ABSTRACT

Title of Document:	DISTRESS AND RISK TAKING IN BORDERLINE PERSONALITY DISORDER: AN EXAMINATION OF NEUROCOGNITIVE MECHANISMS
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Borderline personality disorder (BPD) is a severe mental illness characterized by high rates of engagement in distress-induced risk behavior. Unfortunately, extant laboratory-based risk paradigms have failed to account for the role of distress in precipitating risk behavior, so many questions remain about processes mechanisms that underlie this behavior. The current study examined affect as a moderator of the relationship between diagnostic status and risk behavior, as measured by a behavioral risk task, and affective and non-affective neurocognitive functioning as potential mediators of this relationship. Results indicated that individuals with BPD engaged in more risk behavior in the distress condition than in the neutral condition, whereas individuals without BPD showed a decrease in risk behavior across the two conditions. However, corresponding changes in executive functioning were not observed, suggesting the need for continued research to identify alternative mechanisms (e.g., neurocognitive, motivational) to explain this effect.

DISTRESS AND RISK TAKING IN BORDERLINE PERSONALITY DISORDER:

AN EXAMINATION OF NEUROCOGNITIVE MECHANISMS

By

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Chapter 1: Introduction

1.1 Borderline Personality Disorder

      Borderline personality disorder (BPD) is a severe, persistent mental illness characterized by marked disturbances in emotional, behavioral, cognitive and interpersonal functioning (Linehan, 1993; Gunderson, 2001). The symptoms of BPD include (1) frantic efforts to avoid abandonment, (2) a pattern of unstable and intense interpersonal relationships characterized by alternating extremes of idealization and devaluation, (3) markedly and persistently unstable self-image or sense of self, (4) chronic feelings of emptiness, (5) transient, stress-related paranoid ideation or severe dissociative symptoms, (6) recurrent suicidal behavior, gestures, threats, or deliberate self-harm, (7) impulsivity in at least two areas that are potentially self-damaging, (8) affective instability due to a marked reactivity of mood, and (9) inappropriate, intense anger or difficulty controlling anger (APA, 2001). BPD is associated with high rates of psychiatric comorbidity, poor physical health and diminished academic and professional attainment (Zanarini, Frankenburg, Dubo, Sickel, Trika et al., 1998). Further, despite extensive utilization of outpatient mental health services and the increasing accessibility of empirically supported interventions for BPD (Bender, Dolan, Skodol, Sanislow, Dyck et al., 2001; Linehan, Comtois, Murray, Brown, Gallop et al., 2006; Bateman & Fonagy, 1999, 2001), these individuals frequently demonstrate poor response to treatment and are more likely to utilize costly emergency and inpatient psychiatric services (Paris, 2005; Ames-Frankel, Devlin, Walsh, Strasser, Sadik, Oldham et al., 1992; Zanarini, Frankenburg, Khera & Bleichmar, 2001). The rate of BPD is estimated to be 50% in inpatient psychiatric samples, 15-25% in outpatient samples (Widiger & Weissman,

1991), and as high as 5.9% in the general population (Grant, Chou, Goldstein, Huang, Boji, Stinson et al., 2008). Given the prevalence, degree of functional impairment and substantial costs associated with BPD, the disorder poses a considerable clinical and public health challenge.

Although there is considerable heterogeneity in the clinical presentation of BPD, it is widely considered to be a disorder of emotional dysfunction (Paris, 2005; Skodol, Gunderson, Pfohl, Widiger, Livesley et al., 2002; Linehan, 1993). Emotional dysfunction encompasses the tendency to have higher negative affectivity at baseline (Trull, Stepp & Durrett, 2003; Depue & Lenzenweger, 2001), which is compounded by greater sensitivity, intensity and duration of emotional responses to environmental and internal stimuli (Linehan, 1993; Skodol et al., 2002; Conklin & Westen, 2006). BPD symptom severity is positively correlated with negative affect intensity and reactivity (Yen, Zlotnick & Costello, 2002) and individuals with BPD often report greater state negative affect and trait neuroticism relative to healthy controls (Trull et al., 2003; Ebner-Priemer, Kuo, Kleindienst, Welch, Reisch et al., 2007). When assessed periodically over two weeks, individuals with BPD demonstrated greater variability in mood over the course of the day and greater instability of mood across days, compared to depressed and healthy controls (Cowdry, Gardner, O'Leary, Leibenluft & Rubinow, 1991). Koenigsberg and colleagues (2002) found that individuals with BPD reported greater vacillation between anger and anxiety compared to individuals with other personality disorders, and comparable to the variability observed in individuals with bipolar II disorder (Koenigsberg et al., 2002). In sum, findings consistently support a constellation of emotional vulnerabilities characteristic of BPD, highlighting the importance of

understanding affective processes independent of, and in relation to other symptoms of this disorder.

Beyond the emotional vulnerabilities associated with BPD, this disorder is characterized by striking rates of engagement in distress-induced risk behavior. For example, individuals with BPD demonstrate elevated rates of deliberate self-harm and suicide attempts, which are believed to occur in 70-75% of those with BPD, as well as high rates of substance abuse (64%), binge eating and/or purging (26%), impulsive sexual behavior (46%), excessive spending, and verbal outbursts, (APA, 2001; Gunderson, 2001; Zanarini, Frankenburg, Hennen & Silk, 2003; Zanarini et al.,1998; Trull, Sher, Minks-Brown, Durbin & Burr, 2000), with the majority of individuals with BPD engaging in multiple forms of self-destructive behavior (Zanarini et al., 2003). These self-damaging behaviors may be conceptualized as risk behaviors, to the extent that they are associated with the possibility of loss (e.g., serious physical harm or interpersonal loss), as well as the opportunity for reward (e.g., emotion regulatory or social functions; Leigh, 1999; Linehan, 1993; Nock & Prinstein, 2004).

1.2 Risk Behavior in BPD: Clinical Findings

Engagement in risk behavior is a defining characteristic of BPD, with high rates of involvement observed across a range of self-destructive behaviors (Zanarini et al., 2003). These behaviors are considered particularly dangerous, persistent and difficult to treat (Links, Helesgrave & van Reekum, 1999; Linehan, 1993; Lacey & Evans, 1986), highlighting the need to understand the basic processes involved in risk behavior. Extant studies of risk behavior in BPD have sought to understand the emotional factors

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associated with engagement in risk behavior, relying exclusively on retrospective self-reports of emotional antecedents, consequences and reasons for risk behavior. For example, in two studies of deliberate self-harm in BPD, all participants reported dysphoric affect prior to engagement in deliberate self-harm, including anger, anxiety, sadness, tension, emptiness and loneliness (Chapman & Dixon-Gordon, 2007; Kleindienst, Bohus, Ludascher, Limberger, Kuenkele et al., 2008). In another study comparing reasons for deliberate self-harm and suicide attempts among outpatients with BPD, 96% of participants endorsed emotion relief as a reason for deliberate self-harm and 86% cited emotion relief as a reason for a suicide attempt (Brown, Comtois & Linehan, 2002). Further, individuals with comorbid BPD and substance use disorders were found to be more likely than substance users without BPD to use alcohol in response to negative emotional states (Kruedelbach, McCormick, Schulz & Grueneich, 1993). Taken together, research shows a significant association between negative affect and engagement in risk behavior among individuals with BPD.

