discussed.

The descriptive phenomenologic literature concerning cognitive and emotional processes in borderline personality disorder (BPD) has grown

From the Department of Psychology, Harvard University (M.F.L.); the Personality Disorders Institute and the Department of Psychiatry, Weill Medical College of Cornell University (J.F.C., O.F.K.); and the Department of Psychiatry, Weill Medical College of Cornell University (E.A.F.).

This research was funded in part by a Distinguished Investigator Award to Mark F. Lenzenweger from the National Alliance for Research in Schizophrenia and Depression (NARSAD) and, in part, by the Borderline Personality Disorder Research Foundation. Support for equipment was also provided by Harvard University (Dr. Lenzenweger).

We thank Daniel N. Klein for his comments on an earlier version of this article. We thank Heather Bergida, Simone Hoermann, Kenneth Levy, Judith Leone, Joel McClough, Laurie Scott, and Jennifer Warner for their assistance with data collection.

Eric A. Fertuck is now at the Department of Psychiatry, College of Physicians and Surgeons, Columbia University.

Address correspondence to Mark F. Lenzenweger, Department of Psychology, State University of New York at Binghamton, Science IV, Binghamton, NY 13902–6000; E–mail: mlenzen@binghamton.edu.

considerably over the past 35 years following the groundbreaking clinical observations of Kernberg (1967, 1968) and Grinker, Werble, and Drye (1968). However, to date, relatively little is known about the cognitive and emotional processing of BPD individuals from a laboratory–based research perspective. With respect to cognitive functioning in BPD, some clinical neuropsychological assessments (Judd & Ruff, 1993; Swirsky–Sacchetti et al., 1993) revealed subtle memory deficits on complex (affectively neutral) tasks involving delayed recall. Other investigations of neuropsychological performance in BPD did not reveal compelling evidence of deficits (Driessen et al., 2000; Kunert, Druecke, Sass, & Herpertz, 2003). However, the negative findings of Driessen et al. (2000) are not unambiguous as they reflect the impact of statistical removal of depression from the data (see Meehl, 1971) and the Kunert et al. (2003) study used a nonstandard diagnostic threshold for the categorical diagnosis of BPD (see p. 500). Moreover, the methods of the experimental psychology laboratory (see Lenzenweger & Hooley, 2003; cf. Bornstein, 2003) with its direct probes and process–focused tasks, have only recently been brought to bear upon cognitive and memory functioning in BPD. An example of such work is the creative study by Korfine (Korfine, 1998; Korfine & Hooley, 2000) that examined directed forgetting task performance in BPD, which united the cognitive and emotional realms.

One area of potential interest for the study of cognitive processing in BPD is represented by cognitive functions that require controlled (or effortful) processing (Hasher & Zacks, 1979; Shiffrin & Schneider, 1977; Posner, 1978). As contrasted with "automatic" processes that require minimal effort and minimal processing capacity/resources, "controlled" processes require effort and the allocation of processing capacity/resources for successful execution. Controlled cognitive processes are of considerable interest in the exploration of BPD as such patients are frequently described as impulsive, dyscontrolled, and/or erratic in terms of behavior and psychological functioning (e.g., DSM–IV, APA, 1994). Does such dyscontrol reflect itself in the cognitive functioning of BPD patients on tasks that require control and effort?

Just as more refined laboratory investigations of cognition and emotion in BPD have begun, a recently proposed integrative model has sought to link underlying neurobehavioral systems to personality processes and personality disorder symptomatology (including BPD) (Depue & Lenzenweger, 2001). The Depue–Lenzenweger neurobehavioral model of personality disorders, which draws heavily upon the extant animal and human neurobiological literature, posits that personality disorders represent *emergent* phenomena that result from the *interaction* of underlying neurobehavioral systems. In this model, PDs are described as emergent because the disorders arise from the underlying neurobehavioral systems (in interaction with environmental forces) to create a configural phenotype that is *not readily reducible* to individual input components, although the necessary components can be known. This model, influenced *in part* by the work of Tellegen (1985), involves the agentic extraversion, anxiety, fear, affiliative (bonding), and nonaffective constraint systems, each of which is linked to both behavioral expression and neurobiological processes (see Depue & Lenzenweger, 2001 for details).

Briefly, according to Depue and Lenzenweger (2001), agentic extraversion reflects a positive incentive motivation process involving dopamine; anxiety reflects largely anxiety via tonic aspects of norepinephrine activity; nonaffective constraint reveals itself through control and reflects a modulatory, neural constraint process underpinned by serotonin (5–HT); fear involves phasic aspect or norepinephrine; and affiliation involves oxytocin, vasopression, and endogenous opiate activity. The Depue–Lenzenweger model differs from other conceptualizations of the etiology of PDs as it does *not* view PDs simply as quantitative extremes on dimensions of lexical adjectives (e.g., Five-Factor Model)[1], or as subclinical variants of an Axis I disorder dimension (e.g., 5–HT dysfunction and Cluster B disorders; Siever & Davis, 1991), or the result of numinous constructs (e.g., transcendence, Cloninger, Svrakic, & Przybeck, 1993) (see Depue & Lenzenweger, 2001 for details). In this context we note that propositions regarding other personality trait schemes in relation to BPD specifically (e.g., Five–Factor solution; Pukrop, 2002; Trull, 2001; Zweig & Paris, 1995) and PDs generally are available elsewhere (Saulsman & Page, 2004). Our goal is not an extensive review of that literature, nor can we explore the limitations of other personality models (see Block, 1995, 2001; Shedler & Westen, in press; Westen, 1996).

