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Evaluation of behavioral impulsivity and aggression tasks as endophenotypes for borderline personality disorder

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Abstract

Borderline personality disorder (BPD) is marked by aggression and impulsive, often self-destructive behavior. Despite the severe risks associated with BPD, relatively little is known about the disorder's etiology. Identification of genetic correlates (endophenotypes) of BPD would improve the prospects of targeted interventions for more homogeneous subsets of borderline patients characterized by specific genetic vulnerabilities. The current study evaluated behavioral measures of aggression and impulsivity as potential endophenotypes for BPD. Subjects with BPD (N = 127), a non cluster B personality disorder (OPD N = 122), or healthy volunteers (HV N = 112) completed self report and behavioral measures of aggression, motor impulsivity and cognitive impulsivity. Results showed that BPD subjects demonstrated more aggression and motor impulsivity than HV (but not OPD) subjects on behavioral tasks. In contrast, BPD subjects self-reported more impulsivity and aggression than either comparison group. Subsequent analyses showed that among BPD subjects behavioral aggression was associated with self-reported aggression, while behavioral and self-report impulsivity measures were more modestly associated. Overall, the results provide partial support for the use of behavioral measures of aggression and motor impulsivity as endophenotypes for BPD, with stronger support for behavioral aggression measures as an endophenotype for aggression within BPD samples.

Keywords

Borderline personality disorder; Endophenotype; Aggression; Impulsivity

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Contributors

Dr. McCloskey analyzed the data, wrote the first draft of the manuscript, and worked on subsequent revisions. Drs. New and Goodman revised the manuscript. Drs. Coccaro and Siever designed the study and wrote the protocol. Drs. Siever and Koenigsberg also edited and revised the manuscript. Dr. Flory assisted with statistical analyses.

Conflict of Interest statement

None declared.

1. Introduction

Borderline personality disorder (BPD) is a chronic, debilitating mental illness that affects approximately 2% of individuals in the community (Coid et al., 2006; Samuels et al., 2002) and up to 20% of psychiatric inpatient samples (Skodol et al., 2002). Marked by an unstable self-concept, poor impulse control and emotional dysregulation, individuals with BPD often show severe distress and impairment in interpersonal functioning that result in significant suffering for the patient and their loved ones. Roughly three-quarters of all BPD patients report engaging in suicidal behavior at some point (Paris et al., 2004; Paris and Zweig-Frank, 2001; Zanarini et al., 2004) with up to 10% eventually committing suicide (Lieb et al., 2004).

Currently, there are few efficacious treatments for BPD, with the most effective treatments (e.g., Dialectical Behavioral Therapy) providing only moderate symptom relief (Lieb et al., 2004). Identification of genetic correlates of BPD would improve the prospects of targeted interventions for more homogeneous subsets of borderline patients characterized by specific genetic vulnerabilities. However, despite the prevalence and severity of BPD, its genetic underpinnings have only recently begun to receive significant attention (New et al., 1998; New and Siever, 2003; Ni et al., 2006, 2007; Pascual et al., 2007; Schmahl et al., 2002; Zetzsche et al., 2008) and are hampered by the heterogeneity and complexity of the BPD diagnosis, which is based on symptom-clustering, not underlying neurobiology (APA, 1994). This has led to a call for alternative methodological approaches including identification of endophenotypes (Siever et al., 2002).

Endophenotypes are measurable characteristics associated with a phenotype (e.g., a disease or disorder) that that are more closely related to an underlying genotype than the phenotype itself (Gottesman and Gould, 2003). Though the term “endophenotype” was originally limited to phenotypes that were not visible (e.g., microscopic or biochemical), it has been expanded to refer to any measurable phenotype below the level of a diagnosis. The underlying beliefs are; (a) the endophenotype lies along a developmental pathway between genes and disorder, and (b) because the endophenotype is less complex than the disorder phenotype, it will be dependent on fewer genes and thus more amenable to genetic analysis. This approach of searching for endophenotypes is being employed with some success for a number of severe mental illnesses including schizophrenia (Greenwood et al., 2007; Hong et al., 2008; Leppanen et al., 2008; Smesny et al., 2007) and bipolar disorder (Savitz et al., 2005).

Several criteria must be met for a biological marker to classify as an endophenotype. First and foremost, it should be associated with the illness in the relevant population, occurring at a higher level among individuals with the disorder than among the general population. Ideally, this would include having the putative endophenotype differentiate between the disorder of interest (in our case BPD) and other psychiatric disorders. The endophenotype should also be largely state independent, occurring during both active and residual phases of the disorder (though it may require challenge or provocation). Furthermore, the putative endophenotype should be heritable (Gottesman and Gould, 2003).

Measures of aggression and impulsivity may serve as endophenotypes for BPD as both traits vary within the general population and are strongly associated with BPD. Patients with BPD report more anger, aggression and impulsivity than healthy volunteer or Axis I control subjects (Gardner and Cowdry, 1986; Jacob et al., 2007; Snyder and Pitt, 1985). In fact, impulsive behavior, anger dyscontrol/aggression and self-aggression (i.e., self-mutilation and suicidal behavior) represent three of the nine BPD criteria (APA, 1994). Furthermore, results of twin and family studies have shown impulsivity and aggression to be partially

heritable (Coccaro et al., 1993; Dougherty et al., 2003; Hines and Saudino, 2004; Pedersen et al., 1988; Seroczynski et al., 1999). Impulsivity and aggression are also associated with underlying biological deficits including serotonergic dysregulation and functional impairment of frontal-limbic circuits (Brown et al., 1982; Coccaro et al., 1997a; Haberstick et al., 2006; Manuck et al., 1999) that are also present in BPD (Coccaro et al., 2007; Donegan et al., 2003; McCloskey et al., 2005; Minzenberg et al., 2007; New et al., 2004).

Behavioral (laboratory) measures of aggression and impulsivity are promising candidates to evaluate as potential endophenotypes for BPD as they obtain an observable sample of the behavior of interest, albeit under an experimenter controlled context (Siever et al., 2002). The point subtraction aggression program (PSAP) is one of the few validated laboratory measures of aggression (Cherek et al., 2003). In the PSAP, subjects are provoked by having money indirectly taken from them by a (unbeknownst to the subject) fictitious opponent during a money acquisition task (Cherek et al., 1992). The PSAP has been shown to discriminate between violent and non-violent groups, including criminals and drug abusers (Allen et al., 1997; Cherek et al., 2000, 1997). Furthermore, preliminary data from the authors suggest that aggressive responding on the PSAP is partially heritable. Support for the PSAP as an endophenotype for BPD comes from a finding that a group of 14 hospitalized female BPD subjects were more aggressive on the PSAP than a comparison group of 17 healthy controls (Dougherty et al., 1999).

Impulsivity is a multifaceted construct that can include concepts as varied as sensation-seeking, lack of planning, lack of persistence, inability to delay gratification, insensitivity to delayed consequences, alteration in the perception of time, urgency, and risk taking (de Wit et al., 2002; Reynolds et al., 2006; Smith et al., 2007). However, most major theories of impulsivity include dimensions of motor impulsivity (the inability to delay or inhibit a proponent motor response) and cognitive impulsivity (impulsive decision making such as the inability to shift sets or delay gratification despite negative or less than optimal consequences) (Reynolds et al., 2006; Winstanley et al., 2004). Behavioral measures of both motor impulsivity (e.g., the Immediate Memory Task in which you have to inhibit a prepotent motor response) as well as cognitive impulsivity (e.g., the Passive Avoidance Task in which subjects have to discriminate numbers associated with monetary reward from those associated with monetary loss) are shown to discriminate between impulsive and non-impulsive groups including adults with attention deficit hyperactivity disorder (Malloy-Diniz et al., 2007), bipolar disorder (Christodoulou et al., 2006), substance abusers (Hanson et al., 2008) and pathological gamblers (Forbush et al., 2008). Behavioral measures of cognitive and motor impulsivity also appear to discriminate individuals with BPD from healthy controls. (Chapman et al., 2008; Dougherty et al., 1999; Grootens et al., 2008; Hochhausen et al., 2002; Rentrop et al., 2008), though negative findings for motor impulsivity have also been found (Lampe et al., 2007). Finally, there is evidence of familial transmission for commission errors on the Immediate Memory Task, supporting the heritability of this task (Dougherty et al., 2003).