However, the literature on risk behavior is limited in scope and in the modes of assessments used. Self-report measures have inherent limitations due to biases in recall and responding (i.e., the participant may not have a clear memory of the circumstances under which risk behavior occurred, or may be reluctant to disclose behaviors perceived as negative). In addition, only a fraction of potentially relevant factors can be assessed using retrospective self-report, leaving many unanswered questions about biobehavioral and contextual correlates of risk behavior. Finally, although the clinical literature suggests that risk behavior in BPD is characterized by its frequency, severity and breadth (e.g., APA, 2001; Linehan, 1993; Zanarini et al., 2003), little is known about how risk

behavior and risk-related processes differ among individuals with and without BPD; this information may prove useful in understanding the intractability these behaviors in BPD. Thus, despite what is known about risk behavior in BPD, many questions remain unanswered, highlighting the need for broader research questions and novel modes of assessment.

1.3 Risk Behavior in BPD: Experimental Findings

One way to improve our understanding of risk behavior in BPD is to study it a controlled laboratory setting. For obvious ethical reasons, self-damaging behavior cannot be evoked in the lab, however, risk behavior can be studied using standardized behavioral analogues. Performance-based assessment is objective, limiting threats to internal validity, and it permits experimental control and manipulation of contextual and intrapersonal factors thought to influence risk behavior (Matusiewicz, Reynolds & Lejuez, *in press;* Lejuez, Read, Kahler, Richards, Ramsey et al., 2002). In general, behavioral risk tasks require the participant to evaluate competing options to balance immediate rewards with probabilistic, delayed losses. For example, the Balloon Analogue Risk Task (BART; Lejuez et al., 2002) is a computerized task that assesses the participant's tendency to pursue immediate certain rewards in the face of increasing probability of risk. During the task, the participant pumps up a simulated balloon by clicking a button on the screen. With each pump, the balloon inflates and the participant adds $0.05 to her temporary winnings. At any point, the participant can choose to deposit the earnings into the bank where they can no longer be lost, at which point the trial ends and a new trial begins. However, if the balloon pops before the participant banks the money, the winnings are lost (Lejuez et al., 2002). Advantageous responding requires

balancing the possibility of gain against the potential for loss in each trail. Given the tendency of individuals with BPD to persist in certain behaviors despite the potential for extremely negative consequences, it has been hypothesized that participants with BPD should exhibit greater risk behavior on the BART relative to controls. However, findings have not supported this hypothesis. For example, Coffey and colleagues (2009) found that found that individuals with BPD do not differ from healthy controls in terms of risk behavior on BART (Coffey, 2009), findings which have been corroborated in unpublished studies conducted by our group. Notably, these unexpected results have been corroborated by findings from a related task of risky decision-making, the Iowa Gambling Task (McCloskey et al., 2009).

In sum, behavioral measures of risk and risky decision-making have failed to show elevated risk behavior among individuals with and without BPD, denoting a discrepancy between clinical reports and experimental results. One possible explanation is the inconsistency between clinical observations and existing experimental risk paradigms, particularly with regard to the role of negative affect in precipitating risk behavior among those with BPD (Paris, 2005). Current experimental risk paradigms have failed to account for the effect of distress on risk behavior. Accordingly, the lack of findings from laboratory-based studies of risk behavior is not altogether surprising. In light of results that implicate negative affect as an antecedent of risk behavior, it is possible that distress functions as a moderator of the relationship between BPD and risk behavior. That is, individuals with BPD might not be expected to differ from healthy controls during periods of neutral or euthymic mood, but group differences in risk behavior emerge during periods of heightened distress (i.e., distress-induced risk

behavior). However, the question of affect as a moderator of the relationship between BPD and risk behavior has yet to be examined directly in a laboratory setting.

Characterizing the effect of distress on risk behavior constitutes an important first step toward identifying the factors that underline explain the strong association between BPD and distress-induced risk behavior. Although it is clear that distress and risk behaviors tend to co-occur, relatively little is known about the processes through which distress influences engagement in risk behavior, and how these processes may differ among individuals with and without BPD.

1.4 Neurocognitive Processes Underlying Risk Behavior

A recently proposed theory of risk behavior emphasizes the contribution of neurocognitive factors to engagement in risk behavior (Cohen, 2005). Briefly, the neurocognitive model attempts to explain how problems with the executive control system may lead to risk behavior (Cohen, 2005; Leith & Baumeister, 1996; Slovic, Finucane, Peters & MacGregor, 2004). This model suggests that risk behavior emerges as the product of competition between the executive control system and the affective system (Cohen, 2005; Casey et al., 2008). The executive control system is responsible for effortful, logical information processing and inhibition. In addition, the system has a unique capacity for forethought, anticipation of consequences, and delay of gratification in the service of long-term goals (Atance & O'Neill, 2001). The executive control system is opposed by the affective system, which encompasses limbic structures (amygdala, ventral striatum, insula; Clark, Boutros & Mendez, 2005; Cohen, 2005), as well as dopaminergic centers in the midbrain involved in motivation and appetitive responding

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(McClure, Laibson, Loewenstein, & Cohen, 2004; Cohen, 2005). In contrast to the executive control system, the affective system is automatic, reward-driven, and present-focused (Weber et al., 2004; Cohen et al, 2005).

Within the neurocognitive framework, the executive control system is usually dominant, inhibiting the impulses of the affective system to produce mostly rational, self-controlled behavior. However, when the inhibitory functions of the executive control system are overridden by the affective system, behavior is directed toward immediate gratification, whether in the form of reward or escape/avoidance (Weber et al., 2004). The affective system is associative and present-focused, so behavior is determined by the previous success of the response in achieving a given objective, whereas potential future consequences are not salient. Thus, risk behavior is not inherently maladaptive or dysfunctional, but may be against the individual's longer-term interests.

