AFFECTIVE INSTABILITY AND IMPULSIVITY IN BORDERLINE PERSONALITY DISORDER

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MARIKA SOLHAN

Dr. Timothy J. Trull, Thesis Supervisor

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The undersigned, appointed by the dean of the Graduate School, have examined the thesis entitled

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presented by Marika Solhan,
a candidate for the degree of Master of Arts,
and hereby certify that in their opinion it is worthy of acceptance.
Professor Timothy Trull
Due forgon Donia McCombry
Professor Denis McCarthy
Professor Phillip Wood
Professor Sara Gable

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INTRODUCTION

Borderline personality disorder (BPD) is a complex disorder in which individuals have a maladaptive personality style that is exhibited in a variety of contexts, emerges by early adulthood, and leads to distinct patterns of dysfunction in their behavior and relationships (APA, 2000). This disorder is primarily characterized by intense, frequently shifting moods (affective instability) and self-destructive, impulsive behavior (impulsivity) (APA, 2000). Both affective instability and impulsivity play major roles in the diagnosis, treatment, and course of BPD (Gunderson, 2001). While neither of these features is necessary or sufficient for a BPD diagnosis, research has consistently indicated that both are strongly associated with the diagnosis of BPD and with other features and life events that are clinically relevant to BPD. These two features are theorized to contribute to the other characteristics of the disorder (Linehan, 1993), including dysfunctional relationships, identity disturbance, suicidal and parasuicidal behaviors, frantic efforts to avoid abandonment, feelings of emptiness, inappropriate and intense anger, and stress-related transient psychotic symptoms.

In the present study, the relationship between these two core constructs (affective instability and impulsivity) and additional BPD features will be examined. One influential theory suggests that the interaction between affective instability and impulsivity uniquely defines BPD and largely accounts for other BPD features (Siever & Davis, 1991). In accordance with this theory, we first hypothesized that the interaction of the two personality features of negative affectivity and disinhibition (which are analogous to affective instability and impulsivity, respectively) are associated with concurrent BPD

symptomatology over and above what can be accounted for by either personality feature independently. Further, we hypothesized that negative affectivity and disinhibition, as assessed at an initial time of measurement, would significantly predict BPD symptomatology when assessed two years later. We also hypothesized that the interaction of negative affectivity and disinhibition would possess a unique ability to predict BPD symptoms two years later, over and above either construct alone.

The results of the present study enable a better understanding of the relative contributions of affective instability and impulsivity to the development of BPD features. Research examining affective instability and impulsivity can ultimately lead to a better understanding of the etiology of BPD, more effective treatments, and an increased ability to predict the course of the disorder.

Affective Instability

An individual with BPD who experiences affective instability commonly has a baseline affective state of general dysphoria, which is frequently interrupted by the individual's shifts to anxiety, anger, or intense depression (APA, 2000). These shifts are typically triggered by the individual's emotional reactivity to interpersonal or environmental stressors. This conceptualization of affective instability highlights three important parts of this complex construct. First, affectively unstable individuals experience intense and frequent mood variability. Second, they are emotionally reactive and their moods are greatly influenced by environmental stimuli. Finally, their moods typically shift between various negative affects (e.g., depression, anxiety, and anger). In other words, one would expect the emotional state of a person with affective instability to fluctuate often (e.g., several times per day), from a baseline mood to a variety of negative

affects, and to vary in response to environmental cues. Affective instability is central to BPD and has been shown to predict other BPD features as well (Bagge et al., 2004; Yen et al., 2004; Koenigsberg et al., 2001). Affective instability also helps to distinguish BPD from other disorders such as major depression (Gunderson & Phillips, 1991) and bipolar disorder (Cowdry, Gardner, O'Leary, Leibenluft, & Rubinow, 1991; Koengisberg et al., 2002; Henry et al., 2001; Paris, 2004; Yen, Zlotnick, & Costello, 2002).

Mood variability. In an individual with affective instability, mood shifts are frequent and may seem random. Cowdry and colleagues (1991) examined mood variability in four groups: psychiatric outpatients with major depression, women with premenstrual syndrome (PMS), outpatients with borderline personality disorder (BPD), and normal controls. Participants rated their general mood each morning and evening for two weeks. The patients with BPD showed more day-to-day and within-day variability than the normal controls and patients with depression. Although mood states of the BPD and PMS participants were equally variable, the patterns of fluctuation indicated that one morning's mood had virtually no effect on the subsequent morning's mood for the BPD patients, while the PMS patients showed significant day-to-day lagged effects in their mood ratings (Cowdry et al., 1991). One possible explanation for this apparent randomness in the mood ratings of BPD patients is that their mood shifts may occur in response to equally unpredictable environmental cues.

Reactivity of mood. Linehan describes individuals with BPD as being "emotionally vulnerable" (1993). She contends that part of this vulnerability includes being extremely sensitive to emotionally relevant stimuli. According to her theory, borderline individuals react quickly with extreme emotional intensity to events that may

evoke a more moderate response from other individuals (Linehan, 1993). For example, a borderline individual may react to her husband's brief business trip with great anger, profound sadness, and feelings of rejection. In addition to reactivity towards events, individuals with BPD may also react strongly to various emotional cues, such as facial expressions of emotion in others or the perception of negative affect within themselves (Levine, Marziali, & Hood, 1997). This is possibly due to the individual's inability to properly identify and process his or her own emotions or properly interpret emotional facial expressions in others (Levine et al., 1997). These features distinguish the emotional experience of a BPD individual from that of individuals with other mood disorders, such as major depression, for example, in that various external stimuli may be more responsible for the affect exhibited by those with BPD.

Although the idea of emotional reactivity is based primarily on clinical observation, the construct has received limited support from cognitive models of psychopathology. Korfine and Hooley (2000) administered a directed forgetting task to a group of individuals diagnosed with BPD from hospital and community settings. In this task, the participants were presented with positive and negative words and words that were salient to a typical individual with BPD (e.g. abandonment, rage, rejection). The participants were instructed after the presentation of each word to either remember or forget the word. Korfine and Hooley (2000) found that individuals with BPD features were more likely than normal controls to recall negative or BPD salient words when they were instructed to forget these particular words (i.e., they were unable to inhibit the impulse to remember these words). Further, the number of BPD salient words borderline subjects recalled was significantly positively correlated with the number of BPD

symptoms they displayed. The authors suggested that participants with BPD reacted so strongly to these emotionally charged stimuli that they were then unable to inhibit encoding the word (i.e. they could not forget the word when instructed to do so). These findings support the clinical observation that affectively unstable individuals react more intensely to emotionally relevant stimuli than do individuals without this personality feature.

Fluctuation between negative affects. For those individuals that exhibit affective instability, their moods typically shift between various negative affects throughout the day. Koenigsberg and colleagues (2002) examined the degree and intensity of affective instability in BPD outpatients. They found patients with BPD to be more variable in their anger and anxiety, and to exhibit more oscillations between anxiety and depression than patients with other personality disorders. There were no differences between these two groups on measures of elation, depression, or the oscillation between elation and depression. In contrast, in this same sample, there were significant differences found between the BPD group and a group of patients with bipolar II disorder, such that the bipolar group was significantly more likely to shift from euthymia to depression and elation and to shift between depression and elation (Henry et al., 2001). This is consistent with the DSM-IV conceptualization of bipolar II disorder being characterized by periods of hypomania alternating with periods of depression (APA, 2000).

In summary, affective instability appears to distinguish BPD from other personality disorders in that the affective shifts in BPD appear to involve particular negative affects (e.g. anger, anxiety, and depression). Affective instability also

distinguishes BPD from bipolar II disorder in that the affective shifts in BPD occur between different negative affects instead of between both positive and negative affects.