As stated, optimally an endophenotype would discriminate BPD from other psychiatric diagnoses as well. However, impulsivity and aggression are not specific to BPD. Other Axis I (e.g., intermittent explosive disorder) and Axis II (e.g., paranoid personality disorder) disorders are associated with increased levels of impulsivity and aggression, even when not co-morbid with BPD (McCloskey et al., 2008; Moeller et al., 1997). Furthermore, impulsivity and aggression are not pathognomonic for BPD, with other traits (i.e., emotion regulation) also central to the disorder. For other disorders (e.g., schizophrenia) investigators have had considerable difficulty finding endophenotypes that discriminate them from different psychiatric disorders (Burdick et al., 2006). If behavioral measures of impulsivity and aggression are found not to be specific for BPD, they may still represent an

endophenotype for aggression or impulsivity within BPD, which would have considerable utility for case control and gene mapping studies of BPD. The possibility of identifying endophenotypes that reflect key dimensions of the disorder would provide an opportunity to potentially identify and study the underlying genetics of that dimension, e.g. aggression and its contribution to the genetics of BPD. The utility of behavioral tasks as endophenotypes of impulsivity and aggression in BPD would be supported if these tasks were found to be associated with the underlying construct among individuals with BPD. In other words, if behavioral and self-report trait measures of aggression or impulsivity were correlated among subjects with BPD.

Previous research suggests that behavioral measures of impulsivity and aggression are associated with BPD; however, these studies have been limited in that they have generally used relatively small sample sizes and low level control groups (e.g., healthy volunteers) without controlling for other psychopathology. The current study evaluated behavioral measures of impulsivity and aggression as candidate endophenotypes for BPD in a large sample of individuals with varying levels of psychopathology. Specifically, individuals with BPD were compared to two control groups, a psychopathology free group that does not control for other psychopathology (healthy volunteers) and a second comparison group that controls for the presence of Axis II psychopathology. This latter control group consists of individuals with a non cluster B personality disorders (i.e., personality disorders other than borderline, antisocial, histrionic and narcissistic). All participants completed self-report measures of anger, aggression, and impulsivity and then completed behavioral measures of motor impulsivity (the Immediate Memory Task), cognitive impulsivity (the Passive Avoidance Task and the Bechara Gambling Task) and aggression (the Point Subtraction Aggression Paradigm).

We hypothesized that subjects with BPD would show greater impulsivity and affective aggression on behavioral measures than either comparison group. We also hypothesized that patients with BPD would endorse higher levels of anger, aggression and impulsivity on self-report measures than either comparison group. Finally, we hypothesized that behavioral and self-report measures of aggression/impulsivity would be associated with each other among subjects with BPD.

2. Method

2.1. Participants

Participants consisted of 180 men and 181 women (age $M = 34.87$; $SD = 10.51$) recruited via advertisements for healthy volunteers and individuals with emotional problems at The University of Chicago ($N = 205$) and The Mount Sinai School of Medicine ($N = 156$) as a part of a study on endophenotypes of BPD. The majority of subjects were Caucasian ($N = 200$) and African American ($N = 87$). Participants were excluded if they had a lifetime diagnosis of psychosis or bipolar disorder, organic brain syndrome, mental retardation, or a current diagnosis of substance dependence or major depressive disorder. Additional exclusion criteria included current suicidal or homicidal ideation, or current use of psychotropic medication. This study was carried out in accordance with the latest version of the Declaration of Helsinki. The University of Chicago and Mount Sinai School of Medicine Institutional Review Boards approved the protocol. All participants provided written informed consent prior to enrollment in the study.

The 361 participants were categorized into three diagnostic groups. Healthy volunteers [HV, $N = 112$] denied any lifetime Axis I or Axis II psychopathology. Individuals with borderline personality disorder [BPD, $N = 127$] met DSM-IV criteria for BPD. Finally individuals in the other personality disorder [OPD, $N = 122$] group did not meet criteria for a cluster B

personality disorder but did meet criteria for another DSM personality disorder. Group assignment was based on the results of a psychiatric interview. Not all subjects completed all measures (see data analysis).

2.2. Psychiatric interview measures

Structured Clinical Interview for the DSM-IV [SCID] (First et al., 1996)—The SCID is a semi-structured clinical interview used to assign diagnoses for mood disorders, psychotic disorders, substance abuse and dependence, anxiety disorders, somatoform disorders, eating disorders, and adjustment disorders. The SCID has adequate inter-rater reliability with kappa values for modules reported to be between .70 and 1.00 (First et al., 1996).

Structured Interview for DSM-IV Personality [SIDP] (Pfohl et al., 1995)—The SIDP was employed to diagnose DSM-IV personality disorders. Estimates of inter-rater reliability for the SIDP are reported to be adequate to strong with intraclass correlation coefficients as high as .88–.99 (Damen et al., 2004). In our laboratories the intraclass correlations for BPD were .83 (Chicago) and .80 (New York).

2.3. Behavioral measures

Point-Subtraction Aggression Paradigm [PSAP] (Cherek, 1992)—Aggressive behavior was assessed using the PSAP, a laboratory aggression measure in which subjects have the option to press buttons that will either accumulate points exchangeable for money (A button), take points away from an opponent (B button) or protect their points (C button). Proportion of B responses to total responses (PSAP B-Ratio) is considered an index of aggressive behavior. Evidence supporting the validity of the PSAP comes from several sources. Studies using the PSAP have demonstrated the facilitation of aggression by drugs associated with violence in non-experimental studies (Kelly and Cherek, 1993). The paradigm also discriminates groups of participants theoretically expected to exhibit elevated levels of aggression such as violent criminals (Cherek et al., 1997). Responding is elicited by provocation, and is reduced when participants are informed that they are interacting with a computer rather than another individual (Kelly and Cherek, 1993).

Participants performing the PSAP were led to a cubicle and seated at a desk containing a computer monitor and response panel. The participant was informed that the task involved interacting with other people (who remained unidentified) located at similar computer terminals elsewhere in the building. Three buttons, labeled A, B, and C, were located in a row across the response panel. Participants were told that they could accumulate points (displayed on the computer monitor) by pressing button A, which would be exchanged for money at the end of the day. This non-aggressive point acquisition response option was maintained on a fixed ratio 100 (FR100) schedule. That is, participants were told that 100 consecutive responses earned one point. Participants were informed that pressing B ten times (FR10) would subtract one point from other participants, but would not increase the participants own point total. Participants were therefore led to believe that an aversive stimulus would be delivered to another person (i.e., subtraction of one point) for every 10 presses of Button B. Finally, the participants were informed that pushing C button 10 times (FR10) would protect the participant points for a period of time, thus presenting a non-aggressive protective option. In actuality, all points subtracted by the fictitious individual were controlled by a computer program. A running total was displayed at the top of the monitor, which allowed the participant to know how much money they had accumulated. To ensure that participants recognized when money was subtracted from their total, the running total was enlarged and flashed on the screen when money was being subtracted.

Each participant completed a single 25 min PSAP session with a high level of provocation (i.e., the longest period of time a participant could be protected from having a point subtracted was 62.5 s). As stated, aggression in the PSAP is defined of the proportion of total button presses that were aggressive (PSAP B-Ratio = B button presses/A + B + C button presses). Subjects received all money won on the PSAP at the end of the study.