In the Depue–Lenzenweger model, BPD is reflective of a complex interaction involving (a) diminished incentive motivation (positive emotion) in relation to increased anxiety (negative emotion) in interaction with (b) diminished activity of the modulatory neural (nonaffective) constraint system, and (c) exaggerated reactivity of the fear system within a broader context of affiliation. According to the model, BPD subjects should reveal empirically lower levels of agentic extraversion (positive emotion) and constraint as well as higher levels of anxiety (negative emotion) relative to population norms on a reliable and valid measure of such constructs. The proposition that BPD is characterized, in part, by a deficient nonaffective constraint (i.e., control) system, is of particular salience as it predicts poor modulation (i.e., dysregulation) of both cognitive and emotional systems in

1. The Depue–Lenzenweger model (DLM) of neurobehavioral systems differs from the popular Five–Factor Model (FFM) of Personality trait dimensions in critical ways. The DLM model separates agentic and affiliative components of extraversion, whereas the FFM combines them; the DLM separates anxiety and fear into distinct systems, whereas the FFM combines them; and the DLM emphasizes a nonaffective constraint system and such a system is not represented in the FFM (conscientiousness is *not* nonaffective constraint). The DLM also differs substantially from the Cloninger model (Cloninger, Surakic, & Przybeck, 1993) in that the primary components of the Cloninger model represent heterogenous blends of underlying personality dimensions, whereas the DLM model emphasizes relatively homogenous dimensions corresponding to underlying neurobehavioral systems.

BPD–affected individuals. This proposition regarding the effect of diminished control would suggest that (a) BPD-affected individuals will display lower nonaffective constraint levels relative to population norms and (b) diminished levels of nonaffective constraint should be reliably and inversely correlated with performance on tasks that tap controlled or effortful information processing. One would reasonably predict that the disruption of controlled cognitive processing might be more pronounced on tasks that are more complex and require greater effort for efficient/accurate processing or performance.

To bring together these two broad areas of cognitive processing in BPD and neurobehavioral model–based predictions concerning the disorder, this exploratory preliminary investigation sought to (a) clarify whether BPD subjects display deficits on tasks assessing sustained attention, working memory, and/or executive cognitive processing (all tasks requiring effort and controlled processing), (b) determine whether BPD subjects as a group reveal lower levels of positive emotions and constraint as well as higher levels of negative emotion relative to population norms, and (c) determine whether performance on cognitive processing tasks is substantially related to diminished constraint levels. We also evaluated if any observed associations between the cognitive processing tasks and control would be explained by mental state factors such as anxiety and depression.

METHOD

SUBJECTS

Two samples of subjects were used in the current study; one consisted of those diagnosed with Borderline Personality Disorder and the other represented a normal control group with no prior history of psychosis or BPD. The two groups of subjects were recruited through different facilities, however both drew upon urban Northeast city populations in the U.S.

Clinical BPD Sample. Twenty–four (24) female BPD patients were recruited through the Weill Cornell BPD (New York City metropolitan area) treatment study at the beginning of their treatment. Inclusion criteria included meeting DSM–IV (APA, 1994) BPD criteria on the International Personality Disorders Examination (IPDE; Loranger, 1999) and being between the ages of 18 and 50. The BPD sample contained 15 Caucasian American, 5 African American, 1 Asia American, and 3 Other subjects. Exclusion criteria included any psychotic disorder, substance dependence, mental retardation, and/or bipolar I. Additionally, we excluded any participant with a major depressive episode, eating disorder, and/or current episode of substance abuse the 6 months prior to assessment. The patients completed an extended assessment battery, including evaluation of Axis I diagnoses (SCID–I).

Normal Control Sample. The 68 female normal control subjects were selected from the community in the greater Boston, MA metropolitan region as part of a larger community–based study of psychopathology conducted by MF. Lenzenweger. They were recruited through posters and announcements placed in the communities surrounding Boston that described the study. Upon initial contact with a subject and prior to entrance in the study, *all* subjects were carefully screened by trained research personnel for any prior history of psychosis, head injury, neurological illness, or substance abuse. If subjects passed the initial screen and were between the ages of 18 and 45 years, they were invited to participate in the study. All subjects who entered the protocol were assessed for (a) any prior history of psychosis using a computerized assessment (see below), (b) current alcohol use (using an Alco–Sensor IV breathalyzer), and (c) any BPD features using the International Personality Disorders–Screen (IPDE–S). For this study, a subsample of female subjects was drawn from the large pool of subjects who met all screening criteria *and* had screened positive for no more than one possible BPD feature on the IPDE–S. This resulted in a sample of 68 women. The normal control sample contained 56 Caucasian American, 3 African American, 5 Asia American, and 4 Other subjects.

MEASURES

Clinical and Personality Measures

International Personality Disorder Examination (IPDE; Loranger, 1999; Loranger et al., 1994). The IPDE (Loranger, 1999) is a semistructured interview selected for the WHO–ADAMHA International Pilot Study of Personality Disorders (Loranger et al., 1994), and is designed for use by experienced clinicians for the diagnosis of personality disorders. Prof. Armand W. Loranger provided extensive training to the interviewers on the use and scoring of the IPDE in this study. The reliability and validity of the IPDE are well established (Loranger, 1988, 1999).

International Personality Disorder Examination Screen (IPDE–S; Loranger, 1999). The IPDE–S is the instrument that was designed to be used to screen for possible personality disorders. It is a true/false format instrument and can be used to screen for any of the DSM–IV Axis II disorders. The BPD module of the IPDE–S, which contains one screening item for each of the nine BPD diagnostic criteria, was used in this study. The psychometric characteristics and diagnostic efficiency of the original version of the IPDE–S can be found in Lenzenweger, Loranger, Korfine, & Neff (1997).

Structured Clinical Interview for DSM–IV (SCID–I; First, Spitzer, Gibbon, & Williams,1997). The SCID–I is the structured clinical interview for the assessment of DSM–IV Axis I disorders.

Psychosis Screening. All control subjects completed the self–administered, computerized screening version of the Diagnostic Interview Schedule (DISSI; Robins, Helzer, Croughan, & Ratcliffe, 1981) to assess lifetime presence of a schizophrenia–schizophreniform psychosis. *No* subject in the control group met screening criteria for schizophrenia–schizophreniform psychosis.