Affect intensity. The construct of affect intensity refers to the strength with which one subjectively experiences emotion. Affect intensity is frequently examined in conjunction with affective instability; although some research indicates that affective intensity plays a less significant role in BPD. For example, Koenigsberg et al. (2001) examined the factor structure of BPD using 20 affective state, impulsivity, and hostility variables. While factor analysis yielded two factors (affective instability and impulsivity), the only affective intensity variable included in the study failed to load on either factor above .50. Similarly, other research suggests that the differences in affective lability between individuals with BPD and individuals with other personality disorders are not purely due to the subjectively more intense emotional experience of the BPD individual (Koenigsberg et al., 2002). Rather, affective instability differences remain significant even after controlling for BPD individuals' elevated affect intensity scores (Koenigsberg et al., 2002). Other research indicates that measures of affective instability significantly predict the number of BPD symptoms, over and above affect intensity (Yen et al., 2002). According to this body of research, while individuals with BPD experience intense moods, the mood intensity component does not define the construct of affective instability as well as the mood variability component does. As previously mentioned, the affect intensity seen in individuals with BPD appears to be most important when explaining these individuals' emotional reactivity. However, interpretation of this literature is limited because positive and negative affect intensities have not been examined separately. It may be the case that the affect intensity BPD individuals

experience is of greater significance when associated with negative, rather than positive, affect states.

Impulsivity

Another core feature of borderline personality disorder is impulsivity. Generally, impulsivity refers to a lack of planning with respect to behaviors or an inability to inhibit behavioral, cognitive, and emotional urges (Critchfield, Levy, & Clarkin, 2004). The impulsivity criterion of a BPD diagnosis, according to the DSM-IV-TR, primarily focuses on impulsive behaviors. However, impulsivity is implied in many of the other BPD diagnostic criteria, such as frantic efforts to avoid abandonment, for example. The impulsivity typically seen in individuals with BPD can be conceptually organized into impulsive behaviors, cognitions, and emotions.

Individuals with BPD typically engage in a number of impulsive behaviors, such as substance abuse, sexual promiscuity, reckless driving, and excessive spending (APA, 2000). These behaviors are particularly concerning because they are highly likely to result in negative consequences and to endanger the BPD individual or others (APA, 2000). Borderline individuals engage in these activities hastily, with little or no forethought to the possible consequences of their behavior. These behaviors can result in a variety of interpersonal, professional, health, financial, or legal difficulties. For example, comorbid substance abuse disorders are common in individuals with a BPD diagnosis (Trull, Sher, Minks-Brown, Durbin, & Burr, 2000). Also, the suicidal and parasuicidal behaviors of borderline individuals, considered by some to be another example of impulsivity (Wint & Shapira, 2003), typically result in a number of severely negative consequences. These impulsive behaviors and the resulting consequences

frequently exacerbate an individual's other BPD features, leading to increased distress and dysfunction.

Individuals with BPD may also experience difficulty inhibiting impulsive cognitions. For example, one diagnostic criterion for BPD is evidence of "frantic efforts to avoid real or imagined abandonment" (APA, 2000, p. 710). Individuals with BPD who exhibit this feature may make severely distorted interpretations of others' actions and may impulsively conclude that important people in their lives will soon abandon them. Finally, research findings like those of Korfine and Hooley (2000), discussed earlier, suggest that BPD individuals have a number of cognitive deficits that result in their inability to inhibit learning or memory recall in certain situations.

Some theorists also include emotional components, such as hostility or irritability, in their definition of impulsivity in BPD. While many view affective instability as independent of impulsivity (e.g. Linehan, 1993), others believe that the emotional reactivity seen in individuals with BPD reflects an underlying emotional dyscontrol or impulsivity (van Reekum, Links, & Fedorov, 1994). Proponents of this view of BPD suggest that BPD individuals show great emotional fluctuations and reactivity to their environment primarily because they are unable to inhibit or moderate their emotional urges. In part, this view has been supported by neurobiological research that indicates the presence of serotonergic abnormalities in BPD individuals who show an inability to inhibit or regulate their negative affect (Davidson, Putnam, & Larson, 2000). These behavioral, cognitive, and affective components of impulsivity offer many examples of the ways in which impulsivity may drive the expression of other BPD symptoms.

Much of the research on impulsivity in BPD has focused on biological markers, neurotransmitter dysfunction, response to pharmacological treatments, and comorbidity and heritability rates (see Silk, 1994 for a review). These studies indicate many neurobiological, and possibly heritable, factors that significantly influence this feature of BPD. More behaviorally focused studies indicate that impulsivity predicts poorer general functioning over time (Links, Mitton, & Steiner, 1990) and predicts other BPD features such as transient stress-related psychosis, interpersonal problems, and affective instability (Links, Heslegrave, & van Reekum, 1999; Bagge et al., 2004). Others have found impulsivity to be the only BPD criterion that accounts for a significant portion of the variance in previous suicidal behavior (Brodsky, Malone, Ellis, Dulit, & Mann, 1997). Taken together, these findings argue for the inclusion of impulsivity as a core feature of BPD.

Relationship between Affective Instability and Impulsivity

Several theories exist regarding the relationship between affective instability and impulsivity and the relationship between these core features and the other features of BPD. One theory holds affective instability as the driving force in BPD, suggesting that this feature contributes causally to the development and expression of the other BPD features, including impulsivity (Linehan, 1993). An opposing theory suggests that BPD is primarily an impulsive-spectrum disorder and that all of the other BPD features, including affective instability, arise from a general disinhibition in the individual (Zanarini, 1993). Finally, Siever and Davis (1991) state that affective instability and impulsivity are equally important in the development and expression of BPD. Further,

they propose that the interaction of affective instability and impulsivity uniquely distinguishes BPD from other Axis I and II disorders.

The first theory suggests that affective instability leads to all other BPD features, including the impulsive behaviors often seen in individuals with the disorder. Linehan (1993) suggests that most borderline features result from the individual's attempts to regulate his or her emotions or from consequences of the unstable emotions. For example, an affectively unstable individual would be expected to have difficulty relating to others, due to his or her chaotic emotional state. The individual might be viewed as unpredictable, dramatic, aggressive, or depressed by peers, family, and co-workers, thereby discouraging the formation of lasting intimate relationships. This lack of connectedness (in addition to the affective instability) could lead to intense feelings of emptiness or a poorly defined sense of self. Such feelings might cause one to engage in impulsive behaviors to draw the attention of others or to take extreme actions to avoid being abandoned by important people in one's life. Also, the negative affect one experiences may be so intense in times of stress that the individual experiences transient psychotic symptoms. Finally, one might engage in self-harm behaviors to distract oneself from the constant emotional pain and upheaval that one experiences. According to Linehan (1993), behaviors such as self-harm, promiscuity, and substance use are often employed by borderline individuals as a means of coping with their intense and chaotic emotional states.

Although this theory is primarily derived from clinical observation, several empirical studies have given some support to this model of BPD. For example, Yen and colleagues (2004) examined the predictive ability of BPD features over two years and

found affective instability to be more predictive of self-harm behaviors than was impulsivity. They also found that affective instability was the only BPD feature to significantly predict suicide attempts over a two year period (Yen et al., 2004). Similarly, Koenigsberg and colleagues (2001) demonstrated that suicidal and self-harm behaviors were significantly correlated with a latent affective instability factor and not with an impulsivity factor. The affective instability factor also showed significant positive correlations with the DSM-III-R criteria regarding affective instability, feelings of emptiness, identity disturbance, and inappropriate anger. Finally, some longitudinal studies examining the course of BPD have found affective instability to be the primary feature that predicts poorer general functioning over time (McGlashan, 1985; Paris, Nowlis, & Brown, 1988). Results indicating that affective instability is more stable over time than impulsivity have been interpreted by some to mean that affective instability is the true core of BPD (Paris, 2003).

The second major theory suggests that impulsivity is the most central feature of BPD (Links, Heslegrave, & van Reekum, 1999; Zanarini, 1993). Often, these theorists cite biological markers, neurotransmitter dysfunction, and comorbidity and heritability rates as evidence that BPD is an impulsive-spectrum disorder. One account of the relationship between affective instability and impulsivity states that impulsive behavior and the consequences of such behavior cause the unstable and intense emotional shifts exhibited in individuals with BPD (van Reekum et al., 1994). Proponents of this theory suggest that borderline individuals first engage in impulsive, self-destructive behaviors due to their decreased inhibition. Then, in response to the negative consequences of their behavior, they experience intense negative affect. Other theorists suggest that affective

instability is merely another impulsive behavior (van Reekum et al., 1994). The rapid emotional reactions that borderline individuals have to meaningful stimuli may be analogous to the rapid, or impulsive, decisions they make to engage in certain behaviors (Herpertz et al., 1997). Although research in this area is limited, according to this theory, individuals with BPD would generally fail to moderate their urges to respond immediately, with respect to both their emotions and their behaviors.