2.3.1. Immediate Memory Task [IMT] (Dougherty and Marsh, 2003b)—The IMT is a modified Continuous Performance Task that measures response initiation (a.k.a. motor) impulsivity. During the IMT subjects are shown a series of briefly presented 5-digit numbers on a computer monitor. The sequence of numbers is randomly generated and each number appears for 500 ms at a rate of one per second. Subjects are instructed to click a mouse button when the 5-digit number they see is identical to the one that preceded it. On one-third of the trials, the stimulus is a number that differs from the preceding number by only one digit (its position and value determined randomly). Responses to catch stimuli are recorded as commission errors, which are believed to reflect motor impulsivity in this task. The remainder of the trials are equally divided between target (number matches preceding number) and filler stimuli. A target stimulus is a 5-digit number that is identical to the preceding number. The participant is instructed to respond to these numbers and these responses are recorded as correct detections. A filler stimulus is a random 5-digit number that appears whenever a target or catch trial is not scheduled to appear. The proportion of commission errors to correct detections, known as the IMT Ratio is the primary dependent measure of impulsivity for this task (Dougherty et al., 2008).

2.3.2. Bechara Gambling Task [BGT] (Bechara et al., 1994)—The BGT is a computerized version of the original game developed by Bechara and colleagues (Bechara et al., 1994, 1997). In this task, the participants sat in front of a computer screen and were presented with four decks of cards—A, B, C, and D—displayed face down. Participants were instructed to turn over cards from the decks (by clicking on the computer mouse) to maximize gain over time. As each card was turned over, the computer provided feedback with regard to the net gain or loss associated with that selection. Decks A and B were designed to produce higher rewards (average reward = $125), but at unpredictable points these decks also produced even higher losses (average loss = $187.50). Thus overall selection from decks A and B was disadvantageous and resulted in net losses. Decks C and D provided relatively smaller rewards (average reward = $62.50) but also had even smaller losses (average loss = $31.25). Selection from these decks resulted in advantageous net gains over time. The frequency of a loss was comparable for the disadvantageous (loss f(A) + loss f(B)/2) and the advantageous decks (loss f(C) + loss f(D)/2). Participants made 100 deck selections during the task, and the primary impulsivity outcome measure on the BGT was the number of disadvantageous deck selections (decks A or B) that occurred within each 25-trial block (4 blocks).

2.3.3. Passive Avoidance Learning Task [PAT] (Hochhausen et al., 2002)—Participants completed a computerized Passive Avoidance Learning Task (PAT) in which participants are repeatedly shown 10 different 2-digit numbers (e.g. 23). Five of the 2-digit numbers served as winning numbers and the other five 2-digit number served as losing numbers. Participants learn to respond (hit spacebar) to positive discriminative stimuli (S+) in order to receive a monetary reward ($ 0.10) and inhibit responding to the negative discriminative stimuli (S−) in order to avoid monetary punishment ($ −0.10). No money was won or lost when participant did not press the spacebar. Participants were instructed to use trial-and-error to learn when hitting the spacebar would result in monetary reward or loss. Subjects were given five learning trials in which the five S+ numbers were presented. This was followed by 90 trials in which S+ and S− numbers were pseudorandomly

presented (9 trials per 2-digit combination). The proportion of responses to S− stimuli to S+ stimuli, known as the PAT Ratio is the primary dependent measure of impulsivity for this task.

Participants began the experiment with 10 chips, worth 10 cents each. After pressing the button in response to a winning number, participants received a plastic chip from the experimenter and the computer monitor read, “You WIN 10 cents!,” while emitting a high pitched tone. After a losing response, the experimenter removed one chip and the computer read, “You LOSE 10 cents”, while emitting a low-pitched tone. No feedback occurred in the absence of a response (spacebar press). An experimenter, unaware of participant diagnosis, sat next to participants to dispense and remove chips. Subjects received the amount of money won in the task at the end of the experiment.

2.4. Self-report scales

2.4.1. Life history of aggression [LHA] (Coccaro et al., 1997b)—The LHA is a semi-structured interview that assesses total number of aggressive (five items), antisocial (four items), and self-aggressive (two items) acts engaged in since adolescence. The LHA has adequate to strong reliability and has demonstrated construct and discriminant validity (Coccaro et al., 1997b).

2.4.2. Buss–Perry aggression questionnaire [BPAQ] (Buss and Perry, 1992)—The BPAQ is a self-report measure of trait aggressiveness. The BPAQ consists of 29 items each scored using a 4-point Likert-type scale. The BPAQ consists of four scales: physical aggressiveness, verbal aggressiveness, anger, and hostility (i.e., suspiciousness and resentment). The BPAQ has well-known psychometric properties (Buss and Perry, 1992).

2.4.3. State-trait anger expression inventory [STAXI] (Spielberger, 1996)—The STAXI consists of 44 items that form six scales: State Anger, Trait Anger, Anger-In, Anger-Out, Anger Control, and Anger Expression. In responding to each of the STAXI 44 items, individuals rate themselves on 4-point scales that assess either the intensity of their angry feelings or the frequency that anger is experienced, expressed, suppressed, or controlled. The STAXI has been shown to be a valid and internally consistent measure of anger and anger expression (Spielberger, 1996).

2.4.4. Barratt impulsivity scales – 11 [BIS] (Patton et al., 1995)—The BIS-11 is a 34-item questionnaire that assess motoric (acting without thinking), cognitive (hasty decisions) and nonplanning (failure to plan ahead) impulsiveness. Each item is rated on a 4-point scale ranging from “Rarely/Never” to “Almost always/Always.” The BIS-11 is an internally consistent (= .79–.83), valid measure of impulsiveness (Patton et al., 1995).

2.4.5. Life history of impulsive behaviors self-report [LHIB] (Schmidt, 2000)—The LHIB is a 53-item questionnaire designed to assess lifetime history of impulsive behavior (as opposed to impulsive tendencies) as well as the level of distress and impairment associated with these behaviors. The LHIB consists of scales for clinically significant impulsivity, non-clinically significant impulsivity, and impulsivity related distress/impairment. In the current study the LHIB clinically significant impulsivity and impulsivity related distress/impairment scales were multicollinear ($r > .90$), therefore the impulsivity related distress/impairment scale was omitted from subsequent analyses. The LHIB has excellent internal validity (= .96) and test-retest reliability ($r > .87$) (Schmidt, 2000). LHIB scores correlate significantly with other measures of impulsivity and discriminate subjects with impulse control problems from those without such problems (Schmidt, 2000).

2.5. Procedure

Participants completed a 3–4 h diagnostic interview conducted by trained graduate-level diagnosticians who were blind to the study hypotheses. Presence of BPD and other personality disorders were assessed using the SIDP. Axis I diagnoses were assigned using the SCID. The LHA was administered to assess the frequency of aggressive, self-aggressive and antisocial acts. Diagnoses were confirmed using a "best estimate procedure" in which the diagnostic report was reviewed by a committee of psychiatrists, psychologists, and/or expert diagnosticians (Klein et al., 1994). Between visits 1 and 2, participants completed a booklet containing the BPAQ and STAXI. For visit 2, participants completed a urine drug test and (in Chicago) alcohol breathalyzer test to ensure they were not under the immediate influence of alcohol or other drugs of abuse. Participants then completed behavioral measures of aggression (PSAP) and impulsivity (IMT, BGT, PAT) that were counterbalanced across subjects. After the PSAP, participants completed a short self-report questionnaire to confirm that the participants were attending to the task, and believed they were interacting with other individuals. At the end of the study, subjects were debriefed. Examination of responses to the PSAP self-report questionnaire and debriefing responses revealed that all participants appeared to accept the social conditions of the task; that is, that they were interacting with another individual and that the purpose of the task was to accumulate points exchangeable for money.

2.6. Data analysis

Analyses were conducted 2-tailed at the .05 level of significance. Significant interactions were analyzed using simple effects. Post-hoc mean comparisons were performed using Tukey's HSD test ($p < .05$) for between-subjects effects and Bonferroni corrected pairwise comparisons for within subjects effects. Effect sizes are provided using partial eta squared ($\acute{\eta}_p^2$) for analyses of variance. For $\acute{\eta}_p^2$ .01, .06 and .14 are considered small, medium and large effect sizes, respectively (Cohen, 1988). Initial analyses showed that participants from the Chicago and New York locations did not differ from each other with respect to demographic variables or general psychopathology (all $p > .10$), therefore the two locations were combined. Age, education and Axis I psychopathology were found to vary across diagnostic groups and were therefore included as covariates in the primary analyses. Three measures (Passive Avoidance Task, Immediate Memory Task and the Self-Aggression Scale of the Life History of Aggression) were significantly skewed (skew statistic > 2). Analysis of transformed data did not change the pattern of results; we therefore used the raw data for ease of interpretability. Correlations were used to examine associations between self-report and behavioral measures across the entire sample and within diagnostic groups. Fisher's Z transformations were used to compare the strength of correlations across diagnostic groups.