Beck Depression Inventory (BDI; Beck, Ward, Mendelsohn, Mock, & Erbaugh, 1961). The BDI is a 21-item, self-report inventory used to measure depressive/dysphoric features.

State-Trait Anxiety Inventory (Form Y; Spielberger, 1983). The State Anxiety scale of the STAI is a 20-item, self-report inventory that was used to measure state anxiety.

Multidimensional Personality Questionnaire (MPQ, Tellegen, 1982; Tellegen & Waller, in press) . The MPQ is a 300–item self–report instrument for the assessment of normal personality features. Its scales represent 11 primary personality dimensions and 3 higher–order traits. The MPQ has excellent psychometric properties, and was administered to the BPD subjects in this study and the resulting raw scores were transformed to *T*–scores (Mean = 50, *SD* = 10) using normative data previously published on the MPQ (DiLalla, Gottesman, & Carey, 1993). The measures of central interest in this study were agentic extraversion (operationalized as MPQ positive emotion [PEM]), anxiety (operationalized as MPQ negative emotion [NEM]), and MPQ control. Control was used as the measure of "nonaffective constraint" as it most closely defines the nonaffective constraint construct articulated in the Depue–Lenzenweger model.[2] Fear has not been evaluated in this study using the MPQ owing to the absence of a *social fear* dimension in the MPQ. The constructs embodied in the MPQ, though similar in some respects to dimensions in other models, are not isomorphic with other popular approaches (see Church, 1994; see footnote 1).

Cognitive Processes Measures

Three measures of controlled cognitive processing were selected for this study. All three tasks involved controlled, executive processing that involved appreciable effort, ranging from lower load in the form of a

2. We acknowledge a useful consultation by Dr. Richard A. Depue regarding the use of MPQ "control" as the measure of choice to operationalize the nonaffective constraint construct. Empirical data supportive of this choice of MPQ control as a measure of nonaffective constraint, particularly with respect to 5–HT activity, can be found in Depue (1995).

high-load sustained attention task, through a more demanding spatial working memory task, to the higher load and more complex WCST.

Continuous Performance Test–Identical Pairs Version (CPT–IP). S u s-tained attention was measured using the Continuous Performance Test–Identical Pairs Version developed by Cornblatt and colleagues, which is described in detail elsewhere (CPT–IP; Cornblatt, Risch, Faris, Friedman, & Frlenmeyer-Kimling, 1988; Cornblatt, Lenzenweger, & Erlenmeyer-Kimling, 1989; Lenzenweger, Cornblatt, & Putnick, 1991). The CPT–IP is a low a priori signal probability task that taps sustained attention. The primary performance indexes for the CPT–IP are the signal detection as *d'* (sensitivity) and *lnβ* (response bias or criterion).

Spatial Delayed Response Task (DRT). A computerized oculomotor spatial delayed response task developed by Luciana, Depue, Arbisi, and Leon (1992) was used to assess spatial working memory. In brief, subjects were required to observe a central fixation point on a computer monitor, after which a visual cue appeared in peripheral vision within a 360° circumference for a period of time too brief in which to make a saccade to the cue (< 200 ms). After the occurrence of the visual cue, the cue and fixation point disappeared, and the screen turned black for delay intervals of 500ms or 8 seconds. After the delay, the screen lit instantly, and the subject indicated the screen location of the cue with a fine–pointed light pen. The point at which the light pen touched the screen was then recorded by the program. The 500-ms and 8-second delay trials were randomly interspersed and the locations of the visual cues were also appropriately randomized (see Luciana et al., 1992 for details). The basic performance index of interest in this protocol concerned the subjects' indicated location of the visual cue on each trial. This index was computed for the 500-ms and 8-second delay and, for each, it was the composite function of the horizontal and vertical distance from the actual cue location. Composite error scores were computed for each trial by the Pythagorean theorem. Average error scores were calculated by taking the mean of the composite error scores that were summed across the total trials administered for each of the two delay intervals. The average error score for the 8-second trials represents the working memory condition and is reported here.

Wisconsin Card Sorting Test (WCST). A computerized WCST was administered (Heaton, 1981) and scored using a computer program (Harris, 1988). The WCST is a neuropsychological task that measures abstraction ability and cognitive flexibility. The WCST is viewed as a measure of executive functioning that evaluates an individual's capacity to adhere to a cognitive principle and respond in a manner consistent with this principle. Further it assesses the individual's capacity to ascertain shifts in rules during the task, and respond to the new rule in a consistent manner. Performance on the WCST is commonly hypothesized to be associated with the functioning of the dorsolateral prefrontal cortex (see Goldman–Rakic, 1991).

PROCEDURES

The BPD study subjects were tested in quiet laboratory rooms by trained research assistants at the Weill Medical College of Cornell University. The research assistants were blind to *all* individual difference information for the BPD subjects. The normal control subjects were also tested in controlled laboratory conditions by experienced research assistants in the first author's laboratory at Harvard University. These research assistants were also blind to all diagnostic and individual difference information about the subjects. The order of administration of the CPT–IP, WCST, and DRT was randomized across subjects at both testing sites. The BPD subjects were paid $30.00 and the normal controls were paid $50.00 for their participation (the normals completed a more extensive test battery). Both the BPD and control subjects completed the CPT–IP, WCST, and DRT at the beginning of their respective testing sessions; all neurocognitive and mental state testing procedures were completed in one session for all subjects, and no subjects reported fatigue during or at the end of the assessments. The clinical diagnostic assessments (IPDE, SCID, MPQ) for the BPD subjects were completed during an assessment session several days prior to the neurocognitive assessment session. The procedures were fully approved by the Internal Review Boards at the Weill Medical College of Cornell University and Harvard University respectively.