A number of factors may serve attenuate the executive control system, leaving it more vulnerable to be overridden by the affective system (e.g., including sleep deprivation and hunger; Durmer & Dinges, 2005; Gailliot, Baumeister, DeWall, Maner, Plant et al., 2007). Two factors in particular are of relevance to BPD, providing two plausible mechanisms to explain the relationship between BPD and distress-induced risk behavior. One factor responsible for a weak executive control system is the existence of stable, trait-like deficits (observable through stable deficits in executive functioning). These deficits are non-affective in nature (i.e., observable independent of contextual factors such as negative affect) and are thought to reflect neuromodulatory failure caused by chronic underfunctioning of the serotonergic system (Depue, 1995; Lenzenweger, Clarkin, Fertuck & Kernberg, 2004). The characteristic deficiency of the executive

control system reduces its ability to inhibit inappropriate impulses from the affective system, producing a propensity to engage in maladaptive risk behavior (Depue, 1995).

On the other hand, it has been suggested that the executive control system may be weakened by temporary, distress-related disruptions in functioning (i.e., affective deficits; Loewenstein & Lerner, 2006; Tice, Bratslavsky & Baumeister, 2001). Within this model, deficits in executive control and, consequently, the tendency to behave in risky ways, are transient and linked with the distress response. Specifically, it has been suggested that limbic over-activation in response to distress and distress-cues produces a hyperdopaminergic response that takes the executive control system "off-line" leaving behavior to be controlled the impulsive affective system (Arnsten & Goldman-Rakic, 1998; Cohen, 2005). Thus, the acute disruptive effects of distress on executive control may mediate relationship between distress and risk behavior.

In light of these distinct neurocognitive pathways to risk behavior, it is unclear which model has the most relevance to understanding the association between BPD and distress-induced risk behavior. While current research provides some support for the presence of non-affective deficits in executive control in BPD (see Fertuck et al., 2006, the proposed relationship between these deficits and risk behavior has not been tested experimentally. On the other hand, the social cognitive literature provides evidence for the relationship between affective deficits in executive control and risk behavior (e.g., Leith & Baumeister, Loewenstein & Lerner, 2003; Metcalfe & Mischel, 1999) but it has not yet been applied to BPD. However, given the prominence of emotional intensity and reactivity in BPD, and the strong relationship between distress and risk taking in this group, this model has potential relevance to BPD. Thus, an examination is warranted of

non-affective and affective deficits in executive functioning, and the extent to which these factors relate to risk behavior.

1.4.1. Non-Affective Executive Functioning in BPD

Findings suggest that BPD is associated with a specific deficit in executive functioning: the tendency to make perseverative errors. This type of error reflects difficulty transferring attention from one rule to the next and difficulties adapting behavior to align with new contingencies (Konishi, et al., 1998). Across multiple studies, findings support this as critical deficit among individuals with BPD. For example, Lenzenweger and colleagues (2004) compared patients with BPD to healthy controls on a battery of tests of executive function. Participants with BPD did not differ from controls on less demanding attention-based tasks, but evidenced a significantly greater percentage of perseverative errors (13% vs. 10%) relative to the control group. Though a seemingly minor difference, this discrepancy in performance corresponds to a medium effect size (d = .48; Cohen, 1988). Similarly, O'Leary and colleagues (1991) compared neurocognitive performance in a sample of BPD outpatients and healthy controls. While results did not reach statistical significance, perhaps due to small sample size (n = 16 in each group), participants with BPD made considerably more perseverative errors than controls (d = .55). Likewise, Burgess and colleagues (1991) examined neurocognitive deficits in outpatients with BPD and those with chronic depression. Participants with BPD made more perseverative errors (d = .57) than depressed controls. Moreover, Lenzenweger and colleagues (2004) reported that set shifting errors were not correlated with trait-level positive or negative emotionality, nor were they correlated with state anxiety or current depressive symptoms, providing additional support for the non-affective nature of these

deficits among participants with BPD. Taken together, findings suggest that, relative to controls, individuals with BPD exhibit a specific, non-affective deficit in executive functioning, characterized by persistence in a response set despite negative feedback (Barcelo & Knight, 2002).

Although findings support the presence of non-affective deficits in executive functioning in BPD, there is little research to support the relationship of these deficits to risk behavior. Research has not yet examined the relationship of executive cognitive deficits to BPD symptom clusters or specific behavioral patterns. Although there is general theoretical and empirical support for the association of executive dysfunction and risk behavior (e.g., Brand et al., 2007), it is unclear whether a causal relationship exists between non-affective neurocognitive deficits observed in BPD and risk behavior, or whether these are two distinct phenomena common to BPD. Clearly, continued research is necessary to provide support for the relationship among non-affective deficits and risk behavior in BPD, and to understand the extent to which non-affective deficits constitute a unique vulnerability to risk behavior among those with BPD relative to controls.

1.4.2 Affective Executive Functioning in BPD

In addition, it has been suggested that the executive control system may be weakened by temporary, distress-related disruptions in functioning (i.e., <u>affective deficits;</u> Loewenstein & Lerner, 2006; Tice, Bratslavsky & Baumeister, 2001). Within the framework of the affective neurocognitive model, distress disrupts neurocognitive functioning, which in turn leads to risky behavior, for example by impairing inhibitory control over risky affective urges, or impeding rational evaluation (Figner et al., 2009; Metcalfe & Mischel, 1999; Loewenstein & Lerner, 2003). This process is hypothesized to

occur as part of a normal, and perhaps even adaptive response to distress. That is, the switch from slower, resource intensive processing of the PFC to automatic to rapid subcortical functioning may have survival value in threatening situations, however, the stress-reactivity of the executive control system may impede effortful attempts at behavioral self-control and emotion regulation in humans (Arnsten & Goldman-Rakic, 1998; Ochsner, Bunge, Gross & Gabrieli, 2002). Although this process is not specific to BPD, recent findings suggest that individuals with BPD may be especially vulnerable to the effects of negative affect on executive cognitive processes due to heightened limbic reactivity (Silbersweig, Clarkin, Goldstein, Kernberg, Tuescher et al., 2007). Thus, the affective neurocognitive model provides an additional explanation for the prominent relationship between BPD and distress-induced risk behavior.

Affective deficits in executive function among individuals with BPD have not been examined directly using experimental affect manipulation. Rather, the research on affective neurocognitive deficits has tended to use emotionally evocative stimuli during cognitive tasks. Furthermore, the research on affective neurocognition has not yet examined the effect of distress on perserverative errors, as an index of cognitive control. However, several studies have found that individuals with BPD exhibit disruptions in cognitive functioning in response to relatively minor distress-related cues. For instance, findings suggest that BPD is associated with poorer cognitive inhibition of emotionally evocative words (Arntz et al., 2000; Korfine & Hooley, 2002), although they do not differ from controls in their ability to inhibit valence-neutral material (Fertuck et al., 2005). In addition, a recent fMRI study examined motor inhibition in response to distress and neutral cues among individuals with and without BPD (Silbersweig et al., 2007). They

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found that the BPD group had poorer behavioral inhibition in response to distress cues, which corresponded to limbic hyper-reactivity and decreased activity in the prefrontal cortex. Taken together, findings suggest two cognitive processes that are disrupted by exposure to distress-related cues (if not distress, *per se)* among individuals with BPD. The disruption of executive function by affect appears to be particularly exaggerated among those with BPD, which may partially explain why these individuals are likely to engage in risk behavior when distressed.