All subjects completed the PSAP. Software problems resulted in the loss of IMT and BGT data for 30 and 28 subjects, respectively. Of the 361 subjects, 323 correctly completed the BPAQ and 328 correctly completed the BIS. The PAT, LHA, STAXI and LHIB were not initially a part of the protocol at both sites. Consequently, PAT, LHA, STAXI and LHIB data was collected from only 240, 261, 276 and 272 subjects, respectively. Individual Group *N*s are provided each measure of aggression and impulsivity in Table 3 and Table 4.

3. Results

3.1. Preliminary analyses

3.1.1. Demographic variables—The groups differed with regard to age, F (2,358) = 6.26, $p = .002$. Post-hoc analyses showed that the HV group was younger than both the BPD and OPD groups who did not differ from each other. The group also differed with respect to

Education, F (2,358) = 19.51, p = .001. Post-hoc analyses showed that the HV group had more education than the OPD and BPD groups, which did not significantly different from each other (p > .10). Neither race [χ^2 (6,N = 358) = 9.74, p = .14], nor gender [χ^2 (2, N = 361) = 1.51, p = .47] was significantly different across groups (see Table 1 for demographic variable means and percentages). Because age and education varied across diagnostic groups they were included as covariates in the primary analyses.

3.1.2. Psychopathology—As evidenced on (Table 2), BPD subjects were more likely to have a lifetime Axis I disorder than their OPD counterparts. This included an increased prevalence of lifetime major depressive disorder (MDD), posttraumatic stress disorder (PTSD), Non-PTSD anxiety disorder, alcohol dependence, and non-alcohol substance dependence. The only group of disorders studied in which the BPD and OPD groups did not differ was Non-MDD Mood Disorder. Overall, BPD subjects had almost twice the number of Axis I disorders (M = 2.68, SD = 1.85) as OPD subjects (M = 1.38, SD = 1.29), a difference that was highly significant, t (247) = 6.15, p < .001. Therefore, number of Axis I disorders was included as a covariate in the primary analyses.

BPD subjects had a higher incidence of cluster B personality disorders than the OPD group by virtue of the presence of a cluster B disorder being exclusionary criteria for the OPD group. BPD subjects were also more likely to have co-morbid Paranoid personality disorder, while the OPD group was more likely to have personality disorder not otherwise specified (PD NOS), and to a lesser extent schizoid PD. PD NOS as well as obsessive-compulsive personality disorder represented the most common forms of Axis II psychopathology in the OPD group, with each occurring is approximately one-third of OPD subjects.

3.2. Primary aggression analyses (see Table 3 for group mean [SD], F-ratio and effect sizes)

3.2.1. Behavioral aggression (PSAP)—A 3 (group) × 2 (gender) Multivariate Analysis of Covariance (MANCOVA) on percentage of monetary (“A” button), aggressive (“B” button), and defensive (“C” button) responses revealed a significant Multivariate effect of group, Wilks F (4,702) = 5.11, p < .001. Subsequent ANOVAs revealed a group effect for both monetary and aggressive button selections. Post-hoc analyses showed that both BPD and OPD subjects made a higher proportion of aggressive (“B”) button selections and lower proportion monetary (“A”) button selections than HV subjects. BPD and OPD groups did not differ in their C button response tendencies (p > .10). There was no significant multivariate effect of gender or gender × group (both Wilks F < 1).

Trait Aggression (BPAQ): A 3 (Group) × 2 (Gender) MANCOVA on the BPAQ scales revealed a significant multivariate effect of diagnostic group, Wilks F (8,622) = 18.55, p < .001. Subsequent univariate analyses revealed a significant effect of group for all four BPAQ scales. Post-hoc analyses revealed a pattern of BPD > OPD > HV for each scale of the BPAQ. There was also a significant multivariate effect of gender [Wilks F (4,311) = 5.65, p < .001)]. This stemmed from a significant main effect of gender on the physical aggression scale [F (1,323) = 12.44, p < .001, $\acute{\eta}_p^2$=.04] with men reporting more trait physical aggression (M_{adj} = 21.37 SD = 7.39) than women (M_{adj} = 18.82, SD = 7.08). There was no multivariate group × gender interaction (Wilks F < 1).

3.2.2. Aggressive history (LHA)—A 3 (group) × 2 (gender) MANCOVA on the LHA aggression, self-aggression, and antisocial scales revealed multivariate effects of group [Wilks F (6,500) = 19.03, p < .001], gender [Wilks F (3,250) = 5.86, p < .001] and their interaction [Wilks F (6,500) = 2.42, p < .05]. Univariate analysis of the aggression scale indicated a main effect of group. BPD subjects reported more aggression than OPD subjects,

who in turn reported more aggression than HV subjects. A 3 × 2 ANOVA on the self-aggression scale revealed a significant main effect of group that was limited by a significant gender × group interaction, F (2,252) = 5.94, $p < .005$, $\eta_p^2 = .05$. Simple effects revealed a main effect of group on self-aggression for women [F (2,252) = 10.52, $p < .001$] but not for men [F (2,252) = 2.19, $p = .11$]. Post-hoc Tukey tests showed that women in the BPD group (M_{adj} = 1.66, SD = 2.43) were more self-aggressive than women in the OPD (M_{adj} = 0.39, SD = 0.97) or HV (M_{adj} = 0.00, SD = 0.00) groups. For the antisocial scale, there was a significant effect of group in which BPD subjects reported more antisocial behavior than OPD subjects who in turn reported more antisocial behavior than HV subjects. There was also a significant effect of gender [F (2,252) = 12.62, $p < .001$, $\eta_p^2 = .05$] with men endorsing more lifetime antisocial behavior (M_{adj} = 5.05, SD = 4.89) than women (M_{adj} = 3.16, SD = 3.49).

Anger Expression (STAXI): A 3 (group) × 2 (gender) MANCOVA on the STAXI state, trait, anger-in, anger-out, anger control and anger expression scales revealed multivariate effect of group [Wilks F (10,526) = 11.51, $p < .001$]. Subsequent univariate analyses revealed a significant effect of group for all six STAXI scales. Post-hoc analyses showed that BPD and OPD groups each reported more state anger and less anger control than HV subjects, but did not differ from each other. For the other four STAXI scales (trait, anger in, anger out, anger expression), BPD subjects scored significantly higher than OPD subjects who in turn scored higher than HV subjects. No other multivariate effects were significant (Wilks $F < 1$).

3.3. Primary impulsivity analyses (see Table 4 for group mean [SD], F-ratio and effect sizes)

3.3.1. IMT—A 3 (group) × 2 (gender) ANCOVA of IMT Ratio revealed a main effect of group. Post hoc analyses showed that BPD and OPD did not differ from each other, but both groups were more impulsive on the IMT than HV subjects. There was no main effect of gender, F (2,322) = 1.96, $p = .16$. Nor was there a significant group × gender interaction ($F < 1$).

3.3.2. PAT—A 3 (group) × 2 (gender) ANCOVA of PAT Ratio failed to show a main effect of group ($F < 1$), gender [F (2,231) = 2.53, $p = .12$] or their interaction ($F = 1$).

3.3.3. BGT—A 3 (group) × 2 (gender) × 4 (block) mixed design ANCOVA on disadvantageous BGT deck selections failed to reveal a significant effect of block, group or gender (all $F < 1$) or their interactions (all $p > .32$) Exploratory 3 (group) × 2 (gender) ANCOVA's at each block confirmed the lack of a group effect at any block (see Table 4).