STATISTICAL ANALYSIS

The mean performance levels of the BPD and control subjects on the neurocognitive tasks were contrasted using the Mann–Whitney two samples test, due to the heterogeneity of variance in the two groups, and differences were tested with a directional (one–tailed) procedure. Effect size was indexed by Cohen's *d* (Cohen, 1988). To determine whether BPD subjects as a group revealed lower levels of positive emotions and constraint as well as higher levels of negative emotion relative to representative population norms, one–sample *t*–tests were used that assumed a mean of 50. Associations between levels of MPQ control and neurocognitive performance were evaluated using the Spearman correlation coefficient (due to distributional properties) and tested with a one–tailed procedure reflective of the a priori hypotheses. Partial correlations were computed using the Spearman partial correlation procedure [Statistical Analysis System (SAS), v. 9.0; SAS Institute, Inc., 2003)]. Additional analyses were conducted to determine if medication

TABLE 1. Performance of Borderline Personality Disordered (*n* = 24) and Normal Control Subjects (*n* = 67) on the Wisconsin Card Sorting Test

	Subject Groups						
	Borderline (*n* = 24)		Control (*n* = 67)				
Task	***M***	***SD***	***M***	***SD***	***Z***	***p***	***d***
Category	5.33	1.83	5.84	.83	–1.10	.10	.36
Persev. Resp (%)	13.13	6.93	10.19	5.31	–2.37	.009	.48
Persev. Err (%)	12.08	6.30	9.67	4.44	–2.27	.012	.44
Errors (%)	24.08	15.30	18.73	9.72	–1.76	.039	.42

Note. WCST –Wisconsin Card Sorting Test. The Z–value is the normal variate for the test statistic from a Mann–Whitney U test, the p–values are exact and based on one–tailed tests of significance as detailed in the text. *d* = Cohen's effect size index. The n for the controls on the WCST was 67 due to one lost case, due to equipment failure.

status of the BPD subjects was associated with their cognitive processing performance as well as to determine if mental state factors (depression, anxiety) were related to performance on the neurocognitive measures.

RESULTS

All of the subjects in the present study were female. The average age of the normal controls was 29.24 yrs ($SD = 6.67$) and for the BPD subjects it was 31.92 yrs. ($SD = 9.15$), which did not differ significantly ($t(90) = 1.32$, $p < .20$, two–tailed). Education was coded ordinally on a 6–point scale ranging from less than 12 years to greater than 16 years of education. The mean educational level of the normal controls was 4.93 ($SD = .89$) and 4.33 ($SD = 1.52$) for the BPD subjects, which did *not* differ significantly [$t(89) = 1.47$, ns]. The racial composition of the normal control group was 82.4% Caucasian, 4.4% African American, 7.4% Asian/Pacific Islander, 5.9% Other (NonHispanic), and among the BPD subjects it was 62.5% Caucasian, 20.8% African American, 4.2% Asian/Pacific Islander, 12.5% Other (NonHispanic).

In addition to being diagnosed for BPD according to the DSM–IV criteria, the BPD sample subjects were also assessed for depression using the BDI as well as state anxiety using the STAI. The mean BDI score for BPD subjects was 17.16 ($SD = 11.18$) and mean State Anxiety (STAI) score for the BPD subjects was 51.17 ($SD = 14.67$). As per our exclusion rules, none of the BPD subjects in this sample currently have (or had in the past) bipolar disorder, delusional disorder, or schizophrenia. No BPD subject had a current substance dependence diagnosis. With respect to other Axis I conditions, 83.3% of the sample met the criteria for at least 1 *current* Axis I condition. Specific current Axis I disorder frequencies in the sample were as follows: Dysthymia (41.7%), Depressive Disorder NOS (4.2%), Panic Disorder (without agoraphobia) (8.3%), Panic Disorder (with agoraphobia) (4.2%), Social Phobia (12.5%), Obsessive–Compulsive Disorder (8.3%), Posttraumatic Stress Disorder (4.2%), Generalized Anxiety Disorder (16.7%), and Eating Disorder NOS (33.3%). space. This general pattern of comorbid Axis I condition is not uncommon for BPD patients (see APA, 1994). History of past Axis I disorders is consistent with this general pattern for current disorders, but those data were omitted to conserve space. Among the BPD patients, 87.5% had at least one other comorbid Axis II diagnosis.

Comparison of the BPD and Normals on the Cognitive Process Measures

The BPD and normal controls on the CPT–IP, DRT, and WCST were compared using the Mann–Whitney test for the comparison of means. For the CPT–IP (sustained attention), the BPD subjects did not differ from controls on either *d'* or *lnβ* ([BPD *d' M*=2.16 (*SD* = .96) vs. Control *d' M*=2.17 (*SD* = .76), *Z*= –.26, *ns*, *d* = .01; BPD *lnβ M* = .36 (*SD* = 1.04) vs. Control *lnβ M* = .62 (*SD* = .90), *Z* = –.97, *ns*, *d* = .27]). For the DRT (spatial working memory), the BPD (*M*=11.26, *SD* = 4.17) and control (*M* = 10.78, *SD* = 2.45) subjects did not differ (*Z* = –11, *ns*, *d* = .14).

The results of the BPD versus control comparisons for the Wisconsin Card Sorting Test can be found in Table 1. The performance of the two groups on the WCST, however, is characterized by clear–cut and statistically significant differences across three of the four WCST performance indexes of interest (percentage of perseverative responses, percentage of perseverative errors, and percentage of errors, all $p < .05$), with the remaining index (categories achieved) revealing a trend ($p < .10$) toward difference. All of the differences on the WCST are in the direction of BPD subjects performing more poorly relative to controls. Of greater scientific interest than *p*–values, the effect sizes (Cohen's *d*) for these differences are displayed in Table 1 and all indicate "medium"–sized effects (Cohen, 1988). Finally, statistical control for education (as a proxy for intelligence) and age did *not* alter this pattern of findings substantially.