Interaction between Affective Instability and Impulsivity

As indicated above, some propose that the combination of affective instability and impulsivity is what makes BPD unique. The presence of either feature without the other may indicate psychopathology besides BPD. For example, Siever and Davis (1991) suggest that the presence of impulsive traits without affective instability is more indicative of an antisocial personality. Conversely, affective instability without impulsivity is more characteristic of some mood disorders or anxious-cluster personality disorders (Siever & Davis, 1991). Therefore, affective instability and impulsivity may be viewed as reciprocally influencing forces. Borderline individuals engage in impulsive behavior in response to intense feelings of rejection, disappointment, or frustration, while affective shifts are exaggerated due to a general lack of inhibition (Siever & Davis, 1991). In contrast, antisocial individuals typically engage in impulsive behavior without provocation and bipolar individuals cycle through emotions without necessarily behaving impulsively.

Consistent with Siever and Davis' theory are results from a recent study in which BPD and antisocial personality disorder (ASPD) features were compared (Fossati et al., 2004). The authors examined the ability of various impulsivity measures to predict BPD

and ASPD symptoms in a nonclinical sample. In separate regression analyses, motor impulsivity significantly predicted the symptoms of both ASPD (after controlling for BPD symptoms) and BPD (after controlling for ASPD symptoms), indicating that both disorders are characterized by a tendency to act without thinking about consequences. Further, after controlling for ASPD and depression symptoms, BPD symptoms were significantly predicted by scales associated with emotional impulsivity and aggression, such as irritability, resentment, and guilt. In contrast, after controlling for BPD and depression symptoms, ASPD symptoms were significantly predicted by measures of overt and relational impulsivity and aggression, such as physical aggression, indirect aggression, and negativism scales. Given that these analyses controlled for depressive symptoms, the guilt, resentment, and irritability which predicted BPD symptoms are independent of comorbid depression and may be attributable instead to the affective BPD features, such as affective instability. Further, these results seem to indicate that the combination of impulsive actions and affective features is what makes BPD unique and distinct from ASPD. Unfortunately, the interaction between impulsivity and affective instability was not directly examined in this study.

In a study by Bagge and colleagues (2004), affective instability and impulsivity were found to predict grade point average, academic probation, and social adjustment in college students with borderline personality features, over the course of two years. These results remained significant even after controlling for high school academic performance, Axis I features, and other maladaptive personality variables. These results indicate that both affective instability and impulsivity are important factors in the negative outcomes related to borderline personality symptomatology (Bagge et al., 2004). However, neither

the relative importance of each feature nor the interaction between the two features was evaluated in this study.

The current study evaluates these three major theories (affective instability as primary, impulsivity as primary, and the interaction between affective instability and impulsivity) regarding affective instability, impulsivity, and their influence on other BPD features. More specifically, we were interested in the ability of an initial assessment of negative affectivity and disinhibition (analogues of affective instability and impulsivity) to account for the variance in the concurrent level of BPD symptomatology and the level of BPD symptomatology as assessed two years later. Increased negative affectivity and disinhibition were predicted to be associated with increased BPD symptoms, both at the initial assessment and at the follow-up assessment occasion, two years later. Additionally, we hypothesized that the interaction between negative affectivity and disinhibition would significantly predict BPD symptomatology over and above what is predicted by either construct alone.

METHODS

Screening Procedure

Approximately 5,000 18-year-old freshmen at the University of Missouri – Columbia were initially screened in a prospective study on the development of borderline personality disorder in young adults. The total screening sample was drawn from two cohorts of students, from the 1997-1998 and 1998-1999 academic years. During the screening phase, students were contacted through mailings, classes, telephone calls, and emails. Screening sessions were held in campus and Greek residence halls and in classrooms. Participating students were paid five dollars or given credit in their introductory psychology course.

The screening battery consisted of the Personality Assessment Inventory — Borderline Features Scale (PAI-BOR; Morey, 1991), and validity items from the Personality Diagnostic Questionnaire — Revised (PDQ-R; Hyler & Rieder, 1987). The PAI-BOR is a 24-item self-report measure that assesses the major features of BPD, including affective instability, identity problems, negative relationships, and self-harm or impulsive behaviors. The validity items from the PDQ-R were included to detect individuals who presented themselves in an overly positive fashion or responded randomly (Hyler & Rieder, 1987). The screening battery also included items gathering demographic information, familial information (e.g. parents' occupations, education, etc.), and contact information.

From the screening pool, individuals that scored at or above (B+) and below (B-) a cutoff score of 38 on the PAI-BOR were identified. Scores at or above this threshold

are indicative of clinically significant borderline features, but not necessarily a BPD diagnosis (Morey, 1991). Prior to the laboratory phase of the study, individuals were required to again complete the PAI-BOR. This was done to ensure that each individual's membership in either the above- (B+) or below- (B-) threshold category was stable over time. From these two categories of participants, individuals were randomly selected to participate in the laboratory phase of the study. An effort was made to sample approximately equal numbers of B+ and B- individuals, as well as approximately equal numbers of men and women from each threshold group. The laboratory phase of the study consisted of two waves of data collection, wave one at age 18 and wave two at age 20

Final samples. Four hundred and twenty-one individuals completed the laboratory phase of the study at wave one of data collection and 361 individuals completed the second wave of data collection. The majority of participants were female and Caucasian (see Table 1). For the analyses of this study, 418 participants provided complete data necessary for the wave one concurrent analyses (see Figure 1), 346 participants provided complete data necessary for the wave two concurrent analyses (see Figure 2), 347 provided complete data necessary for the simple prospective analyses (see Figure 3), and 345 provided complete data necessary for the comprehensive prospective analyses (see Figure 4).

Laboratory Procedure

During the laboratory phase of the study (at both wave one and wave two), several self-report measures and structured clinical interviews were administered. The sequence of the assessments was randomized to control for possible order effects.

Participants were paid \$10 per hour or were given credit for their introductory psychology course for compensation. On average, the laboratory sessions required approximately five hours to complete. Written consent to participate in the laboratory sessions was obtained from each participant, as well as consent to videotape the interview sessions for the purpose of calculating reliability estimates.

Prior to their involvement in the study, three interviewers underwent two months of supervised training in the administration and scoring of the Diagnostic Interview for Borderlines – Revised (DIB-R; Gunderson & Zanarini, 1992), the Structured Clinical Interview for DSM-IV Axis I Disorders / Nonpatient Version 2.0 (SCID-I/NP; First, Spitzer, Gibbon, & Williams, 1995), and the Structured Interview of DSM-IV Personality (SIDP-IV; Pfohl, Blum, & Zimmerman, 1994). The interviewers were unaware of the borderline features status (i.e., B+ vs. B-) of each participant. After data collection, eighty participants per interviewer were randomly selected to examine the inter-rater reliability of interview scores.

Measures

Interview assessments. At both wave one and wave two, participants were administered the SCID-I/NP, which assesses the presence and severity of symptoms related to DSM-IV Axis I disorders in non-patients. In the current analyses, comorbid Axis I pathology was controlled for using two dichotomous variables indicating the presence or absence of any lifetime diagnosis of any mood disorder and any substance use disorder. For wave one, kappas of these two variables were .84 and 1.0, respectively (see Table 2). For wave two, kappas of these variables were both 1.0 (see Table 2).