Findings that executive functioning is disrupted by negative affect are generally consistent with the neurocognitive model of risk behavior. However, very limited has been conducted to establish the relationship between affective executive dysfunction and risk behavior. Preliminary findings come from Korfine and Hooley's (2002) study of directed forgetting with BPD and non-BPD participants, in which the extent of deficits in affective cognitive inhibition was positively associated with overall BPD symptom severity, affective instability and impulsivity symptoms (i.e., engagement in multiple forms of risk behavior; *r*s = .35-.39). These findings highlight an ongoing need to clarify whether affective neurocognitive deficits and risk behavior are simply correlated, or whether a causal link exists between them.

1.5. Summary and Significance

Borderline personality disorder is a severe mental illness characterized by characterized by emotional dysfunction and high rates of engagement in self-destructive behavior (e.g., self-harm, substance use, binge eating and purging). These behaviors may be conceptualized as risk behaviors to the extent that they are associated with the possibility for gain (e.g., emotion regulatory or social functions), as well as negative

consequences (e.g., serious physical harm, interpersonal conflict). The clinical literature indicates a strong association between risk-taking and BPD. However, laboratory studies rarely find differences in risk taking comparing those with BPD and healthy controls, leaving little understanding of the key processes that underlie this relationship.

One limitation of many laboratory studies is that most current experimental risk paradigms examine risk behavior independent of affect. Thus, the role of negative affect in precipitating risk behavior (i.e., distress-induced risk behavior) has not been studied, highlighting a fundamental inconsistency between clinical observations of those with BPD, and experimental methods. Characterizing the effect of distress on risk behavior is a necessary first step toward identifying the basic mechanisms that underlie the striking relationship between BPD and distress-induced risk behavior.

A related question is what basic processes may underlie this relationship. One process thought to influence engagement in risk behavior is disruption in neurocognitive functioning. Within a neurocognitive framework, risk behavior occurs as a product of competition between the rational cognitive control system and the impulsive, reward-focused affective system. The cognitive control system usually determines behavior by inhibiting the impulses of the affective system, however, when the executive control system is weak, the affective system exerts greater influence, resulting in behavior that is present-moment focused and therefore often risky. Recent findings suggest two ways in which the cognitive control system may be compromised in BPD, which may explain the distinct pattern of distress-induced risk behavior in this group. That is, deficits in cognitive control may be non-affective (i.e., stable, traitlike), or they may be affective (i.e., transient, affect-linked) in nature. Although there is preliminary support for both

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non-affective and affective neurocognitive deficits in BPD, the relationship of these cognitive vulnerabilities to engagement in distress-induced risk behavior has not yet been examined empirically.

1.6 Current Study

The primary aim of the current study is to examine the relationship between diagnostic group (BPD, non-BPD) and risk behavior as a function of induced affect (neutral, distress). In addition, the current study examines whether group differences in risk behavior are mediated by affective or non-affective executive functioning. Finally, in the BPD group, we examine whether within-subject change in risk behavior (as a function of affect) is mediated by corresponding changes in executive functioning. To address these questions, participants will complete a computerized behavioral measure of risk behavior and a computerized measure of executive function, which will be completed over the course of three experimental sessions. Affect will be manipulated using neutral and distress imagery scripts (Sinha et al., 1999, 2000). To our knowledge, this will be the first experimental study to examine the relationships among distress-induced changes in risk behavior and executive functioning among participants with BPD. We propose:

Primary Aim.   To examine group (BPD, non-BPD) differences in risk behavior under

conditions of induced affect (neutral, distress).

Hypothesis   The relationship between diagnostic group and engagement

in risk behavior on a laboratory risk task will vary as a

function of affect, such that:

a) the BPD group will exhibit a significant increase in risk

behavior in the distress condition; and

b) the two groups will not differ in the neutral condition

but the BPD group will exhibit greater risk behavior

than the non-BPD group in the distress condition.

Secondary   To examine group (BPD, non-BPD) differences in executive functioning

Aim   under conditions of induced affect (neutral, distress).

Hypothesis   The relationship between diagnostic group and executive

functioning will vary as a function of affect, such that:

a) the BPD group will exhibit poorer executive

functioning at baseline, relative to the non-BPD group

b) the BPD group will evidence a decrease in executive

functioning in the distress condition compared to the

neutral; the non-BPD group will not show this effect.

Exploratory   To examine executive functioning (Aim #2) a mediator of the

Aim   relationship of diagnosis and affect (Aim #1).

Chapter 2: Research Design and Methods

2.1 Overall Design

A 2 x 2 mixed factorial design was used to compare risk behavior (BART score). Condition (2; neutral script, distress script) was the within-subject factor and diagnosis (BPD, non-BPD) was the between-subjects factor. Each participant completed the BART (Lejuez et al., 2002) twice, once in the neutral condition and once in the distress condition. Each participant completed the Wisconsin Card Sorting Task (WCST; Grant & Berg, 1948; Heaton et al., 1993) three times (in an effort to control for practice effects; Collie et al., 2003): once as a baseline, once in the neutral condition and once in the distress condition. Condition order was randomly assigned for each participant, and the order of the tasks was counterbalanced across participants.

2.2. Participants

Participants were recruited from the Washington, D.C. metropolitan area using flyers and postings on internet message boards (e.g., Craigslist). Recruitment materials announced a study of emotion and cognition seeking women between 18-35 who (1) had difficult or painful relationships; (2) felt their emotions are very intense or out of control and (3) did things while upset that they regret later. Interested individuals were advised to contact the study by phone or e-mail to complete a phone screening to determine eligibility.

During the phone screening, participants were asked demographic and psychiatric questions to determine their eligibility for the study. Participants were enrolled if they were (1) female and (2) between the ages of 18-35. Participants were excluded if the

endorsed: (1) current depressive episode (past month); (2) current substance dependence (past six months); (3) a lifetime history of mania; and (4) a lifetime history of psychotic symptoms. Individuals who were disqualified for any reason were offered referral information for free or low-cost mental health services.

Recruitment occurred in two stages. In the first stage (6/10 through 8/10), no questions related to BPD were asked during the phone screening. In the second stage (9/10 through 12/10), BPD screening questions were added to increase the proportion of participants with a probable BPD diagnosis. The BPD screening consisted of questions adapted from the BPD diagnostic interview to assess four symptom domains: anger, affective instability, difficult/painful relationships and impulsive behavior. In the second phase of recruitment, participants who endorsed *none* of those symptoms were enrolled, as were participants who endorsed *three or more* symptoms.