Within the BPD group, a subgroup of BPD subjects ($n = 17$) was medicated whereas the remainder ($n = 7$) were not. A comparison of the performance of the medicated versus nonmedicated BPD subjects did *not* reveal a single statistically significant difference on the CPT–IP, DRT, and WCST performance indexes reported in Table 1, no $p < .50$ (two–tailed). These results are summarized here to conserve space.

Evaluation of the MPQ Indexes for the BPD Subjects

As noted above the three personality constructs of central interest to this study are PEM, NEM, and control (nonaffective constraint). Recalling that the Depue–Lenzenweger (2001) predicts that BPD subjects should reveal lower levels of PEM and control as well as higher levels of

TABLE 2. Associations among Control, Positive Emotion, Negative Emotion, Depression, Anxiety, and Wisconsin Card Sorting Test Performance in BPD Patients (n = 24)

	MPQ–Control	MPQ–PEM	MPQ–NEM	BDI–Depression	STAI–State Anxiety
Category	.20	.19	–.12	–.07	.02
Persev. Resp (%)	–.66****	.02	–.13	–.07	–.02
Persev. Err (%)	–.41*	–.04	.05	.13	–.04
Errors (%)	–.36*	–.13	.16	.14	–.04

Note. Values reported are Spearman correlation coefficients, tested for significance using a one–tailed test as detailed in the text. †$p < .10$. *$p < .05$. **$p < .0002$.

NEM relative to population norms, the means and *SD*s for these MPQ dimensions, in *T*–score form, were as predicted: PEM (M = 38.88, SD = 11.03), NEM (M= 63.84, SD = 11.36), and control (M = 41.55, SD = 12.37).[3] These mean values for PEM, NEM, and control were tested against the population *T*–score mean of 50 using a 1-sample *t*–test. All three of the group means, PEM, NEM, and control, differed significantly from the test value of 50 as follows: PEM [$t(23) = 4.89$, $p < .0001$], NEM [$t(23) = 5.97$, $p < .0001$], and control [$t(23) = 3.35$, $p < .003$).

Associations between the WCST indexes and Individual Difference Measures

Our third hypothesis concerned an explicit prediction with respect to the relationship between the control (nonaffective constraint) dimension and performance on the neurocognitive laboratory tasks. To investigate this hypothesis, we examined the associations between control and the WCST indexes listed in Table 1. We did not examine the CPT–IP and DRT indexes owing to the lack of group differences for these tasks. As can be seen from Table 2, the control dimension of the MPQ was substantially and inversely related to measures of poor performance on the WCST. Higher levels of control were associated with lower levels of perseverative responses (%), perseverative errors (%), and errors (%) on the WCST. We also examined the WCST indexes in relation to PEM and NEM and found *none* of these correlations to be statistically significant. In fact, the WCST indexes that were most strongly correlated with MPQ control [i.e., perseverative responses (%) and perseverative errors (%)]

3. We note that the values for PEM, NEM, and control for this sample of 24 BPD subjects do not reflect the influence of outliers in the sample based on boxplot analysis. Moreover, the means and *SD*s for these same measures (PEM, NEM, control) were obtained on a much larger series of DSM–IV=diagnosed BPD subjects at our center (N = 92) and the results are virtually identical to those reported here for the 1-sample *t*–tests (see McClough, Clarkin, Lenzenweger, & Kernberg, 2003)

were essentially unrelated to both PEM and NEM for practical purposes. Depressive features (BDI) and state anxiety (STAI–S) were also essentially unrelated to WCST performance (Table 2). One might consider whether the robust relationships between MPQ control and the three WCST indexes with which it was significantly associated would remain significant even if the effect of depression and anxiety were removed from these associations. This concern was addressed through the use of Spearman partial correlations. The Spearman partial correlation coefficients taking into account anxiety and depression for the MPQ and WCST indexes are as follows: Control × Perseverative Responses (%) $pr_s = -.71$ ($p < .0001$); Control × Perseverative errors (%) $pr_s = -.59$ ($p < .002$); Control × Conceptual Level (%) $pr_s = .47$ ($p < .013$); Control × Trials Administered $pr_s = -.41$ ($p < .03$); and Control × Errors (%) $pr_s = -.55$ ($p < .004$). Clearly, removal of the mental state factors of depression and anxiety did not diminish the MPQ control × WCST relationships. It appears that depression and/or anxiety may actually be serving a *suppressor* function in these relationships such that the removal of their influence allows the associations between MPQ control and the WCST to be more pronounced. This would be consistent with control revealing its role as a *nonaffective* modulatory or constraint process. Finally, removal of the effects of education (as a proxy for intelligence) did not alter the significant zero order relations between the MPQ Control and WCST variables.

DISCUSSION

The principal goals of this preliminary investigation were to (a) examine controlled information processing (sustained attention, working memory, and executive functioning) in BPD subjects relative to normal (nonBPD) control subjects using established laboratory measures, (b) characterize those BPD subjects in this sample in terms of positive emotion, negative emotion, and nonaffective constraint (control); and (c) examine the associations between nonaffective constraint (control) in relation to performance on the controlled information processing tasks. These particular vectors of inquiry were explicitly informed by the Depue–Lenzenweger (2001) neurobehavioral model of personality disorders. The primary results of the present study can be summarized succinctly. Individuals diagnosed with BPD display deficits on a task that putatively taps higher processing load executive functioning (i.e., Wisconsin Card Sorting Test) relative to normal subjects who do not have BPD features, whereas the two subject groups did not differ with respect to performance on tasks that tap sustained attention or spatial working memory. The differences on the WCST indexes were reflective of "medium" effects. The BPD subjects in this sample display elevated levels of negative emotion as well as decreased levels of positive emotion and nonaffective constraint relative to population norms. This finding is also broadly consistent with those found in other personality–based approaches to BPD (see Saulsman & Page, 2004 for review). Finally, the deficits observed for the BPD subjects on the WCST were substantially associated with decreased levels of control (nonaffective constraint). These associations between control and WCST performance did *not* reflect the impact of state anxiety and/or depression. The WCST deficits were *not* significantly related to either PEM or NEM, which enhances the *specificity* of the findings observed for control process.