At each wave, participants were also administered two semi-structured clinical interviews to assess borderline personality disorders features, the SIDP-IV and the DIB-R. The SIDP-IV measures the presence and severity of the nine DSM-IV Axis II criteria over the past two years. From this interview, a total count of BPD symptoms was created. The intraclass correlation coefficients (ICC) indexing interrater reliability for the SIDP-IV BPD symptom count were .83 at wave one and .94 at wave two (see Table 2). The DIB-R assesses affect, cognition, and behavior associated with a diagnosis of BPD, as present in the past two years. Scores from the four major sections (affect, cognition, impulse action patterns, and interpersonal relationships) were combined to calculate a total score for the DIB-R (range = 0 to 10; Zanarini, Gunderson, Frankenburg, & Chauncey, 1989). ICCs of the total DIB-R score were .86 at wave one and .95 at wave two (see Table 2).

The levels of psychopathology endorsed by participants were generally appropriate and expected given the non-clinical nature of the sample (see Table 3). On average, participants endorsed one or none of the DSM-IV diagnostic criteria for BPD (as assessed by the SIDP-IV) and received very low scores on the DIB-R. However, some individuals did endorse significant personality pathology (e.g. met at least 5 of 9 diagnostic criteria for BPD) and it is important to note that the full range of possible scores on the SIDP-IV (maximum score of 9) and DIB-R (maximum score of 10) is represented in this dataset. Further, the lifetime history of mood and substance use disorders were more common in this sample than in the general population. At wave one, 34.7 percent of the sample met criteria for lifetime history of a mood disorder and 39.7 percent met criteria for lifetime history of a substance use disorder. Finally, these

participants endorsed lifetime psychiatric hospitalizations and outpatient psychological treatment at slightly higher rates than the general population (see Table 3). Therefore, despite the non-clinical nature of this sample, individuals with significant Axis I and Axis II pathology and consistent treatment-seeking histories were represented in this sample.

Self-report assessments. Two self-report instruments measured BPD symptomatology at both waves. All participants completed the PAI-BOR (described above) and the internal consistency (coefficient alpha) of this measure was .92 at wave one and .91 at wave two (see Table 2). Participants also completed the MMPI-Borderline Personality Disorder Scale (MMPI-BPD; Morey, Waugh, & Blashfield, 1985). This scale consists of 22 items from the MMPI that resemble DSM-III diagnostic criteria for BPD, and which distinguish between high and low scorers on the total scale score. The internal consistency (KR-20) of this measure was .76 at wave one and .77 at wave two (see Table 2).

Participants also completed the NEO-PI-R (Costa & McCrae, 1992), a 240 item self-report inventory of personality based on the Five Factor Model (FFM). The NEO-PI-R assesses the five major domains of the FFM (Neuroticism, Extraversion, Openness to Experience, Agreeableness, and Conscientiousness) and each domain is further broken down into six trait facets. In the current analyses, six of the 30 facets of the NEO-PI-R were combined to form the latent factors of trait Negative Affectivity and trait Disinhibition. The facets Anxiety, Angry Hostility, and Depression make up the trait Negative Affectivity factor. The facets Impulsiveness, Self-discipline, and Deliberation make up the trait Disinhibition factor. At wave one and wave two, high reliability was demonstrated for each of these six facets (see Table 2).

FFM traits as predictors of borderline symptoms. Many researchers feel that the five domains of the FFM are too broad to provide meaningful clinical utility. Therefore, use of the lower-order facets is important to increase specificity when examining personality disorder features (Reynolds & Clark, 2001). Trull (2001) used factor analytic techniques to define two personality traits, Negative Affectivity and Disinhibition, composed of three lower-order facets each, which are closely related conceptually to the BPD features of affective instability and impulsivity. In the current analyses, trait Negative Affectivity and trait Disinhibition, created from the NEO-PI-R, were used to concurrently and prospectively predict BPD symptoms, in order to avoid confounding these predictors with the BPD symptoms of affective instability and impulsivity that are measured in the criterion assessments of BPD symptomatology (i.e. PAI-BOR, MMPI-BOR, SIDP-IV, and DIB-R).

Negative affectivity. This trait can be defined as a tendency to experience various types of negative affect. As operationalized by Trull (2001), it is composed of 3 facets (Anxiety, Angry Hostility, and Depression) from the Neuroticism domain of the NEO-PI-R (Costa & McCrae, 1992). The Anxiety facet taps into a variety of anxious feelings, from specific fears to more general, pervasive worry (Piedmont, 1998). The Angry Hostility facet measures one's propensity to feel anger, bitterness, and resentment. Finally, the Depression facet assesses one's tendency to feel sad, lonely, and hopeless. Negative Affectivity is an appropriate trait to assess in a borderline sample because anger, anxiety, and depression are the most common types of negative affect expressed in affectively labile borderline individuals (Koenigsberg et al., 2002). Further, Negative Affectivity has been found to be positively associated with BPD features and other

measures of affective instability (Trull, 2001). For the current analyses, Negative Affectivity was used as a trait indicator of affective instability.

Disinhibition. Trait Disinhibition can be conceptualized as a general lack of control over one's emotional and behavioral reactions and urges. The three facets that define this trait are Deliberation and Self-discipline (from the Conscientiousness domain) and Impulsiveness (from the Neuroticism domain) (Trull, 2001). Deliberation measures the extent to which one carefully considers a behavior before engaging in action (i.e. to inhibit urges to act immediately). Self-discipline measures one's ability to focus on and complete a necessary or desired task (i.e. to inhibit feelings of boredom or distraction). Both Deliberation and Self-discipline are inversely related to trait Disinhibition and are therefore reverse-scored in the current analyses. Finally, Impulsiveness measures the extent to which one is unable to inhibit urges and desires for immediate action. Impulsiveness is distinct from Self-discipline in that it refers to one's ability to refrain from acting on urges with undesirable outcomes, while Self-discipline refers to one's ability to maintain involvement with wanted activities (Piedmont, 1998). Disinhibition closely resembles the impulsivity feature of BPD and has been found to account for a significant amount of the variance in borderline personality disorder pathology (Trull, 2001).

RESULTS

Structural Models

Structural equation modeling is an appropriate analytic technique for these analyses for several reasons. First, it allows one to use latent factors as predictor and outcome variables, which are presumed to be free of measurement error and are therefore a better estimation of the true construct (Loehlin, 2004). Second, this analytic technique allows more freedom to model possible measurement error in the observed variables. Third, structural equation modeling allows one to estimate the general fit of one's model (e.g. loglikelihood ratios) and allows for comparisons between certain types of models (Tomarken & Waller, 2005). Finally, structural equation modeling is particularly well-suited for models involving interactions between latent constructs (Tomarken & Waller, 2005).

This study's analyses included four structural models (see Figures 1-4). Each modeled Negative Affectivity and Disinhibition factors (each composed of three NEO-PI-R facet scores) predicting a latent factor of Borderline Features. The Borderline Features factor was composed of the DIB-R total score and the SIDP-IV borderline personality disorder symptom count, as well as the total scores of the PAI-BOR and MMPI-BPD self-report inventories. In each model, the error terms of the self-report measures (PAI-BOR and MMPI-BPD) and of the interviews (DIB-R and SIDP-IV) were correlated with each other, to more accurately represent probable measurement error. In each model, information from the SCID was used to control for the lifetime presence of Axis I diagnoses of mood disorders and substance use disorders, either as assessed at the

initial (see Figure 1) or follow-up time of assessment (see Figures 2-4). This Axis I diagnostic information was ultimately represented by two dichotomous manifest variables, indicating the lifetime presence of any mood disorder or any substance use disorder. Mood disorders and substance use disorders are the two groups of Axis I pathology most closely related conceptually to the constructs of affective instability/negative affectivity and impulsivity/disinhibition. Further, these two groups of Axis I disorders are quite commonly comorbid with BPD. Initially, in order to test the most conservative possible models, dichotomous variables indicating the presence or absence of anxiety disorders, eating disorders, and other Axis I disorders were initially included in the current analyses. These additional Axis I variables were neither significantly related to BPD features nor significantly improved model fit and were therefore dropped from subsequent models. Finally, a comprehensive prospective model, which included both wave one and wave two measures of Borderline Features, examined change in Borderline Features over the course of two years by controlling for the initial assessment of Borderline Features at wave one (see Figure 4).