Figure 1 provides recruitment and retention information for participants enrolled in the study. Diagnostic groups were formed on the basis of a structured clinical interview for BPD. Participants were assigned to the non-BPD group ($n = 19$) if they endorsed three or fewer symptoms of BPD, and to the BPD group ($n = 11$) if they endorsed five or more symptoms. Consistent with previous research, participants who endorsed four symptoms of BPD ($n = 2$) were excluded from planned analyses (Gratz, Rosenthal, Tull & Lejuez, 2006).

2.3 Assessment Procedures

The study consisted of three sessions, all held at the Center for Addictions, Personality and Emotion Research at the University of Maryland College Park. All procedures were approved by the University of Maryland's Institutional Review Board.

2.3.1 Session 1 (Baseline)

Once consent was provided, participants began the baseline assessment in a private room. Session 1 began with the administration of two semi-structured clinical interviews to diagnose Axis I psychopathology and BPD. Following completion of the diagnostic assessment, participants completed an imagery training. The first part of the imagery training consisted of a 10-minute progressive muscle relaxation exercise, to help the participant fully engage in the subsequent imagery training. Next, the imagery training had participants practice imagining common, emotion-neutral scenes and events (e.g., reading a magazine, doing sit-ups in gym class), "as if it were happening right now." After each practice script, participants were asked to describe the imagined scene and were given tips to enhance the vividness of the imagined scenes. Participants were encouraged to notice physiological changes that accompanied the imagery. This type of training has been shown to reduce variability in baseline imagery ability, and prolong the effects of mood manipulations (Miller et al., 1987; Zoellner et al., 2003; Sinclair et al., 1994).

Following completion of the imagery training, participants were asked to recount a stressful event to be used to develop the stress imagery script. Finally, participants

completed the WCST (the WCST was administered three times in total to control for practice effects over multiple administrations; the first administration was the baseline, and the second and third administrations were compared to evaluate change as a function of affect). Following the WCST, participants were compensated $20 and scheduled for their next session.

2.3.2 Sessions 2 & 3

For the next two sessions, condition order was randomly assigned (neutral first or distress first). To encourage participants' effort on the tasks, participants were informed that they would be compensated between $10 and $20, based on their performance on the WCST and BART, however, all participants were compensated $20 for each 45-minute session.

With the exception of the script content, the sessions followed identical procedures. Before beginning, participants were provided with instructions for both tasks and reminded to use their vivid imagery techniques. Participants completed a rating of mood (PANAS 1), listened to the imagery script and completed another rating of mood (PANAS 2). Then, participants completed the WCST and BART; the order of the tasks was randomly assigned across participants. Participants were then compensated $20 for each session and scheduled for their next session (Session 2) or debriefed about the purpose of the study (Session 3).

2.4 Affect Induction

2.4.1 Distress Imagery Script

Prior to the distress condition, a personalized distress imagery script was developed for each participant, using the information provided by participants during the script development interview in Session 1 (Sinha et al., unpublished manual). During the interview, participants were asked to think of a situation that made them "sad mad or upset" and which "in the moment [they] felt like they could not do anything to change" (Sinha et al., unpublished manual). Participants rated the level of distress they experienced on a 1-10 scale, and only situations that were scored a 7 or higher were used. Example situations included break-ups, arguments, hearing bad news, being in a car accident, and taking an important exam. Scripts were written to include emotions, thoughts, urges and interoceptive cues that participants reported during the interview. The script was then audio-recorded and uploaded to the study computer. Scripts were approximately 3 minutes in length, with a 15 second period to "keep imagining." Prior to hearing each script, participants were instructed to "close [their] eyes and imagine the situation as if it were happening right now." Appendix I contains an example distress script.

2.4.2 Neutral Imagery Script

A standard neutral imagery script was used for all participants. The script described a peaceful day at the beach, and was approximately 3 minutes in length, followed by a 15 second imagery period afterward. The neutral imagery script was administered in the manner described above. Appendix II contains the standard neutral script used.

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Domain	Measure
Axis I Psychopathology	Structured Clinical Interview for DSM-IV (First et al., 2002)
BPD	Diagnostic Interview for DSM-IV Personality Disorders: BPD Module (Zanarini et al., 1996)
Personal Information	Demographics and Treatment History
Affect	Positive and Negative Affect Scale (Watson, Clark & Tellegen, 1988)
Executive Functioning	Wisconsin Card Sorting Test (Grant & Berg, 1948)
Risk Behavior	Balloon Analogue Risk Task (Lejuez et al., 2002)

2.5.1 Clinical Interview and Questionnaires

1. *Demographic and Treatment History Questionnaire.* Participants provided basic demographic information including age, gender, education level, and family income. In addition, participants provided information about past and current psychological and psychiatric treatment.

2. *Structured Clinical Interview for DSM-IV* (SCID-NP, non-patient version, First, Spitzer, Gibbon & Williams, 2002). Diagnostic inclusions/exclusions and lifetime prevalence of Axis I diagnoses were determined using the Structured Clinical Interview for DSM-IV (SCID-NP, non-patient version, First, Spitzer, Gibbon &

Williams, 2002). The SCID-IV-NP has demonstrated reliability in non-clinical

samples (First et al., 2002). Interviews were conducted by senior research staff,

trained and supervised in the administration of this interview.

3. *Diagnostic Interview for Personality Disorders- DSM-IV* (DIPD-IV, Zanarini, et al.,

1996). The DIPD-IV is an empirically supported semi-structured interview used to

diagnose Axis II personality disorders. The BPD module of the DIPD-IV consists of

53 individual questions that correspond to the nine DSM-IV symptoms of BPD. The

DIPD-IV has demonstrated good inter-rater and test-retest reliability (Zanarini et al.,

2000). Diagnoses were confirmed through discussion and consensus.

4. *Positive and Negative Affect Scale* (PANAS; Watson, Clark Tellegen, 1988*).* This 20-

item measure will be used as a manipulation check, to assess positive and negative

mood at four timepoints throughout the experiment. The scale assesses both positive

(PA) and negative (NA) affect. PA reflects the extent to which a person feels

enthusiastic, alert, and active while NA reflects a person's subjective distress and

encompasses anger, contempt, disgust, and guilt. The PANAS commonly is used to

detect changes in emotional reactions to stimuli in the manner proposed here (e.g.,

Sharpe & Gilbert, 1998). The PANAS has been found to have good internal

consistency (Cronbach's alpha = ..86-.90 for PA, .84-.87 for NA; Crawford & Henry,

2004) and good construct, convergent and discriminant validity in clinical and non-

clinical samples (Watson et al., 1988). The negative affect score will be calculated by

taking the average of ratings for the 10 negative affect items. Likewise, the positive

affect score will be calculated by taking the average of ratings for the 10 positive

affect items. Both scales will have a range from 1 to 5, with higher scores reflecting

greater intensity of negative or positive affect.