How do these neurocognitive results compare with what is known about attention, memory, and executive functioning in BPD? As noted above, this literature is virtually nonexistent and we are currently unaware of any other study that has examined *sustained attention* or *spatial working memory* performance in BPD patients. There have been two prior studies that examined WCST performance in BPD (Swirsky–Sacchetti et al., 1993; Van Reekum et al., 1996); however both of these studies are marred by substantial methodological artifacts that preclude meaningful interpretation of the reported results. The Swirsky–Sacchetti et al. (1993) study examined only ten BPD and ten normal subjects and, therefore, lacked statistical power for detecting group differences. The Van Reekum et al. (1996) report did *not* include a normal control group for comparison with the BPD subjects and, moreover, a large proportion (54%) of those BPD subjects studied had suffered brain injury.

To date, only memory processes have been carefully probed in BPD. The most directly relevant study was the investigation conducted by Korfine (Korfine, 1998; Korfine & Hooley, 2000), which examined directed forgetting task performance in BPD. This study revealed evidence consistent with enhanced encoding of BPD-salient words and perhaps, related themes, in BPD individuals. Korfine (1998; Korfine & Hooley, 2000) suggested that a disinhibitory process might be one possible explanation for the tendency of BPD patients to remember BPD– relevant words that they were told to forget. One could view the results of the present study as complementary to this notion in that diminished control was substantially associated with impairments on the WCST. It may be that cognitive processes in interaction with the neurobehavioral control system represent an important focus for further dissection.

How do the results with respect to PEM, NEM, and control obtained in this study relate to prior work in BPD and BPD–related psychopathology? The BPD subjects in this study were characterized by an elevated level of NEM as well as decreased levels of PEM and control. Lenzenweger, Clarkin, Kernberg, and Foelsch (2001) found that the borderline personality organization constructs of identity diffusion and primitive psychological defensive behaviors were associated with diminished levels of PEM and indexes of aggressive dyscontrol (i.e., assault, irritability) as well as elevated levels of NEM. McClough et al. (2003) also found that BPD patients revealed diminished PEM and control (i.e., nonaffective constraint) as well as elevated NEM as measured by the MPQ. In a broadly relevant vein, studies of the Five–Factor approach to personality have found BPD features to be associated with neuroticism and disagreeableness (e.g., Trull, 2001; see Saulsman & Page, 2004 for review), but it is noted that FFM constructs are not identical to PEM, NEM, or control (Church, 1994).

In evaluating our results, several caveats are in order. First, our sample consisted entirely of females and these results obviously require replication in a male BPD sample. Second, this was a preliminary investigation and it did not contain the equivalent of a psychiatric control group; however we have *not* claimed that the deficits on the WCST are specific to BPD. We did not see deficits in sustained attention and spatial working memory in this BPD sample, whereas deficits in these areas have been observed repeatedly among schizophrenia cases and nonpsychotic schizotypes (Lenzenweger, 1998). Moreover, the BPD subjects in this sample did not show a deficit on the WCST index known as failure to maintain set (FMS) relative to the control subjects ($p < .85$), whereas nonpsychotic schizotypic subjects have been repeatedly shown to have elevated FMS scores (e.g., Lenzenweger, 1998). This study did have critical "within-subject" controls for anxiety and depression, which were shown *not* to attenuate the observed relations between MPQ control and WCST performance. Thus, it is not likely that depression or anxiety account for the control × WCST associations. Future studies of neurocognitive processes in BPD should consider inclusion of other personality disorders as potential control groups.

Third, we chose to focus on measures of sustained attention, spatial working memory, and WCST performance in BPD subjects in this initial effort; however this list is clearly not exhaustive. Fourth, we note that the WCST is a complex task that multiple component processes subserve performance on this task. Although the WCST is argued to be an index of executive functioning, future research is needed to determine the precise nature of the difficulty BPD subjects have in completing the WCST accurately, particularly given that our BPD subjects did not display working memory or sustained attention deficits. Our findings regarding control may prove to be a useful signpost here—that is illumination of which aspect(s) of the multiple processes tapped by the WCST requires the most control for effective WCST performance. Fifth, our research was done under controlled and affectively neutral laboratory testing conditions. It is quite possible that executive cognitive processes in BPD can be differentially disrupted in the presence of emotionally salient stimuli/contexts. In fact, the work of Korfine (Korfine & Hooley, 2000) provides empirical evidence for the impact of emotionally valenced stimuli on memory functioning in BPD. We note that other studies of BPD using experimental tasks and emotionally valenced probes have just recently begun to appear (e.g., Ceumern–Lindenstjerna, Brunner, Parzer, & Fiedler, 2002, Herpertz et al., 2001; Sprock et al., 2000, Wagner & Linehan, 1999); however the results of these studies are inconsistent with respect to the extent emotional stimuli disrupt information processing in BPD and the diagnostic *specificity* of any observed disruptions in BPD subjects remains unclear (e.g., Herpertz et al., 2001). Therefore, in future studies, we plan to undertake studies to evaluate neurocognitive processing using meaningfully affectively valenced stimuli.

Finally, we view this investigation as having been undertaken within a context of discovery and exploration; we do not view it as definitive or confirmatory. This is particularly salient with respect to the implications this study has for the Depue–Lenzenweger (2001) model of personality disorder. This study should not be construed as a definitive test of that model; rather it represents a first step in a developing program of research on but one disorder in a much larger, integrative model. Many studies will be needed to produce what some might term a “strong test” of this model, many of which will need to be done at the interface of personality, cognitive neuroscience, and neurobiology.