The four models also examined the association between the interaction of Negative Affectivity and Disinhibition (NAxDis) and BPD symptomatology. In each model, a significant and positive path from the latent interaction (NAxDis) to the latent Borderline Features factor was expected, in addition to positive and significant paths from each Negative Affectivity factor and Disinhibition factor to the latent Borderline Features factor. Such results would indicate a significant association between the interaction and outcome variable, over and above the main effects of Negative Affectivity and Disinhibition. As previously mentioned, all models controlled for lifetime Axis I

mood and substance use diagnoses and the fourth model also controlled for BPD symptomatology as assessed at wave one.

These four structural models were fit to the largest possible sample the data would allow, in order to maximize power in each analysis. However, due to missing data, the sample sizes are different between each model (N = 418, 347, 346, and 345, respectively). The participants omitted in models 2, 3, and 4 due to missing data did not significantly differ from the rest of the sample on any variable (based on the mean of each variable) included in the models. Therefore, differences between model 1 and models 2, 3, and 4 do not appear to be due to differences between the samples.

While the reliability of the measures used in these analyses are quite good (see Table 2), the mean levels of the variables of interest were examined between wave one and wave two for the 345 participants providing full data at both times of assessment (see Table 3). The only significant difference was found in the total score of the PAI-BOR, such that the mean score was higher at wave one (M=31.25) than at wave two (M=24.37). The effect of this difference on the overall results of this study is unclear. *Structural Equation Modeling with Latent Variable Interactions*

Analyses were conducted using Mplus (Muthen & Muthen, 2005). Mplus utilizes the Latent Moderated Structural Equation (LMS; Klein & Moosbrugger, 2000) approach for structural models which calculate an interaction between two latent variables. The LMS approach is the most appropriate approach to the estimation of latent interactions, because it directly accounts for the nonnormality implied by the interaction term. This approach utilizes Maximum Likelihood (ML) estimation and the EM algorithm, enabling the estimation of standard errors for parameter testing. Although ML estimation assumes

that predictor variables are normally distributed, this approach is still robust to violations of that assumption. However, there are some limitations to this approach. The LMS approach does not permit estimation of standardized path estimates. Therefore, the results presented from the current analyses are unstandardized path estimates, making the comparison of the magnitude of the paths impossible. The LMS approach also does not allow the estimation of commonly used fit indices (e.g. RMSEA, TLI, etc.). Instead, model comparison is achieved using a chi-square difference test of the loglikelihood ratio statistics of nested models (Klein & Moosbrugger, 2000). In addition, the relative improvement in fit between models can be assessed using the Akaike Information Criterion (AIC; Akaike, 1974), which also allows for comparisons between non-nested models. While lower AIC values indicate improved fit relative to higher AIC values, isolated AIC values and the magnitude of change in AIC values are not informative (Schermelleh-Engel, Moosbrugger, & Muller, 2003).

Model Fit

First, a concurrent model was fit to the wave one data (see Figure 1). As predicted, Negative Affectivity, Disinhibition, lifetime history of mood disorders, and lifetime history of substance use disorders were all significant positive predictors of Borderline Features at wave one. The interaction between Negative Affectivity and Disinhibition (NAxDIS) was marginally significant as a positively-related predictor of wave one Borderline Features. A chi-square difference test of the loglikelihood ratios of this full model and a nested model (i.e. without the interaction term) indicated that the addition of the NAxDIS interaction term did not significantly improve model fit ($\Delta \chi^2 = 1.57$, df = 1, p > .10). The AIC value decreased with the full concurrent model (Δ AIC =

1.51). While the magnitude of this difference is not informative and this decrease in the AIC value could indicate improved model fit, in general the addition of the interaction term to this model does not appear to improve model fit.

Second, a concurrent model utilizing only wave two data was fit (see Figure 2). In this model, Negative Affectivity and Disinhibition, as assessed at wave two, were significant positive predictors of wave two Borderline Features. In addition, variables indicating the lifetime history of mood disorders and substance use disorders, as assessed at wave two, significantly predicted wave two Borderline Features. Finally, the interaction between wave two Negative Affectivity and Disinhibition was a significant positive predictor of wave two Borderline Features, over and above the other predictors. The chi-square difference test of the loglikelihood ratios of this full model and a nested model indicated that the inclusion of the interaction term resulted in significant improvement in model fit ($\Delta \chi^2 = 10.37$, df = 1, p < .01). The AIC value also decreased with the full model (Δ AIC = 18.73), further indicating improved model fit with the interaction term.

Third, a simple prospective model was fit to the data (see Figure 3). In this model, wave one Negative Affectivity and wave one Disinhibition were significant positive predictors of wave two Borderline Features. Again, variables indicating the lifetime history of mood disorders and substance use disorders, as assessed at wave two, accounted for a significant part of the variance in wave two Borderline Features. Finally, the interaction between wave one Negative Affectivity and wave one Disinhibition was also a significant positive predictor of wave two Borderline Features. The chi-square difference test of the loglikelihood ratios of this full model and a nested model indicated

that the inclusion of the interaction term resulted in significant improvement in model fit $(\Delta \chi^2 = 5.50, df = 1, p < .05)$. The AIC value also decreased with the full model (Δ AIC = 9.01), further indicating improved model fit when the interaction term was included.

Finally, a comprehensive prospective model was fit to the data (see Figure 4). Consistent with the previous analyses, wave one Negative Affectivity and Disinhibition significantly predicted wave one Borderline Features. Variables indicating the lifetime history of mood and substance use disorders significantly predicted wave two Borderline Features. However, wave two Borderline Features was not significantly predicted by wave one Negative Affectivity, wave one Disinhibition, or wave one Borderline Features. Finally, the interaction between Negative Affectivity and Disinhibition was not a significant predictor of wave one Borderline Features, but was a marginally significant predictor of wave two Borderline Features. The chi-square difference test of the loglikelihood ratios of this full model and a nested model (in which both interaction paths were omitted) indicated that the addition of the interaction terms did not significantly improve model fit ($\Delta \chi^2 = 3.74$, df = 2, p > .10). The AIC value decreased in the full model (\triangle AIC = 3.49), but the conclusion of improved model fit is not appropriate. Discussion of Results

Taken together (see Table 7), these four models suggest several things about the relationships between affective instability/negative affectivity, impulsivity/disinhibition, and borderline personality features. First, trait negative affectivity and trait disinhibition are clearly significant predictors of concurrently measured borderline personality features, even after controlling for comorbid Axis I pathology. Second, Axis I mood disorders and substance use disorders are significantly related to the expression of

borderline personality features, over and above important underlying personality traits. Third, the interaction between trait negative affectivity and trait disinhibition seems to uniquely predict concurrently measured borderline personality features, over and above that predicted by either personality trait alone. Given the moderate to strong correlations between negative affectivity, disinhibition, borderline personality features, and lifetime history of mood disorders and substance use disorders (see Tables 4-6), the ability of these predictors to account for unique variance in borderline features, over and above one another, is remarkable. Finally, given the stability of borderline personality features over time, trait negative affectivity, trait disinhibition, and the interaction of these traits do not seem to predict change in borderline personality features over time. In other words, the effects of these traits on the change in personality pathology seem to be mediated by previous levels of borderline personality pathology. In this study, wave one and wave two Borderline Features were very strongly correlated with each other (r = .91; see Table 6). Such a large correlation between these two latent variables helps to explain the inability of the other predictors to account for a significant amount of the change in borderline personality features over time.

DISCUSSION

This study marks the first attempt to empirically investigate an interaction between affective instability and impulsivity as a predictor of BPD features over time. Although affective instability and impulsivity are generally viewed as the core features of BPD, the existing literature is mixed regarding the relative contribution of each feature to the overall development and course of BPD. Research exists in support of Linehan's theory that emotional dysregulation drives BPD as well as in support of BPD as an impulsive-spectrum disorder. An interaction between these constructs may help to explain these mixed results, suggesting that each feature's contribution to the disorder may vary depending on the other feature.