<u>*2.5.2. Behavioral Measures*</u>

1. *Balloon Analogue Risk Task* (BART; Lejuez, Read, Kahler, Richards, Ramsey et al.,

   2002). The BART is a computerized task that assesses risk-taking behavior, defined

   as behaviors that involve the potential for reward as well as the risk of harm (Leigh,

   1999). In the task, the participant is presented with a small balloon and asked to pump

   the balloon by clicking a button on the screen. With each click, the balloon inflates .3

   cm and $0.05 are added to the participant's temporary winnings. At any point, the

   participant has the option to press a button labeled "Collect $$$" which deposits the

   amount in temporary winnings to the bank (i.e., it can no longer be lost) and ends the

   trial, at which point new trial begins. However, each balloon is programmed to pop

   somewhere between 1 and 128 pumps, with an average breakpoint of 64 pumps. If the

   participant fails to press "Collect $$$" before the balloon pops, all earnings for that

   balloon are lost and the next balloon is presented. Risk behavior is operationalized as

   the average number of pumps on unpopped balloons (Lejuez et al., 2002;

   Bornovalova, Lejuez, Daughters, Rosenthal & Lynch, 2005). This index of risk

   behavior has high test-retest stability (*M* across three trials, *r* = .8; Lejuez, Aklin,

   Jones, Richards, Strong et al., 2003), which makes it appropriate for repeated-

   measures designs. The BART will be administered twice, once in the neutral

   condition, and once in the distress condition. Distress-induced change in risk

   behavior will be calculated by subtracting BART score in the neutral condition from

BART score in the distress condition, such that higher scores suggest greater risk

taking in the distress condition.

2. *Wisconsin Card Sorting Test* (WCST; Grant & Berg, 1948; Heaton et al., 1993;

Mueller, 2006). WCST is a measure of executive function during which the

participant must infer a sorting rule and adapt to periodic changes in the rule (i.e., set

shifting). In the task, the participant is shown four piles of cards, each of which has

symbols printed on it. Each pile of cards differs in terms of shape, color and number

(e.g., two green squares, three yellow stars, etc.). The participant is presented with

additional cards that must be sorted into the existing piles based on a rule, which

changes periodically without warning. The participant receives feedback following

each sort and must adapt her responses to fit the new rule. The primary dependent

variable from the WCST is the percentage of perseverative errors, which is indexed as

the total number of perseverative errors divided by total number of errors. This index

has been found to have marginal to good test-retest stability ($r = $ .56 to.83; Ingram et

al., 1999; Strauss et al., 2006). Although we considered applying statistical

corrections to the data to control for practice effects (e.g., regression-based

standardized Z-scores, as suggested by Hermann and colleagues [2006]), we

ultimately decided to follow an approach to maxinimze the interpretability of our

data. Collie and colleagues (2003) reported that practice effects on WCST are marked

from Trial 1 to Trial 2, but are minimal in subsequent trials when tasks are re-

administered at brief intervals. Therefore, the WCST was administered three times.

The first administration (Session 1) was used as a baseline measure. The second and

third administrations occurred during the second and third sessions (condition order

was randomly assigned). Change in executive functioning as a function of affect was calculated by subtracting performance in the neutral condition from performance in the distress condition, such that higher scores denotes a higher error rate (hence *poorer* executive functioning) in the distress condition.

2.6 Statistical Analyses

All analyses were conducted using the PASW Statistics Package (v. 18.0). First, the distributional properties of all noncategorical variables were assessed to determine whether they met the statistical assumptions for the analyses. Violations of normality were assessed using skewness and kurtosis, and addressed using square root transformations. Participants with missing data for BART or any session of the WCST were excluded from the primary analyses (see Figure 1).

Before addressing the primary, secondary and exploratory aims, a number of steps were taken. A manipulation check was conducted to determine whether the two imagery scripts produced significant changes in positive and negative affect from pre-script to post-script. To do this, a 2x2x2 mixed factorial ANOVA was conducted for negative affect. The within-subjects factors were condition (2; distress, neutral) and time (2; pre-script, post-script) and the between-subjects factor was diagnosis (BPD, non-BPD). Pre- and post-script negative affect ratings were used for each condition. This analysis was repeated for positive affect. Next, we examined potential covariates, including demographic and clinical variables, as well as several variables related to study procedures (e.g., condition order, pre-script NA across sessions 2 and 3). No covariates were identified, thus, so no were included covariates in subsequent analyses.

To address the primary study aim that affect will moderate the effect of diagnosis

on risk behavior, we conducted a 2x2 mixed factorial ANOVA for with risk behavior

(BART score) as the dependent variable, condition (2; distress, neutral) as the within-

subject variable and diagnosis (2; BPD, non-BPD) as the between-subjects variable. The

interaction of condition by diagnosis was probed using separate oneway repeated

measure ANOVAs for each group. The effect of diagnosis on risk behavior in the neutral

condition was probed using a univariate ANOVA with group as the between-subjects

independent variable. This analysis was repeated for the distress condition. We had a

priori hypotheses that 1) BPD group would exhibit greater risk behavior in the distress

condition than in the neutral condition and 2) the BPD group would exhibit greater risk

behavior than the non-BPD group in the distress condition, so these effects were tested at

$p < .05$ (one-tailed). All other planned analyses were conducted using $p < .05$ (two-

tailed).

Our first exploratory hypothesis aimed to test whether within-subject changes in

executive functioning[1] mediated within-subject changes in risk behavior[2], in the BPD

group. We followed guidelines by Judd, Kinney and McClelland (2001) for testing a

within-subject (repeated measure) mediator of a within-subject (repeated measure)

dependent variable. Specifically, this process involves identifying a main effect of

condition on the mediator (WCST), calculating a change score for the mediator (WCST

distress – WCST neutral) and the dependent variable (BART distress – BART neutral),

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[1] Within-subject changes in executive functioning were calculated by subtracting
executive functioning in the neutral condition from executive functioning in the distress
condition.
[2] [2] Within-subject changes in risk behavior were calculated by subtracting risk behavior in
the neutral condition from risk behavior in the distress condition.

then regressing the dependent variable change score on the mediator change score. A mediated effect is present if the mediator change score significantly predicts the dependent variable change score (Judd et al., 2001).