In summary, BPD patients appear to be characterized by generally intact sustained attention and spatial working memory functions, when tested under controlled and affectively neutral laboratory conditions, whereas they manifest impairments in executive functioning as indexed by the WCST relative to normal community control subjects. Furthermore, BPD patients are characterized by diminished levels of PEM and control as well as elevated levels of NEM as predicted by the Depue–Lenzenweger (2001) model. Diminished control (nonaffective constraint) appears to be strongly associated with poor neurocognitive (executive functions) performance on the WCST in a manner suggestive of a disinhibitory process, consistent with the theoretical conjectures of Depue and Lenzenweger (2001) from a neurobehavioral perspective, as well as Korfine (1998; Korfine & Hooley, 2000) from a cognitive science framework. Finally, we note that the laboratory–based probing of neurocognitive processes has been of considerable utility in the study of schizophrenia and schizotypy (see Lenzenweger & Hooley, 2003; Lenzenweger, 1998) with the goal being the identification of potential endophenotypes (Gottesman & Gould, 2003) and their subsequent inclusion in other forms of investigation (e.g., genetic research). We see comparable long–term utility in a laboratory–based approach to the study of BPD personality, neurocognitive, and emotional processes.

REFERENCES

American Psychiatric Association. (APA). (1994). *Diagnostic and statistical manual of mental disorders (4th ed.)*. Washington, DC: Author.

Beck, A.T., Ward, C.H., Mendelsohn, M., Mock, J.E., & Erbaugh, J.K., 1961. An inventory for measuring depression. *Archives of General Psychiatry*, *4*, 561–571.

Block, J. (1995). A contrarian view of the Five–Factor approach to personality description. *Psychological Bulletin*, *117*, 187–215.

Block, J. (2001). Millenial contrarianism: The Five–Factor approach to personality description 5 years later. *Journal of Research in Personality*, *35*, 98–107.

Bornstein, R.F. (2003). Behaviorally referenced experimentation and symptom validation: A paradigm for 21st–century personality disorder research. *Journal of Personality Disorders*, *17*, 1_18.

Ceumern–Lindenstjerna, I–A., Brunner, R., Parzer, P., Fiedler, P., & Resch, F. (2002). Borderline–Stoerung and Verzerrungen der Aufmerksamkeit. Theoretische Modelle and empirische Befunde [Borderline personality disorder and attentional biases. Theoretical models and empirical findings]. *Fortschritte der Neurologie, Psychiatrie, 70*, 321–330.

Church, A.T. (1994). Relating the Tellegen and Five–Factor Models of personality structure. *Journal of Personality and Social Psychology, 67*, 898–909.

Cloninger, C., Svrakic, D., & Przybeck, T. (1993). A psychobiological model of temperament and character. *Archives of General Psychiatry, 50*, 975–990.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences* (2nd ed.). Hillsdale, NJ: Erlbaum.

Cornblatt, B.A., Lenzenweger, M.F., & Erlenmeyer–Kimling, L. (1989). The Continuous Performance Test, Identical Pairs Version: II. Contrasting attentional profiles in schizophrenic and depressed patients. *Psychiatry Research, 29*, 65–85.

Cornblatt, B.A., Risch, N.J., Faris, G., Friedman, D., & Erlenmeyer– 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.

Depue, R.A. (1995). Neurobiological factors in personality and depression. *European Journal of Personality, 9*, 413–439.

Depue, R.A., & Lenzenweger, M.F. (2001). A neurobehavioral dimensional model of personality disorders. In W. J. Livesley (Ed.), *The handbook of personality disorders* (pp. 136–176). New York: Guilford.

Dilalla, D.L., Gottesman, I.I., & Carey, G. (1993). Assessment of normal personality traits in a psychiatric sample: Dimensions and categories. In L.J. Chapman, J.P. Chapman, & D. C. Fowles (Eds.), *Progress in experimental personality & psychopathology research* (vol. 16 pp. 137–162). New York: Springer Publishing Company.

Driessen, M., Hermann, J., Stahl, K., Zwann, M., Meier, S., Hill, A., Osterheider, M., & Peterson, D. (2000). Magnetic resonance imaging volumes of the hippocampus and the amygdala in women with borderline personality disorder and early traumatization. *Archives of General Psychiatry, 57*, 1115–1122.

First, M.B., Spitzer, R.L., Gibbon, M., & Williams, J.B.W. (1997). *User's guide for the Structured Clinical Interview for DSM–IV Axis I Disorders – Clinical Version (SCID–CV)*. Washington, DC: American Psychiatric Press.

Goldman_Rakic, P.S. (1991). Prefrontal cortical dysfunction in schizophrenia: The relevance of working memory. In B. Carroll (Ed.), *Psychopathology and the brain*. New York: Raven Press.

Gottesman, I.I., & Gould, T.D. (2003). The endophenotype concept in psychiatry: Etymology and strategic intentions. *American Journal of Psychiatry, 160*, 636–645.

Grinker, R.R., Werble, R., & Drye, R.C. (1968). *The borderline syndrome: A behavioral study of ego functions*. New York: Basic Books.

Harris, M.E. (1988). *Wisconsin Card Sorting Test: Computer version, research edition*. Odessa, FL: Psychological Assessment Resources, Inc.

Hasher, L., & Zacks, R.T. (1979). Automatic and effortful processes in memory. *Journal of Experimental Psychology: General, 108*, 356_388.

Heaton, R.K. (1981). *Wisconsin Card Sorting Test Manual*. Odessa, FL: Psychological Assessment Resources, Inc.

Herpertz, S., Dietrich, T.M., Wenning, B., Krings, T., Erberich, S.G., Willmes, K., Thron, A., & Sass, H. (2001). Evidence of abnormal amygdala functioning in borderline personality disorder: A functional MRI study. *Biological Psychiatry, 50*, 292–298.

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

Kernberg, O.F. (1967). Borderline personality organization. *Journal of the American Psychoanalytic Association, 15*, 641–685.

Kernberg, O.F. (1968). The treatment of patients with borderline personality organization. *International Journal of Psycho–analysis, 49*, 600–619.

Korfine, L. (1998). *Memory functioning in borderline personality disorder*. Unpublished doctoral dissertation, Harvard University, Cambridge, MA.