The results of this study lend support to the importance of the interaction between affective instability and impulsivity in the manifestation of BPD features. In the concurrent analyses, the interaction between affective instability/negative affectivity and impulsivity/disinhibition was a significant (or marginally significant) predictor of BPD features, over and above either trait alone and after controlling for lifetime history of mood and substance use disorders. In addition, this interaction also predicted a significant amount of variance in BPD features measured two years later. These results suggest that the interaction between affective instability and impulsivity is important in the manifestation of borderline personality pathology and explains unique variance in BPD not otherwise accounted for by individual personality traits and comorbid psychopathology. Therefore, these results carry a variety of implications for the conceptualization, assessment, diagnosis, and treatment of BPD.

The findings of the current study support the theory proposed by Siever and Davis (1991), emphasizing the importance of both affective instability and impulsivity in the development and manifestation of BPD. Consistent with this theory, the present results indicate that neither affective instability nor impulsivity is the sole driving force behind BPD. Rather, both features are important and combine to account for unique variance in BPD. As suggested by Siever and Davis, affective fluctuation and impulsive behavior are symptoms of a variety of other psychological disorders. However, the unique combination of these symptoms helps to distinguish BPD from related psychopathology such as mood disorders (e.g. major depression) and impulsive-spectrum disorders (e.g. substance use disorders). The results of this current study highlight this point particularly well, given that comorbid mood and substance use disorders were controlled for in these analyses.

From a theoretical perspective, the significance of this interaction should inform future research in this area. While these findings highlight the importance of the interaction between affective instability and impulsivity, the nature of the relationship between these BPD features remains ambiguous. The current findings suggest that individuals with increased affective instability and impulsivity also exhibit increased borderline personality features. However, the current study can not determine whether one of these core features "causes" the other, or vice versa. Rather, it can only be concluded that the combination of these two features is important. Future research might examine any causal relationships that may exist between these features, as well as the degree to which the relationship between these features shifts over the course of the disorder. Perhaps affective instability is more influential during the initial development of

BPD, while, later in the disorder, concurrent impulsive behavior serves to perpetuate the other symptoms of BPD (and therefore the diagnosis). An alternate hypothesis might focus on unique aspects of BPD which seem to embody this affective instability/impulsivity interaction. For example, individuals exhibiting affective instability are typically extremely emotionally reactive and display rapid and intense emotional reactions to environmental cues. The emotional reactivity component of affective instability could be conceptualized as a construct which inherently consists of emotional fluctuation (or dysregulation) and impulsive responses. Individuals with increased emotional reactivity might be more likely to exhibit stronger effects of an affective instability/impulsivity interaction (and therefore increased borderline personality pathology).

The findings of this current study also have implications for the process of diagnosing BPD. Given the importance of an affective instability/impulsivity interaction suggested by these analyses and the suggestion by Siever and Davis that such an interaction uniquely defines BPD, it follows that the criteria for diagnosing BPD should more accurately reflect this theoretical and empirical perspective of BPD. Currently, an individual must exhibit any 5 of 9 possible diagnostic criteria for BPD (APA, 2000). Such a diagnostic approach allows for tremendous heterogeneity in the clinical picture of BPD. For example, two individuals could both meet diagnostic criteria for the disorder, but share only one symptom in common. However, current research and theory about BPD consistently support affective instability and impulsivity as core features of the disorder. Changes in the diagnostic system to reflect this (e.g. requiring that these specific criteria be met or weighting these diagnostic criteria more heavily) would help to align

clinical diagnosis with current research and theory. Given the results of the current study, a logical change to the diagnostic system might include requiring that an individual display increased levels of both affective instability and impulsivity in order to achieve a BPD diagnosis. However, short of significant and permanent changes to the DSM, clinicians should consider incorporating the current research on BPD into their assessment and diagnostic practices by considering diagnoses other than BPD for individuals who exhibit only one of these two core features.

Greater understanding of the interaction between affective instability and impulsivity also helps to guide treatment. Currently, Dialectical Behavior Therapy (DBT; Linehan, 1993) is the best-supported empirically-validated treatment for BPD. Because DBT is based upon Linehan's theory of BPD, emotional dysregulation is at the core of the therapy, from the conceptual level of the mechanisms perpetuating the symptoms to the pragmatic targets of treatment. Treatment primarily focuses on teaching patients more effective ways of regulating their emotional states and coping with distress, instead of relying on maladaptive coping strategies, such as self-harm, substance abuse, or other impulsive behavior. Although impulsive behaviors are difficult for patients to continue if they are properly adhering to the principles and teachings of DBT, general impulsivity is not directly addressed in many aspects of this therapy. For example, in the context of emotional regulation, the inherent impulsivity in the emotional reactivity or interpersonal styles of these individuals is not directly targeted. One possible rationale for this could be that generalized disinhibition can have biological causes. However, given the relationship between affective instability and impulsivity, more direct attention to impulsive

emotional reactions, impulsive cognitions, and impulsive interpersonal styles may address some issues common in BPD, not otherwise targeted in DBT.

The results of this study also inform the assessment of borderline personality features. In the current analyses, borderline personality features were modeled using a latent factor comprised of 4 measures of BPD. This method of modeling borderline personality features offered the advantage of combining self-report and interview assessments of BPD symptoms, to create a more comprehensive picture of borderline personality pathology. With this approach, the overall factor of borderline personality features was less colored by the specific limitations of the individual measures. Rather, the unique information and perspectives offered by each assessment were combined in the borderline features factor. The theory-based information provided by the self-report PAI-BOR was complimented by more empirically-derived MMPI-BOR. And the interviews blended information from the SIDP-IV, matching the DSM-IV conceptualization of BPD, with more dimensional information from the DIB-R, assessing an individual's degree of dysfunction in key areas relevant to BPD. Assessment in research and clinical practice would provide a more complete and accurate picture BPD when a similar multi-method approach to symptom measurement was used, incorporating self-report questionnaires and structured clinical interviews. Future research in this area could examine the added benefit of including additional measurement methods, for example, observational measures of interpersonal functioning or laboratory tests of cognitive dysregulation.

It may also be worthwhile to consider the null findings of the final comprehensive prospective model of the current study. In this model, the affective instability/impulsivity

interaction did not predict changes in borderline personality features over time. In this most stringent test of the interaction, it did not appear to account for unique variance in borderline features, over and above that accounted for by previous borderline personality features and Axis I pathology. Although this is an unsurprising finding, given the inherent stability of BPD (especially over a period as brief as two years; see Tables 2 and 6), this does not mean that the interaction is unimportant over the course of the disorder. As previously mentioned, future research might examine the influence of the interaction over a much longer time frame and over many more successive assessments to fully understand the relationship between affective instability and impulsivity over the course of BPD.

The primary strengths of this study include methodological efforts to avoid criterion contamination and the nature of the sample. By using FFM composites for negative affectivity and disinhibition as predictors of BPD features, criterion contamination was avoided and multiple alternative measures could be used as indicators of a latent factor of BPD features. In addition, this approach provided the unique opportunity to examine the relationship between normal personality traits and disordered personality symptoms. The sample utilized in this study was also unique in that the sample consisted of individuals with significant borderline personality features from a non-patient population. It seems reasonable that the relationships between normal personality traits and disordered personality symptoms found in this study would likely also be present in an outpatient sample. On the positive side, detecting such relationships in an early-adulthood non-patient sample enables a unique understanding of the features

of BPD at an earlier stage of development, prior to diagnosis. Nevertheless, efforts should be made to replicate these findings in a clinical sample.

Some additional limitations of this study should be noted. First, the LMS data analysis approach limits the ways in which these data can be evaluated and these models can be compared. Standardized estimates are not available using the LMS approach, so the magnitudes of the models' paths are difficult to compare. Further, the LMS approach limits the model fit indices available for use in these analyses. As such, only the relative fit of these full models to nested models in which the interaction is excluded can be examined. It remains unclear whether these models provide adequate fit to the data, as determined by more common fit indices such as RMSEA, for example. Another limitation of this study is the sample size. For structural models in which interactions between latent constructs are calculated, sample sizes of 400 or greater are recommended (Klein & Moosbrugger, 2000). The sample size used in this study may have limited the power to detect significant relationships or derive reliable parameter estimates.