The second exploratory hypothesis was that the relationship between diagnosis and risk behavior in the distress condition would be mediated by group differences in baseline executive functioning and/or distressed executive functioning. To test this, we followed recommendations to formally test mediation suggested by Baron and Kenny (1986) and Judd and Kenny (1981). For this hypothesis, a series of hierarchical regression analysis was conducted; our independent variable was diagnosis, the dependent variable was risk behavior in the distress condition. The proposed mediators were baseline executive functioning and distressed executive functioning. The test for mediation required the following steps: 1) the independent variable must significantly predict dependent variable; 2) diagnosis must significantly predict the mediator; 3) the mediator must significantly predict the dependent variable and 4) when both the independent variable and the mediator are included in the same model to predict the dependent variable, the mediator must remain significant, and the effect of the independent variable must be reduced to non-significance. A fifth step was added in line with the recommendations of Mackinnon and colleagues (2002) who suggest providing a direct estimate of the effect size of the mediated effect by constructing confidence limits for the mediated effect using standard errors. If the confidence intervals include 0 then the mediated effect is not larger than what would be expected by chance.

Chapter 3: Results

3.1 Participants

A total of 44 completed the full three-session protocol. Analyses for the current study are based on a total sample of 30 participants ($n = 11$ BPD). Figure 1 provides an overview of participant recruitment and retention.

A series of *t*-tests and chi-square analyses were conducted on demographic and clinical variables to identify differences between groups. Findings are presented in Table 1. The two groups did not differ significantly in terms of any demographic characteristics (ps > .10) between the BPD and non-BPD group. Likewise, there were no significant differences in lifetime rates of substance use disorders or lifetime or current rates of anxiety disorders. There was a significant difference in lifetime rates of major depressive disorder in the BPD and non-BPD groups ($\chi^2 = 4.04$, $p < .04$) with a significantly higher rate observed in the BPD group. Lifetime rates of psychiatric treatment did not differ between the two groups ($p = .88$).

3.3 Manipulation Check

As a manipulation check for the affect inductions (i.e., distress and neutral scripts), negative affect (NA) and positive affect (PA) scores from the PANAS were examined for both conditions (Table 2). To characterize the effects of the scripts on affect 2 by 2 by 2 mixed factorial ANOVAs (separately for NA and PA), with condition (neutral, distress) and time (pre-script, post-script) as the within-subjects factors and diagnosis (BPD, non-BPD) as the between-subjects factor. For NA, findings revealed a significant condition by time interaction, $F(1, 28) = 6.13$, $p = .02$, partial $\eta^2 = .18$.

Analysis of simple effects revealed that this effect was driven by an increase in NA from pre- to post-script in the distress condition, with no such change in the neutral condition. Notably, there was a significant main effect of diagnosis on NA, $F(1, 28) = 5.14, p = .03$, $\eta^2 = .16$ such that participants with BPD were higher in NA overall. However, the 3-way interaction of condition by time by diagnosis was not significant, $F(1, 28) = 1.26, p = .27$, partial $\eta^2 = .03$, suggesting that participants with and without BPD experienced comparable increases in NA from pre-script to post-script in the distress condition.

This analysis was repeated with PA as the dependent variable. Results again indicated a significant condition by time interaction, $F(1, 28) = 6.78, p < .02, \eta^2 = .20$). Follow-up analyses revealed that ratings of PA decreased from pre- to post-script in the distress condition, but did not change from pre- to post-script in the neutral condition. The three-way interaction of condition by time by diagnosis was not significant for PA.

Taken together, findings suggest that the distress manipulation increased subjective NA and decreased subjective PA, producing similar changes for both groups from pre- to post- script. The neutral imagery script was not associated with significant changes in PA or NA for either group, suggesting that it is appropriate to consider this a "neutral" mood manipulation.

3.2 Examination of Potential Covariates

Prior to conducting the primary data analyses, preliminary analyses were conducted to examine the effect of demographic (e.g., age, racial background) and clinical factors (e.g., lifetime rates of mood, anxiety and substance use disorders) on variables of interest, to identify potential covariates. Age, racial/ethnic background, and education were not significantly associated with performance on BART or WCST.

Similarly, history of psychiatric treatment and lifetime diagnoses of mood, anxiety and substance use disorders were not associated with performance on BART (neutral or distressed) or WCST variables of interest. Therefore, these variables were not included as covariates in subsequent analyses.

In addition, we evaluated condition order (i.e., whether the participant received the neutral or distress condition first) as a potential covariate. Although order was randomly assigned, 64% of the total sample received the neutral condition first, compared to 34% who received the distress condition first. However, condition order was not significantly associated with the dependent variables ($ps > .24$), and was therefore not included in subsequent analyses.

3.3 Primary Aim

The primary hypothesis was that affect would moderate the relationship between diagnosis and risk behavior, such that the BPD group would not differ from controls in the neutral condition, but would exhibit a significant increase in risk behavior in the distress condition. To test this hypothesis, a 2 by 2 mixed factorial ANOVA was conducted predicting BART score. Condition (2; neutral, distress) was the within-subjects factor, diagnosis (2; BPD, non-BPD) was the between-subjects factor. Results revealed a significant interaction between condition and diagnosis, $F(1, 28) = 13.14; p = .001$, partial $\eta^2 = .32$ (see Figure 2 and Table 3). Notably, there was not a main effect of diagnosis, although this effect approached significance, $F(1, 28) = 3.32, p = .08$, partial $\eta^2 = .11$.

To probe the interaction of condition and diagnosis on BART score, oneway repeated measure ANOVAs were conducted separately for the two diagnostic groups.

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BART score was the dependent variable and condition was within-subject factor. The BPD group showed a significant effect of condition on BART, $F(1, 10) = 3.34$, $p = .03$, one-tailed, partial $\eta^2 = .30$, which reflects a significant increase in risk behavior in the distress condition compared to the neutral condition. On the other hand, the non-BPD group showed a significant decrease in risk behavior in the distress condition compared to the neutral condition, $F(1, 18) = 9.49$, $p = .006$, partial $\eta^2 = .35$).

To test the hypothesis that the groups would not differ in terms of risk behavior in the neutral condition, but would differ significantly in the distress condition, we conducted univariate ANOVAs comparing group differences in BART scores in the two conditions. The two groups did not differ in the neutral condition, $F(1, 28) = .98$, $p = .33$, however, they were significantly different in the distress condition, $F(1, 28) = 6.68$, $p = .015$.

3.4. Secondary Aim

To test our hypotheses related to group differences in executive functioning at baseline, and change in executive functioning as a function of affect, a 3x2 mixed factorial ANOVAs was conducted. The within-subjects factor was condition (baseline, neutral and distress) [3] and the between-subjects factor was diagnosis (BPD, non-BPD). Results revealed a significant main effect of diagnosis on executive functioning, $F(2, 27) = 7.15$, $p = .012$, partial $\eta^2 = .20$. Unexpectedly, the BPD group exhibited significantly *better* executive functioning than the non-BPD group, across conditions. The effect of

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[3] The WCST was measured on three occasions to control for practice effects, as suggested by Collie et al. (2003).

condition on executive functioning was not significant, $F(2, 27) = 1.18$, $p = .32$, partial $\eta^2 = .04$).