BIS: A 3 (group) × 2 (gender) MANCOVA on the BIS motor, cognitive, and nonplanning scales revealed multivariate effects of group [Wilks F (6,634) = 17.12, $p < .001$], and gender [Wilks F (3,317) = 2.66, $p < .05$]. Univariate analysis revealed a significant effect of group for all three BIS scales. Post-hoc analyses revealed an identical significance pattern of BPD > OPD > HV for each BIS impulsivity scale. Univariate analyses also revealed a significant effect of gender for BIS nonplanning [F (1,328) = 5.16, $p < .05$, $\eta_p^2 = .02$], with men (M_{adj} = 26.71, SD = 5.79) reporting greater nonplanning impulsivity than women (M_{adj} = 25.41, SD = 5.71). Men and women did not differ in terms of motor or cognitive impulsivity (both $F < 1$). There was no multivariate gender × group interaction [Wilks F (6,634) = 1.79, $p > .10$].

3.3.4. LHIB—A 3 (group) × 2 (gender) MANCOVA on the two LHIB scales revealed multivariate effects of group [Wilks F (4,524) = 18.47, $p < .001$]. Univariate analysis

revealed a significant effect of group for LHIB clinically significant impulsivity ($p < .001$), but not the non-clinically significant impulsivity scale ($p > .05$). Post-hoc analyses showed that BPD subjects reported more clinically significant impulsivity than OPD subjects who in turn reported more than HV subjects. There was a nonsignificant multivariate trend for gender [Wilks F (2,262) = 2.77, $p = .07$], but neither LHIB scale showed a significant univariate gender effect (both $p > .16$). There was no multivariate gender × group interaction [Wilks F (4,524) = 1.35, $p = .25$].

3.4. Correlational analyses

Aggression Measures—Aggressive (B button) responses on the PSAP were associated with the majority of self-report aggression measures, with only LHA self-aggression and STAXI state anger clearly unrelated to PSAP responding ($p > .10$). However, the magnitude of the significant correlations were modest, with no correlation greater than $r = .20$. Self-report aggression measures were highly correlated with each other, with correlations between some STAXI scales approaching multicollinearity. The nonsignificant relationship among the self-report measures was between LHA self-aggression and state anger on the STAXI (see Table 5).

Impulsivity Measures—As (Table 6) shows, behavioral impulsivity measures were not correlated with each other. In contrast, most of the self-report impulsivity scales were strongly ($p < .001$) correlated with each other. The only self-report impulsivity scale that did not correlate with the other self-report measures was the LHIB non-clinically significant impulsivity scale which was negatively correlated with LHIB clinically significant impulsivity and unrelated to the BIS. Overall, the behavioral and self-report impulsivity measures were moderately correlated. The PAT was unrelated to any of the self-report impulsivity measures. However, the IMT correlated with all three BIS scales; while the BGT was correlated with BIS motor and LHIB clinical impulsivity scales.

3.5. Behavioral – self-report association by diagnostic group

To evaluate the ability of behavioral measures to serve as proxies for trait aggression and impulsivity within each diagnostic group, a series of correlations were performed for each diagnostic group comparing (a) self-report measures of anger/aggression and PSAP aggressive responding, and (b) behavioral and self-report measures of impulsivity.

3.5.1. Aggression—Comparisons of the relationship between trait aggression (BPAQ total) and PSAP aggressive responding for each of the three diagnostic groups showed that the two measures were associated for BPD subjects ($r = .22$, $p = .02$), but not for either OPD ($r = .05$, $p = .55$) or HV ($r = -.04$, $p = .68$) subjects. Comparison of correlation strength using fishers Z transformations showed the relationship between PSAP aggressive responding and BPAQ total to be significantly stronger for BPD subjects relative to HV subjects ($p < .05$), but not relative to OPD subjects ($p = .21$). The n's for specific personality disorders are too small to analyze definitively. However, exploratory analysis within the OPD group showed a great deal of variability in the PSAP–BPAQ relationship across specific personality disorders, raging from high nonsignificant positive correlations among subjects with Schizoid ($n = 5$, $r = .45$) personality disorder to high nonsignificant negative correlations among subjects with Dependent personality disorder ($n = 3$, $r = -.85$), For the remaining personality disorders in the OPD group, the correlation between BPAQ and PSAP aggressive responses was modest to weak ($r = -.15$ to .15).

Analysis of specific BPAQ scales (Table 7) showed that PSAP aggressive responding associated with both physical aggression and anger scales in BPD, but not OPD or HV subjects. For BPD subjects PSAP aggression was also correlated at a trend level with history

of aggressive behavior (LHA aggression, $p < .08$) and trait anger on the STAXI ($p = .10$). HV subjects only showed a trend association with LHA aggression ($p < .07$). OPD subjects did not show any associations between self-report aggression measures and aggressive responding on the PSAP (all $p > .10$).

Comparison of correlations across groups showed that PSAP aggressive responding was more strongly associated with BPAQ anger in BPD subjects than either HV ($p < .05$) or at a trend level ($p < .09$) OPD subjects. The relationship between PSAP aggression and STAXI trait anger also showed a nonsignificant trend towards being stronger for BPD relative to OPD subjects ($p < .09$). Finally, PSAP aggression was more strongly correlated with BPAQ physical aggression for BPD subjects relative to HV subjects. Overall, these results suggest PSAP responding is moderately associated with aggression in BPD subjects but less so for subjects in the OPD and HV groups.

Impulsivity: Among BPD subjects the BGT was significantly associated with self-reported impulsivity, correlating with BIS motor impulsivity (see Table 8). BPD subjects also showed a nonsignificant trend for IMT Ratio to be correlated with BIS motor ($p < .06$) and nonplanning ($p < .07$) scales as well as for PAT ratio to be associated with BIS nonplanning ($p < .08$). For subjects in the OPD group the only significant correlation was between the BGT and LHIB clinically significant impulsivity. There were no significant correlations between behavioral and self-report impulsivity scales among HV subjects, with one correlation (BGT – BIS motor scale) approaching significance, but in the opposite direction from what would be hypothesized.

Comparison of correlation strength across groups showed that relative to HV (but not OPD) subjects, BPD subjects had significantly stronger positive correlations between the IMT and BIS motor impulsivity, between the PAT and BIS nonplanning, and between BGT disadvantageous deck selections and BIS motor impulsivity. The OPD group showed a stronger positive correlation between the BGT and LHIB clinically significant impulsivity than in either the HV or BPD group. Overall, these results suggest that behavioral impulsivity measures are modestly related to self-reported impulsivity among BPD and to a lesser extent OPD groups, but not HV groups.

4. Discussion

The purpose of this study was to evaluate behavioral measures of impulsivity and aggression as potential endophenotypes for borderline personality disorder (BPD). Individuals with BPD did not differ from either the Axis II control group (OPD) or the healthy volunteer group (HV) on behavioral measures of cognitive impulsivity (PAT and BGT). The BPD group did show more motor impulsivity (IMT) and affective aggression (PSAP) in comparison to HV subjects (even after controlling for Axis I comorbidity within BPD), but not in comparison to the OPD group. In contrast, self-report measures of both aggression and impulsivity did, for the most part, discriminate BPD from both comparison groups. Our results suggest that the behavioral measures of impulsivity/aggression were not uniquely associated with BPD, but in the case of the PSAP and IMT, were associated with the illness, thus providing partial support for these measures as a potential endophenotype (Gottesman and Gould, 2003). There was more support for the PSAP as an endophenotype for aggression within BPD samples, as the PSAP was related to self-reported aggression in the BPD group.

BPD subjects made a greater proportion of aggressive responses on the PSAP than HV subjects, even after controlling for Axis I psychopathology. This extends previous research (Dougherty et al., 1999) and provides partial support for the PSAP as an endophenotype for

BPD. However, this is limited by the finding that BPD subjects did not differ from their OPD counterparts in their level of PSAP aggression. The first and most obvious reason for this is that behavioral aggression is not sufficiently unique to BPD. Irritability and aggressiveness is a symptom of antisocial personality disorder (APA, 1994), and several other personality disorders (e.g., narcissistic, paranoid and obsessive–compulsive personality disorder) are also associated with an increased risk of aggressive behavior (Berman et al., 1998; Goldberg et al., 2007; Kim et al., 2008; Villemarette-Pittman et al., 2004). However, most of this research relied on self-report aggression measures. With the exception of antisocial personality disorder (Moeller et al., 1997), it is not clear which personality disorders other than BPD are associated with increased aggression on a behavioral task.