Although the LMS data analysis approach has certain limitations, it is currently the best available method for calculating interaction effects between latent variables because of its ability to accurately account for the non-normality implied by the interaction term. Hopefully the capabilities of statistical software packages such as Mplus will continue to evolve and provide a wider array of fit indices for this type of analysis in the future. However, in lieu of such advancements, these findings should be replicated using a larger sample.

Taken as a whole, the findings of this study support the importance of an interaction between affective instability and impulsivity in the manifestation of borderline

personality features. This study makes a unique contribution to this area of research by providing the first empirical support of such an interaction and for elaborating an alternative conceptualization of how these two core features relate within the context of BPD. While these findings can be useful in guiding the theory, diagnosis, and treatment of BPD, more work is needed to fully understand how these two core features of BPD function together and function in relation to the other features of BPD over the course of the disorder.

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Table 1

Demographics of Total Wave One and Wave Two Samples

	Wave1 (N=421)	Wave2 (N=361)
Sex		
Males	192 (45.6%)	160 (44.3%)
Females	229 (54.4%)	201 (55.7%)
Age	18.32	20.25
Race		
African-American	32 (7.6%)	30 (8.3%)
Asian	20 (4.8%)	14 (3.9%)
Caucasian	354 (84.1%)	303 (83.9%)
Hispanic	4 (1.0%)	3 (0.8%)
Other	11 (2.6%)	11 (3.1%)
Hx of Inpatient Treatment	7 (1.7%)	9 (2.1%)
Hx of Outpatient Treatment	105 (24.9%)	108 (25.7%)

Table 2

Reliability Estimates of Wave One and Wave Two Manifest Variables.

Measure	Wave1	Wave2
MMPI - BPD	.76	.77
PAI-BOR	.92	.91
NEO anxiety	.78	.79
NEO angry hostility	.84	.82
NEO depression	.86	.85
NEO impulsiveness	.70	.71
NEO self-discipline	.81	.86
NEO deliberation	.81	.78
DIB-R	.86	.95
SIDP-IV	.83	.94
Any lifetime Mood Disorder (SCID)	.84	1.00
Any lifetime Substance Disorder (SCID)	1.00	1.00

Note. KR-20 estimates provided for MMPI-BPD. Interclass correlations are provided for the DIB-R total score and the SIDP-IV total symptom count. Kappas provided for the SCID mood and substance use disorder variables are based on a random sample of interviews. All other reliability estimates are coefficients alpha.

Table 3

Means and Standard Deviations for all Manifest Variables

Measure	Wave1	Wave2
MMPI - BPD	10.82 (4.09)	9.69 (4.09)
PAI-BOR	31.25 (14.00)	24.37 (12.48)
NEO anxiety	16.62 (5.31)	15.28 (5.57)
NEO angry hostility	15.68 (6.00)	14.26 (5.78)
NEO depression	15.60 (6.65)	13.62 (6.34)
NEO impulsiveness	17.52 (4.71)	17.15 (4.90)
NEO self-discipline (reverse-scored)	14.33 (5.01)	13.28 (5.48)
NEO deliberation (reverse-scored)	16.06 (5.23)	15.49 (5.11)
DIB-R	1.60 (1.96)	1.68 (2.05)
SIDP-IV	.67 (1.10)	.49 (1.03)
Any lifetime Mood Disorder (SCID)	.35 (.48)	.38 (.49)
Any lifetime Substance Disorder (SCID)	.40 (.49)	.49 (.50)

Table 4

Bivariate Correlations Between Wave One Manifest Variables

	,					1					,	
	1	2	3	4	5	9	7	8	6	10	11	12
1. Anxiety	1.00	.63	.52	.37	.31	.20	.52	.49	.27	.36	.32	40.
2. Depression		1.00	.56	.45	.46	.31	.73	.57	.42	.51	4.	.07
3. Angry Hostility			1.00	.46	.33	.34	.62	.63	.40	.43	.28	.19
4. Impulsiveness				1.00	.50	09:	.58	.53	.35	.37	.21	.27
5. Self-discipline					1.00	.53	.50	.33	.24	.33	.24	.17
6. Deliberation						1.00	.51	.38	.25	.31	.15	.30
7. PAI-BOR							1.00	.71	.45	.58	.47	.24
8. MMPI-BOR								1.00	4.	.51	.36	.35
9. SIDP-IV									1.00	2 6.	.34	.35
10. DIB-R										1.00	.51	.46
11. Mood Disorders											1.00	.14
12 Substance Disorders												1 00

12. Substance Disorders Note. Self-discipline and Deliberation are reverse-scored. When |r| > .10, p < .05.

 Table 5

 Bivariate Correlations Between Wave Two Manifest Variables

	1	2	3	4	5	9	7	8	6	10	11	12
1. Anxiety	1.00	.59	.55	.42	.23	.12	.55	.51	.29	.32	.32	60:
2. Depression		1.00	.62	.49	.50	.29	89:	64.	.40	.43	.43	.13
3. Angry Hostility			1.00	.49	.32	.32	<i>L</i> 9:	.63	.37	.37	.31	.21
4. Impulsiveness				1.00	.50	.55	.52	.47	.34	.36	.31	.27
5. Self-discipline					1.00	.53	.53	.31	.29	.35	.25	.21
6. Deliberation						1.00	.49	.35	.23	.29	.17	.30
7. PAI-BOR							1.00	.63	.50	.56	.53	.28
8. MMPI-BOR								1.00	.39	.45	.30	.36
9. SIDP-IV									1.00	.65	36	.24
10. DIB-R										1.00	.41	.41
11. Mood Disorders											1.00	.13
12 Substance Disorders												1 00

12. Substance Disorders Note. Self-discipline and Deliberation are reverse-scored. When |r| > .10, p < .05.

Bivariate Correlations Between Wave One and Wave Two Predictor and Outcome Variables

Table 6

w1) 1.00 .65 .94 .56 .92 .75 .58 .54 .16 w2) 1.00 .49 .93 .75 .67 .35 .40 .43 w2) 1.00 .69 .80 .87 .51 .57 .24 v1) 1.00 .63 .74 .41 .42 .37 v2) 1.00 .91 .74 .68 .63 w1) 1.00 .91 .74 .68 .63 w1) 1.00 .57 .75 .52 w1) 1.00 .57 .75 .75 .52 w1) 1.00 .57 .75 .75 .52 w1) 1.00 .57 .75 .75 .52 w2) 1.00 .57 .75 .75 .75 .75 w2) 1.00 .57 .75 .75 .75 w2) 1.00 .15) 1.00 .65 .94 .56 .92 .75 .58 .54 1.00 .49 .93 .75 .67 .35 .40 1.00 .69 .80 .87 .51 .57 1.00 .63 .74 .41 .42 1.00 .91 .74 .68 1.00 .57 .75 1.00 .52		1	2	3	4	5	9	7	8	6	10
(a) (a) (b) (b) (c) (c) (c) (c) (c) (c) (c) (c) (c) (c	w2) 1.00 .49 .93 .75 .67 .35 .40 .43 w2) 1.00 .69 .80 .87 .51 .57 .24 v1) 1.00 .63 .74 .41 .42 .37 v2) 1.00 .91 .74 .68 .63 v2) 1.00 .57 .75 .52 w1) 1.00 .52 .14 w2) 1.00 .15 w2) 1.00 .15 w2) 1.00	1. Negative Affectivity (w1)	1.00	.65	.94	.56	.92	.75	.58	54.	.16	.21
w2) 1.00 .69 .80 .87 .51 .57 .24 v1) 1.00 .63 .74 .41 .42 .37 v2) 1.00 .91 .74 .68 .63 v2) 1.00 .91 .74 .68 .63 v2) 1.00 .57 .75 .52 v3) 1.00 .52 .14 v4) 1.00 .15 w2) 1.00	w2) 1.00 .69 .80 .87 .51 .57 .24 v1) 1.00 .63 .74 .41 .42 .37 v2) 1.00 .91 .74 .68 .63 v2) 1.00 .57 .75 .52 v3) 1.00 .57 .14 w1) 1.00 .15 w2) 1.00 .15 w2) 1.00 .15 w2) 1.00 .15	2. Disinhibition (w1)		1.00	.49	.93	.75	.67	.35	.40	.43	.47
v1) 1.00 .63 .74 .41 .42 .37 (2.3) (v1)	3. Negative Affectivity (w2)			1.00	69:	80	.87	.51	.57	.24	24
v1) 1.00 .91 .74 .68 .63 (3) (2) (2) (3) (4) (4) (4) (4) (4) (4) (4) (4) (4) (4	v1) 1.00 91 .74 .68 .63 (3) (2) (2) (3) (4) (4) (4) (4) (4) (4) (4) (4) (4) (4	4. Disinhibition (w2)				1.00	.63	.74	.41	.42	.37	.45
(v2) 1.00 .57 .75 .52 .52 .14 1.00 .52 .14 1.00 .15 1.00 .15 1.00 15 1.00	(w2) 1.00 .57 .75 .52 .14 1.00 .52 .14 1.00 .15 1.00 .15 1.00 .15 1.00 .15 1.00	5. Borderline Features (w1)					1.00	.91	.74	89:	.63	.49
(w2) 1.00 .52 .14 1.00 .15 1.00 .15 1.00	(w2) (w2) (1.00 .52 .14 .1.00 .15 .1.00 .1.00 .1.00 .1.00 .1.00	6. Borderline Features (w2)						1.00	.57	.75	.52	.58
1.00 .15 1.00 (w2)	(w2) (w2) (1.00 (1.5) (2.00) (7. Mood Disorders (w1)							1.00	.52	.14	14.
1.00 (w2)	w1) (w2) .05.	8. Mood Disorders (w2)								1.00	.15	.13
		9. Substance Disorders (w1)									1.00	09:
	Note. When $ r > .10$, $p < .05$.	10. Substance Disorders (w2)										1.00