Korfine, L., & Hooley, J.M. (2000). Directed forgetting of emotional stimuli in borderline personality disorder. *Journal of Abnormal Psychology, 109*, 214–221.

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.

Lenzenweger, M.F. (1998). Schizotypy and schizotypic psychopathology: Mapping an alternative expression of schizophrenia liability. In M.F. Lenzenweger & R.H. Dworkin (Eds.), *Origins and development of schizophrenia: Advances in experimental psychopathology* (pp. 93–121).Washington, DC: American Psychological Association.

Lenzenweger, M.F., Clarkin, J.F., Kernberg, O.F., & Foelsch, P. (2001). The Inventory of Personality Organization: Psychometric properties, factorial composition and criterion relations with affect, aggressive dyscontrol, psychosis–proneness, and self domains. *Psychological Assessment*, *4*, 577–591.

Lenzenweger, M.F., Cornblatt, B.A., & Putnick, M.E. (1991). Schizotypy and sustained attention. *Journal of Abnormal Psychology*, *100*, 84–89.

Lenzenweger, M.F., & Hooley, J.M., (Eds.). (2003). *Principles of experimental psychopathology: Essays in honor of Brendan A. Maher*. Washington, DC: American Psychological Association.

Lenzenweger, M.F., Loranger, A.W., Korfine, L., & Neff, C. (1997). Detecting personality disorders in a nonclinical population: Application of a two–stage procedure for case identification. *Archives of General Psychiatry*, *54*, 345–351.

Loranger, A.W. (1988). *Personality disorder examination* manual. Yonkers, NY: DV Communications.

Loranger, A.W. (1999). *International personality disorder examination: DSM–IV and ICD–10 interviews*. Odessa, FL: Psychological Assessment Resources, Inc.

Loranger, A.W., Sartorius, N., Andreoli, A., Berger, P., Channabasavanna, S.M., Coid, B., Dahl, A., Diekstra, R.F.W., Ferguson, B., Jacobsberg, L.B., Mombour, W., Pull, C., Ono, Y., & Regier, D. (1994). The International Personality Disorder Examination (IPDE): The World Health Organization/Alcohol, Drug Abuse, and Mental Health Administration International Pilot Study of Personality Disorders. *Archives of General Psychiatry*, *51*, 215–224.

Luciana, M., Depue, R.A., Arbisi, P., & Leon, A. (1992). Facilitation of working memory in humans by a D2 dopamine receptor agonist. *Journal of Cognitive Neuroscience*, *4*, 58–68.

McClough, J., Clarkin, J.F., Lenzenweger, M.F., & Kernberg, O.F. (2003). *The Inventory of Personality Organization (IPO): Psychometric properties and criterion relations with affect, constraint, aggression, and psychopathy in a clinical sample of borderline personality disorder patients*. Manuscript submitted for publication.

Meehl, P.E. (1971). High school yearbooks: A reply to Schwarz. *Journal of Abnormal Psychology, 77,* 143–148.

Posner, M.I. (1978). *Chronometric explorations of mind*. New York: Wiley.

Pukrop, R. (2002). Dimensional personality profiles of borderline personality disorder in comparison with other personality disorders and healthy controls. *Journal of Personality Disorders*, *16*, 135–147.

Robins, L.N., Helzer, J.E., Croughan, J., & Ratcliffe, K.S. (1981). National Institute of Mental Health Diagnostic Interview Schedule: Its history, characteristics, and validity. *Archives of General Psychiatry*, *38*, 381–389.

SAS Institute, Inc. (2003). *Statistical Analysis System* (Version. 9.0) user's guide. Cary, NC: Author.

Saulsman, L.M., & Page, A.C. (2004). The Five–Factor Model and personality disorder empirical literature: A meta–analytic review. *Clinical Psychology Review*, *23*, 1055–1085.

Shedler, J., & Westen, D. (in press). Dimensions of personality pathology: An alternative to the five factor model. *American Journal of Psychiatry*.

Shiffrin, R.M., & Schneider, W. (1977). Controlled and automatic human information processing: II. Perceptual learning, automatic attending and a general theory. *Psychological Review*, *84*, 127_190.

Siever, L.J., & Davis, K.L. (1991). A psychobiological perspective on the personality disorders. *American Journal of Psychiatry*, *148*, 1647–1658.

Spielberger, C.D. (1983). *Manual for the State–Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press, Inc.

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.

Tellegen, A. (1982). *Multidimensional Personality Questionnaire manual.* Minneapolis, MN: University of Minnesota Press.

Tellegen, A. (1985). Structures of mood and personality and their relevance to assessing anxiety, with an emphasis on self–report. In A.H. Tuma & J.D. Maser, (Eds.) *Anxiety and the anxiety disorders* (pp.681–706). Hillsdale, NJ: Erlbaum.

Tellegen, A., & Waller, N.G. (in press). Exploring personality through test construction: Development of the Multidimensional Personality Questionnaire. In S.R. Briggs & J.M. Cheek (Eds.), *Personality measures: Development and evaluation (vol. 1).* Greenwich, CT: JAI Press.

Trull, T. J. (2001). Structural relations between borderline personality disorder features and putative etiological correlates. *Journal of Abnormal Psychology*, *110*, 471–481.

Van Reekum, R., Links, P. S., Finlayson, M. A., Boyle, M., Boiago, I., Ostander, L. A., & Moustacalis, E. (1996). Repeat neurobehavioral study of borderline personality disorder. *Journal of Psychiatry and Neuroscience*, *21*, 13–20.

Westen, D. (1996). A model and method for uncovering the nomothetic from the idiographic: An alternative to the Five Factor Model? *Journal of Research in Personality*, *30*, 400–413.

Zweig, F.H., & Paris, J. (1995). The Five–Factor Model of Personality in borderline and nonborderline personality disorders. *Canadian Journal of Psychiatry*, *40*, 523–526.