It is also possible that the aggression task used did not provide the correct context to distinguish individuals with BPD from those with other disorders. Most of the aggressive behavior exhibited by individuals with BPD occurs within the context of relationships with friends, associates and loved ones – relationships where a perceived insult or attack would be particularly threatening. Currently, all laboratory aggression tasks are based on an interaction with a stranger. If the PSAP cover task was modified to simulate aggression from an existing relationship, then groups differences between BPD and other PD's may emerge. Provocation intensity and/or frequency may also have obscured potential differences between BPD and OPD groups. The current aggression task used a low intensity aggressive behavior (taking a small amount of money from an opponent). Lower intensity aggressive acts such as arguing or breaking something inconsequential are more ubiquitous and under less genetic control than more severe acts such as physical assault (Coccaro et al., 1997a). It is possible that behavioral measures of physical aggression may better differentiate individuals with BPD. Conversely, the frequency of provocation used in the study was very high (the highest that is used on the PSAP). This was done to maximize group differences in responding over a single PSAP session. However, it is possible that this high frequency of provocation masked differences between OPD and BPD groups that may have existed at more moderate provocation frequencies.

Aggressive responding on the PSAP was associated with several self-report anger and aggression scales, supporting the utility of the PSAP as an endophenotype for aggression within BPD samples. In contrast, OPD and HV subjects showed no significant association between PSAP aggression responding and self-report aggression measures (there was a single trend level association between PSAP and LHA-Aggression for HV subjects), putting in question whether the PSAP is a valid measure of aggression for these groups. For HV subjects, a possible explanation is that there was insufficient variance in PSAP B button pressing to demonstrate a correlation with other measures. Supporting this, the HV groups only chose a B response on 3% of the trials and had approximately half the standard deviation of the other groups. The OPD group did show high rates of B button presses, but no relationship with self-reported aggression. The reasons for this are not entirely clear. OPD individuals in general may have poor self-knowledge about their aggression. Alternately, the heterogeneity of the OPD group may have obscured significant relationships between PSAP aggressive responses and self-report measure of anger/aggression for specific personality disorders. Remember, we found a high level of variability for correlations between PSAP and BPAQ among the different PDs in the OPD group. For at least a portion of the OPD group (e.g., those with schizophrenia spectrum personality disorders) PSAP B button presses may have been tapping into another symptom, such as paranoia or other cognitive problems.

BPD subjects demonstrated greater motor impulsivity (IMT) than the HV controls but not the OPD comparison group, providing partial support for the motor impulsivity task as an

endophenotype for BPD. In contrast, BPD subjects were not significantly different from either HV or OPD subjects on behavioral measures of cognitive impulsivity (PAT, BGT). This was surprising in light of clinical evidence that individuals with BPD engage in impulsive decision making as well as previous research showing individuals with BPD are more impulsive on behavioral measures of cognitive impulsivity (Dougherty et al., 1999; Hochhausen et al., 2002). More prototypical behavioral measures of cognitive impulsivity such as Delay Discounting might have shown separation between BPD and comparison groups. However, it is also possible that increased cognitive impulsivity among subjects with BPD may be contextually dependent. Much of the behavior in patients with BPD identified as “impulsive” such as suicide attempts, self-harm and other self-destructive behavior occurs in response to acute negative affect (Brown et al., 2002). In the absence of this negative affect, individuals with BPD may not be significantly more cognitively impulsive than other individuals.

There was modest evidence for behavioral impulsivity measures as an endophenotype for impulsivity within BPD. The IMT was correlated at a trend level with both trait motor and trait nonplanning impulsivity. The PAT was also correlated at a trend level with trait nonplanning impulsivity, while the BGT was significantly correlated with trait motor impulsivity. For OPD and HV groups, the relationship between behavioral and self-reported impulsivity was weaker. There were no significant correlations in the HV group and only one (BGT and LHIB clinically significant impulsivity) in the OPD group. Furthermore, in several cases the relationship between behavioral and trait impulsivity trended towards being higher for the BPD subject as compared to HV subjects. This is consistent with research showing modest, inconsistent relationship between behavioral and self-report impulsivity measures in non-clinical samples (Mitchell, 1999; Reynolds et al., 2006; Richards et al., 1999).

In contrast to the laboratory measures, self-report measures consistently showed BPD subjects to be more impaired than either HV or OPD groups. Among the aggression measures, BPD subjects reported more trait physical aggression, verbal aggression, anger, and hostility on the BPAQ than OPD subjects who in turn reported more than HV subjects. An identical pattern was shown for the LHA aggression scale and four of the six STAXI scales (i.e., trait anger, anger in, anger out and anger expression) These findings support earlier research showing that individuals with a personality disorder tend to be angrier, more irritable and more prone to engage in aggressive behavior than those without any such psychopathology (McCloskey et al., 2006), and that may be particularly true among individuals with BPD (Goodman and New, 2000; Joyce et al., 2003).

Individuals with BPD also reported higher levels of motor, attention and nonplanning impulsivity on the BIS than either OPD or HV subjects. Increased impulsivity was distinctly associated with pathologic activity as BPD subjects reported higher levels of clinically significant impulsivity and antisocial behavior than OPD or HV groups, though the three groups did not differ with respect to non-clinically significant impulsivity. This is consistent with earlier research showing individuals with BPD self-reported higher levels of trait impulsivity (Dougherty et al., 1999; Paris et al., 2004; Wilson et al., 2007), as well as clinical observation that individuals with BPD have difficulty delaying gratification, make decisions quickly, and either devalue or fail to consider the consequences of their actions.

BPD women also reported more self-aggression than OPD and HV women, though BPD and OPD men did not differ from each other. We did not expect to find a gender by group interaction for self-aggression. Overall, non-lethal self-aggression (i.e., suicide attempts and self-mutilation) is equally to more prevalent in women (Briere and Gil, 1998; Zlotnick et al., 1999). Among patients with BPD, a history of self-aggression is also equally likely in either

gender (Grilo et al., 2004; Johnson et al., 2003). One possible explanation for our finding is that, in contrast to the aforementioned studies that assessed only the presence or absence of the self-aggression BPD criteria, the LHA self-aggression scale assessed the number of self-aggressive acts. Thus, history of self-aggression may discriminate BPD from OPD across gender, but frequency of self-aggression does so only for women. To test this we performed an exploratory analysis in we dichotomized the LHA self-aggression data (no self-aggression vs. any self-aggression). We found that presence of any self-aggression discriminated BPD from OPD across both genders in our sample ($p < .01$).

Self-report measures were superior to behavioral task at discriminating BPD from comparisons groups. Despite this, self-report and behavioral measures of aggression/impulsivity were often correlated among BPD subjects, at least at a trend level. It is possible that deficits in emotional awareness (Levine et al., 1997) as well as a dramatic “all or nothing” style of presentation (Rosenthal et al., 2007), led BPD subjects to overestimate their impulsive/aggressive tendencies. Alternately, the pattern of results may suggest aggression and impulsivity is secondary to affective lability (a.k.a. emotional dysregulation) in BPD. Despite the heterogeneity of BPD symptoms, affective lability is believed to be a core dimension of BPD (Lieb et al., 2004; Rosenthal et al., 2007), and is the most prevalent and enduring of the BPD diagnostic criteria (McGlashan et al., 2005). Aggression is typically a response to dysregulated anger and many of the impulsive and aggressive behaviors engaged in by individuals with BPD are associated with emotional distress (APA, 2000). Furthermore, recent studies suggest that emotional state may moderate behavioral impulsivity in BPD (Chapman et al., 2008). Therefore, though individuals with BPD have more trait aggression and impulsivity than comparison groups, actual aggressive or impulsive behavior would primarily occur only when they were experiencing significant negative affect. We know BPD and OPD groups did not differ in their reported level of state anger while completing the STAXI. It is unclear if the behavioral tasks used in this study were able to produce a significant increase in negative affect, as this unfortunately was not assessed. The unique signature behaviors of BPD may emerge from the interaction of emotional dysregulation and interpersonal sensitivity (e.g., as in context of termination of important relationship and ensuing feelings of abandonment) driving impulsive and aggressive behaviors. In this sense, the identification of endophenotypes for dimensions of emotional dysregulation and impulsivity/aggression (discussed here) in BPD would serve to validate the diagnosis but as the focus of this paper is specifically impulsivity and aggression in BPD, we will address these broader issues in a subsequent paper.