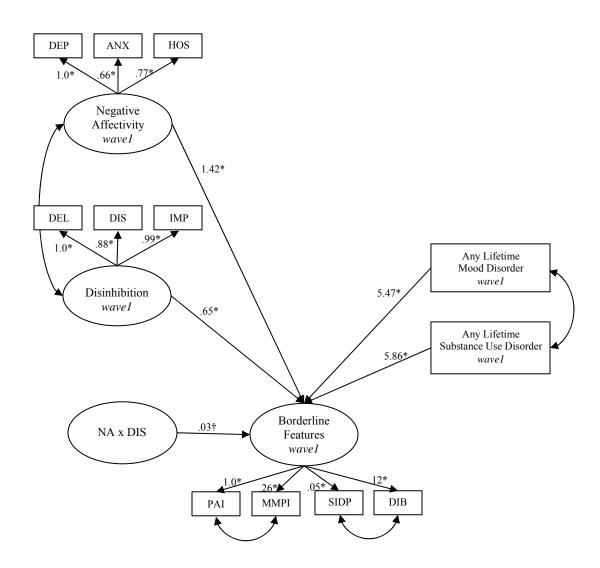
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Table 7
Summary of Model Fit and Model Comparison

	Significance of Interaction	Δχ ² Between Full and Nested Models	Significance	ΔAIC Between Full and Nested Models	Significance
Model 1	Marginal	$\Delta \chi^2 = 1.57$	df = 1, p > .10 No significant improvement	Δ AIC = 1.51	Improved model fit
Model 2	Significant	$\Delta \chi^2 = 10.37$	df = 1, p < .01 Significantly improved fit	$\Delta AIC = 18.73$	Improved model fit
Model 3	Significant	$\Delta \chi^2 = 5.50$	df = 1, p < .05 Significantly improved fit	Δ AIC = 9.01	Improved model fit
Model 4	Non- significant	$\Delta \chi^2 = 3.74$	df = 2, p > .10 No significant improvement	$\Delta AIC = 3.49$	Improved model fit

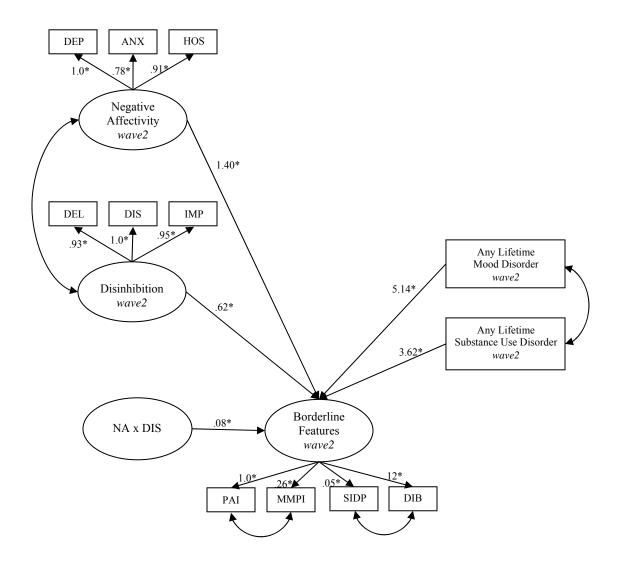
Note. Nested models restricted any path from the NAxDis latent interaction to any predictor to zero, while full models allowed these paths to be freely estimated. These model comparisons therefore reflect improvement to model fit when these interaction paths are allowed to be freely estimated. $\Delta\chi^2$ column represents the chi-square difference test of the loglikelihood ratio statistics of the full and nested models

Figure 1. Concurrent Model Using Wave One Data



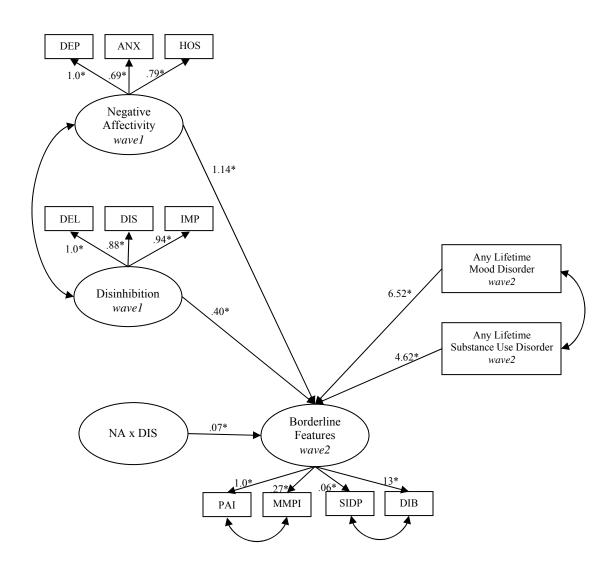
Note. Unstandardized paths presented. * p<.05; †<.10

Figure 2. Concurrent Model Using Wave Two Data



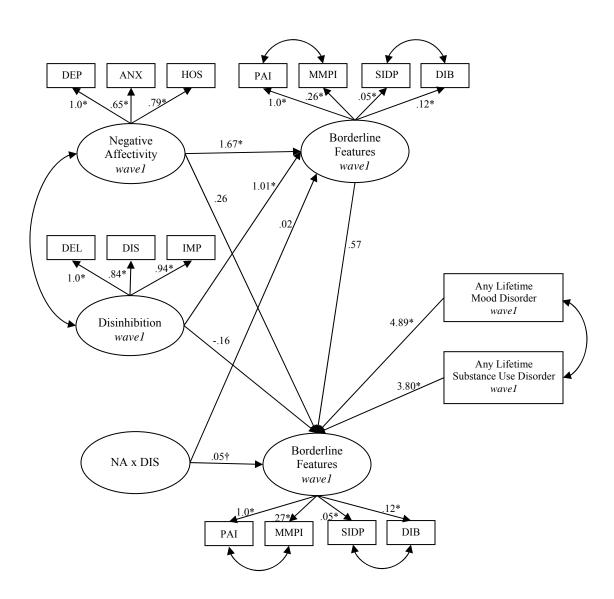
Note. Unstandardized paths presented. * p<.05

Figure 3. Simple Prospective Model



Note. Unstandardized paths presented. * p<.05

Figure 4. Comprehensive Prospective Model



Note. Unstandardized paths presented. * p<.05; †<.10