The results of this study provide limited evidence for the use of behavioral measures of aggression (PSAP) and motor impulsivity (IMT) as endophenotypes for BPD, and support the use of the PSAP as potential endophenotype of aggression within BPD samples. Strengths of the study include the use of a relatively large medication-free clinical sample, inclusion of a non cluster B personality disorder control group in addition to healthy volunteer group, use of multiple self-report and behavioral measures of impulsivity and aggression, and controlling for differences in Axis I psychopathology across diagnostic groups. However, aspects of the study may limit the generalizability of these results. There was a significant amount of missing behavioral impulsivity data due to late introduction of measures and software problems. This reduced our power to detect potentially significant group effects, though the BPD vs. OPD comparison did not approach significance for any of the behavioral impulsivity measures. Also, the OPD and BPD groups were heterogeneous with regard to Axis II personality disorders, including significant cluster B co-morbidity in the BPD group. However, the additional cluster B co-morbidity in the BPD group would be expected to, if anything, accentuate differences between OPD and BPD groups on behavioral aggression and impulsivity measures.

The behavioral measures of aggression and impulsivity we chose were selected on the basis of extensive past research implicating them in aggression and in some cases to BPD. However, it is possible that there are other measures that would better distinguish BPD from other clinical groups, perhaps by recreating the interpersonal and other contextual factors that would increase the negative affect associated with the task. Behavioral measures of other core components of BPD such as affective lability or interpersonal sensitivity (Lejuez et al., 2003) may also serve as useful endophenotypes of BPD. It is also possible that the heterogeneity and complexity in BPD precludes any one behavioral measure from being a specific endophenotype of the disorder. In this case the need to identify genetically homogenous dimensions of BPD will aid in our understanding and treatment of the disorder, allowing for more targeted interventions, both pharmacological and psychosocial. By identifying endophenotypes for the major dimensions of BPD and their underlying genotypes we may be able to identify genetic/endophenotypic signatures for this disorder. Our study suggests the PSAP may be an effective endophenotype for the central trait or dimension of aggression in BPD. Accordingly, future gene mapping and family studies of BPD would be well served to include such a measure.

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Table 1

Demographic variables as a function of diagnostic group.

Variable	Diagnostic group		
	HV	**OPD**	**BPD**
Age (SD)*,a	32.04 (9.69)	36.59 (10.69)	35.70 (10.61)
Gender (%)			
Male	57 (50.9%)	65 (53.3%)	58 (45.7%)
Female	55 (49.1%)	57 (46.7%)	69 (54.3%)
Race (%)			
Caucasian	59 (54.1%)	77 (63.1%)	64 (50.4%)
AA	24 (22.0%)	30 (24.6%)	33 (26.0%)
Asian	13 (11.9%)	5 (4.1%)	10 (7.9%)
Other	13 (11.9%)	10 (8.2%)	20 (15.7%)
Years education (SD)*,b	16.33 (2.38)	14.99 (2.29)	14.54 (2.17)

Note: Race data missing from 3 subjects. HV = Healthy volunteers; OPD = Other (non cluster B) personality disorder; BPD = Borderline personality disorder; AA = African American.

[a] HV < BPD, OPD.

[b] HV > OPD, BPD.

* $p < .05$.

Table 2

Lifetime psychopathology as a function of diagnostic group.

	Group		
	OPD (*N* = 122)	**BPD (*N* = 127)**	**X^2**
Axis I psychopathology (*% subjects*)			
Any Axis I disorder	92 (75.4%)	116 (91.3%)	11.48***
Major depressive disorder	52 (42.6%)	93 (73.2%)	23.96***
Other mood disorder	14 (11.5%)	15 (11.8%)	0.07
Post traumatic stress disorder	18 (14.8%)	38 (29.9%)	8.21**
Other anxiety disorders	43 (35.2%)	62 (48.8%)	4.70**
Alcohol dependence	15 (12.3%)	36 (28.3)	11.01**
Other substance dependence	5 (4.1%)	32 (25.2%)	21.89***
Axis II psychopathology (*% subjects*)			
Paranoid PD	16 (13.1%)	44 (34.6%)	15.77***
Schizoid PD[a]	5 (4.1%)	0 (0.0%)	5.31*
Schizotypal PD	18 (14.8%)	15 (11.8%)	0.47
Borderline PD	0 (0.0%)	127 (100%)	249.00***
Antisocial PD	0 (0.0%)	40 (31.5%)	45.78***
Histrionic PD[a]	0 (0.0%)	6 (4.7%)	5.90*
Narcissistic PD	0 (0.0%)	35 (27.6%)	39.12***
Avoidant PD	20 (16.4%)	27 (21.3%)	0.96
Obsessive–compulsive PD	38 (31.1%)	33 (26.0%)	0.81
Dependent PD[a]	3 (2.5%)	7 (5.5%)	1.50
PD not otherwise specified	46 (37.7%)	0 (0.0%)	58.74***

Note: PD = Personality disorder; OPD = Other (non cluster B) personality disorder; BPD = Borderline personality disorder.

[a]Fishers exact test used.

*$p < .05$.

**$p < .01$.

***$p < .001$.

Table 3

Aggression measures as a function of diagnostic group.

	Group			*F*	η_p^2
	HV	**OPD**	**BPD**		
Point subtraction aggression paradigm	(*N* = 112)	(*N* = 122)	(*N* = 127)		
B/total[a,b]	.03 (.06)	.09 (.11)	.08 (.10)	8.70***	.05
Buss–perry aggression questionnaire	(*N* = 105)	(*N* = 110)	(*N* = 108)		
Physical aggression[a,b,c]	15.86 (4.47)	19.33 (5.81)	25.19 (8.15)	36.59***	.19
Verbal aggression[a,b,c]	13.92 (4.18)	17.36 (5.53)	18.90 (5.31)	15.41***	.09
Anger[a,b,c]	13.27 (4.48)	18.86 (6.64)	23.16 (6.26)	42.48***	.21
Hostility[a,b,c]	13.88 (5.46)	22.17 (6.52)	26.53 (6.58)	65.15***	.29
Life history of aggression	(*N* = 72)	(*N* = 102)	(*N* = 87)		
Aggression[a,b,c]	5.90 (3.64)	13.34 (6.37)	17.05 (4.68)	53.07***	.30
Self-aggression[a,c]	0.22 (.00)	0.46 (1.12)	1.12 (2.44)	7.91***	.06
Antisocial behavior[a,b,c]	1.70 (2.51)	3.79 (4.23)	6.83 (5.10)	17.72***	.12
State trait anger expression inventory	(*N* = 89)	(*N* = 89)	(*N* = 98)		
State anger[a,b]	10.93 (1.50)	12.92 (5.28)	13.87 (5.22)	8.28**	.04
Trait anger[a,b,c]	13.88 (3.03)	20.22 (7.10)	25.49 (7.24)	43.85***	.25
Anger in[a,b,c]	14.80 (3.65)	18.00 (4.32)	20.65 (4.58)	24.06***	.15
Anger out[a,b,c]	13.22 (2.42)	17.54 (6.05)	19.60 (5.54)	21.85***	.14
Anger control[a,b]	26.31 (3.52)	20.67 (6.17)	19.04 (5.49)	27.89***	.17
Anger expression[a,b,c]	17.71 (6.09)	30.87 (12.66)	37.21 (11.14)	45.64***	.26

Note: All means adjusted for number of Axis I disorders, age and education; HV = Healthy volunteers; OPD = Other (non cluster B) personality disorder; BPD = Borderline personality disorder.

[a]BPD vs. HV significant.

[b]OPD vs. HV significant.

[c]BPD vs. OPD significant.

** $p < .01$.

*** $p < .001$.

Table 4

Impulsivity measures as a function of diagnostic group.

	Group			F	η_p^2
	HV	**OPD**	**BPD**		
Passive Avoidance Learning	(*N* = 65)	(*N* = 80)	(*N* = 95)		
PA ratio	.69 (1.12)	.47 (.33)	.53 (.51)	1.46	0.01
Immediate Memory Task	(*N* = 103)	(*N* = 112)	(*N* = 116)		
IMT ratio[a,b]	.26 (.20)	.32 (.17)	.38 (.32)	3.87**	0.02
Bechara Gambling Task	(*N* = 103)	(*N* = 111)	(*N* = 119)		
Block 1	13.46 (4.81)	13.73 (4.39)	13.24 (4.28)	0.30	0.00
Block 2	11.77 (4.56)	11.46 (4.10)	11.18 (4.70)	0.34	0.00
Block 3	11.28 (5.13)	11.53 (5.40)	11.59 (5.30)	0.06	0.00
Block 4	10.38 (6.02)	11.18 (5.43)	11.52 (5.55)	0.64	0.00
Barratt impulsivity scale	(*N* = 103)	(*N* = 114)	(*N* = 111)		
Motor[a,b,c]	17.71 (3.44)	21.08 (4.41)	24.45 (5.17)	35.77***	0.18
Cognitive[a,b,c]	14.30 (2.94)	17.91 (3.52)	19.62 (3.52)	40.77***	0.20
Nonplanning[a,b,c]	23.04 (4.29)	26.29 (5.32)	28.86 (5.60)	24.03***	0.11
Life history of impulse behavior	(*N* = 86)	(*N* = 89)	(*N* = 97)		
CSI[a,b,c]	17.30 (30.97)	51.96 (45.95)	82.05 (57.66)	23.39***	0.15
NCSI	62.52 (31.59)	49.04 (32.16)	63.21 (58.08)	3.04	0.02

Note: All means adjusted for number of Axis I disorders, age and education; HV = Healthy volunteers; OPD = Other (non cluster B) personality disorder; BPD = Borderline personality disorder; CSI = Clinically significant impulsivity; NCSI = Non-clinically significant impulsivity; PAT Ratio and IMT Ratio = commission errors/correct detections; Bechara Gambling Task = disadvantageous deck selections.

[a] BPD vs. HV significant.

[b] OPD vs. HV significant.

[c] BPD vs. OPD significant.

** $p < .01$.

*** $p < .001$.

Table 5

Correlations among aggression variables across subjects.

	BPQ-P	BPQ-V	BPQ-A	BPQ-H	LHA-A	LHA-S	LHA-N	STX-S	STX-T	STX-I	STX-O	STX-C	STX-E
PSAP-B	.20***	.10	.18**	.20***	.20**	.03	.13*	.07	.16**	.13**	.18**	−.16**	.19**
BPQ-P		.43***	.64***	.64***	.46***	.14*	.44***	.28***	.52***	.33***	.46***	−.41***	.50***
BPQ-V			.70***	.58***	.53***	.18**	.44***	.35***	.55***	.32***	.59***	−.44***	.57***
BPQ-A				.75***	.63***	.24***	.45***	.39***	.71***	.46***	.66***	−.62***	.73***
BPQ-H					.52***	.27***	.42***	.38***	.65***	.58***	.53***	−.50***	.67***
LHA-A						.27***	.67***	.38***	.67***	.36***	.62***	−.65***	.69***
LHA-S							.22***	.05	.19**	.16*	.19**	−.16*	.21**
LHA-N								.41***	.57***	.33***	.46***	−.38***	.48***
STX-S									.56***	.37***	.48***	−.36***	.50***
STX-T										.57***	.80***	−.68***	.86***
STX-I											.41***	−.30***	.68***
STX-O												−.66***	.89***
STX-C													−.85***

Note: PSAP-B = Point subtraction aggression paradigm B (aggressive) response ratio; BPQ = Buss–perry aggression questionnaire; BPQ-P = physical aggression scale; BPQV = verbal aggression scale; BPQ-A = anger scale; BPQ-H = hostility scale; LHA = Life history of aggression; LHA-A = aggression scale; LHA-S = self-aggression scale; LHAN = antisocial behavior scale; STX = State-trait anger expression inventory; STX-S = state anger scale; STX-T = trait anger scale; STX-I = anger in scale; STX-O = anger out scale; STX-C = anger control scale; STX-E = anger expression scale.

* $p < .01$.

** $p < .01$.

*** $p < .001$.

Table 6

Correlations among impulsivity variables across subjects.

	IMT	BGT	BIS-M	BIS-C	BIS-N	LHIB-CS	LHIB-NC
PAT	.01	.08	−.04	−.03	.04	−.03	−.05
IMT		.05	.21***	.17**	.18**	.06	.11
BGT			.12*	.08	.09	.13*	.01
BIS-M				.62***	.58***	.50***	.09
BIS-C					.61***	.44***	.12
BIS-N						.61***	.10
LHIB-CS							−.43***

Note: PAT = Passive Avoidance Task ratio; IMT = Immediate Memory Task ratio; BGT = Bechara Gambling Task total disadvantageous deck selections; BIS = Barratt impulsivity scale-11; BIS-M = BIS motor impulsivity scale; BIS-C = BIS cognitive impulsivity scale; BIS-N = BIS nonplanning scale; LHIB = Life history of impulsive behaviors; LHIB-CS = LHIB clinically significant impulsivity; LHIB-NC = LHIB non-clinically significant impulsivity.

* $p < .01$.

** $p < .01$.

*** $p < .001$.

Table 7

Correlation between behavioral and self-report measures of aggression as a function of group.

	PSAP B-ratio		
	HV	**OPD**	**BPD**
BPAQ			
Physical	−.12	.12	.24*
Verbal	−.11	−.01	.11
Anger	−.04	.01	.24*
Hostility	.01	.06	.15
LHA			
Aggression	.22	.02	.19
Self-aggression	_[a]	−.02	−.13
Antisocial	.14	.01	.13
STAXI			
State anger	−.07	−.04	.03
Trait anger	.11	−.06	.17
Anger in	−.01	.02	.02
Anger out	.10	.05	.10
Anger control	−.14	.05	−.09
Anger expression	.13	.01	.10

Note: HV = Healthy volunteers; OPD = Other (non cluster B) personality disorder; BPD = Borderline personality disorder PSAP B-Ratio = Point subtraction aggression paradigm aggressive response ratio; BPAQ = Buss–perry aggression questionnaire; LHA = Life history of aggression; STAXI = State-trait anger expression inventory.

[a] No subjects in the HV group reported any self-aggression.

* $p < .05$.

Table 8

Correlation between behavioral and self-reported impulsivity as a function of group.

	HV			OPD			BPD		
	PA	**IMT**	**BGT**	**PA**	**IMT**	**BGT**	**PA**	**IMT**	**BGT**
BIS									
Motor	−.14	−.10	−.18	−.05	.07	.12	.04	.19	.25**
Cognitive	−.03	−.06	−.06	.05	.12	.03	−.05	.05	.13
Nonplanning	−.06	−.03	.03	.10	.01	.01	.20	.18	.12
LHIB									
CSI	−.08	.01	.00	−.04	−.02	.33**	.00	−.10	.06
NCSI	−.11	−.06	.08	−.03	.06	−.08	−.04	.15	−.00

Note: HV = Healthy volunteers; OPD = Other (non cluster B) personality disorder; BPD = Borderline personality disorder; IMT = Immediate Memory Rask ratio; BGT = Bechara Gambling Task total disadvantageous deck selections; BIS = Barratt impulsivity scale-11; LHIB = Life history of impulsive behaviors; CSI = Clinically significant impulsivity; NCSI = Non-clinically significant impulsivity.

** $p < .01$.