3.5 Exploratory Analyses

The exploratory aim of this study was to evaluate the relationship of executive functioning and risk behavior. Specifically, we examined 1) within-subject change in executive functioning in relation to within-subject change in risk behavior[4]; and 2) group differences in executive functioning (baseline, distress) in relation to group differences in risk behavior in the distress condition. The present analyses focus on percent perseverative errors, as an index of global executive functioning (Barcelo & Nyhus, 2009).

Our first exploratory hypothesis was that the extent to which participants experienced *changes* in executive functioning would predict to the extent to which they exhibited *changes* in risk behavior. To test this effect, we planned to follow procedures outlined by Judd, Kenny and McClelland (2001) to test a time-varying mediator in a within-subject design. This procedure requires that two conditions be met. First, there must be an effect of condition on executive functioning. Second, the difference in executive functioning across conditions must predict the difference in risk taking across conditions. However, as outlined above, we failed to find a significant effect of condition on either measure of executive functioning, and therefore did not meet the first requirement to demonstrate mediation. Accordingly, it was not necessary to test the second condition to demonstrate mediation.

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[4] Given the limited power in this study, we did not conduct an omnibus test to detect interactions of diagnosis and the proposed mediators. Instead, we conducted two analyses to highlight specific relationships of interest *within* the BPD group.

However, given the exploratory nature of the current study, we examined the correlations of risk behavior change score and executive functioning change score (Table 5) with the hopes of identifying a potential neurocognitive correlate of distress-induced increases in risk behavior. Within the BPD group, change in BART and change in percent perseverative errors were modestly positively correlated, $r(11) = .22, p < .50$. This indicates that larger BART change scores, which reflect higher risk behavior in the distress condition compared to the neutral condition, correspond to larger perseverative error change scores, which reflect higher rates of error in the distress condition compared to the neutral condition. Although this relationship did not approach significance, the degree of association corresponds to a small effect size (Cohen, 1988), and may have been meaningful in a larger sample.

In addition, an examination of correlations within the BPD group revealed a surprisingly robust relationship between baseline executive functioning and risk behavior in the neutral condition, $r(10) = .70, p < .017$, and in the distress condition, $r(10) = .57, p < .078$. This suggests that, within the BPD group, poorer baseline executive functioning is associated with greater engagement in risk behavior, regardless of affect.

Our next exploratory aim was to examine whether group differences in risk behavior in the distress condition would be mediated by group differences in executive functioning at baseline (i.e., non-affective executive functioning) and in the distress condition (i.e., affective executive functioning). To test this, we conducted a hierarchical regression to formally test mediation (Baron and Kenny, 1986; Judd and Kenny, 1981). Diagnosis significantly predicted risk behavior in the distress condition, $\beta = .44, t(28) = 2.58, p = .015, R^2 = .16, F(1, 28) = 6.68, p = .015$, so our first condition for mediation

was met. Consistent with our previous analyses, there was a main effect of diagnosis on baseline executive functioning, $\beta = .36$, $t(29) = -2.07$, $p = .048$, such that diagnosis predicted significantly *better* baseline executive functioning in the BPD group. However, there was not a main effect of diagnosis on distressed executive functioning, $\beta = .27$, $t(28) = -1.46$, $p = .16$. Therefore, the next condition for mediation was tested for baseline executive functioning only. To test the third condition for mediation, we regressed risk behavior in the distress condition on baseline executive functioning, however, this step was not significant ($\beta = .08$, $t(29) = .43$, $p = .67$). Therefore, findings did not support affective or non-affective functioning as a mediator of the relationship between diagnostic group and risk behavior when distressed.

Chapter 4: Discussion

4.1 Summary of Main Findings

The current study examined the effect of induced distress on a behavioral measure of risk behavior and a test of executive functioning in a community sample of women with and without BPD. Previous clinical research that has established distress as a critical antecedent of engagement in risk behavior among individuals with BPD (Linehan, 1993; Brown et al., 2002; Chapman, Gratz & Brown, 2006), however, many of these studies have been limited in scope by their use of retrospective self-report methods. In contrast, a small number of studies have examined risk behavior in BPD using laboratory-based behavioral assessment (Coffey et al., 2010; McCloskey et al., 2009), but this work has not accounted for the role of negative affect in precipitating risk behavior in this clinical population. The current study builds upon earlier research by comparing risk behavior in neutral mood to risk behavior in the context of induced distress using a behavioral risk task. Within this novel experimental framework, it also became possible to test a neurocognitive model of risk behavior, to identify executive functioning variables that might explain the relationships among distress and risk behavior among individuals with and without BPD. To do this, we examined within-subject changes in risk behavior and executive functioning, as well as group differences in risk behavior and executive functioning.

In support of the primary study hypothesis, affect was found to moderate the relationship of diagnosis and engagement of risk behavior. Specifically, participants with BPD showed greater risk behavior in the distress condition than in the neutral condition, whereas participants without BPD showed less risk behavior in the distress condition than

in the neutral condition. Furthermore, the two groups did not differ in risk behavior in the neutral condition, but the BPD group exhibited significantly greater risk behavior than controls in the distress condition. Taken together, results suggest that the BPD group responded to distress by engaging in risk behavior, whereas controls responded to distress by engaging in less risk behavior.

Our results are consistent with clinical research, which implicate distress-induced risk behavior as a central characteristic of BPD (APA, 2000; Sansone, 1998; Selby, Anestis, Blender & Joiner, 2008; Levine, Marziali & Hood, 1997; Gratz et a., 2006; Bornovalova, Gratz, Daughters, Nick, Delaney-Brumsey, Lynch et al., 2008; Bornovalova, Matusiewicz & Riggs, *in press*). Theoretical accounts of risk behavior in BPD suggest a number of factors that may explain the effect of distress on engagement in risk behavior among individuals with BPD, as well as the factors that may account for distinct patterns of risk behavior observed among individuals with BPD, as compared to controls (Linehan, 1993; Selby & Joiner, 2009; Chapman et al., 2006; Zanarini et al., 2003; Allen, 1995; Depue & Lenzenweger, 2001). The experimental paradigm presented here may be useful to test any number of hypotheses about the factors underlying these relationships. The current study focused on neurocognitive mechanisms that might explain the effect of distress on risk behavior engagement in BPD, and account for group differences (BPD, non-BPD) in distress-induced risk behavior.

As noted above, we identified a significant effect of affect on risk behavior among participants with BPD. Therefore, our first set of exploratory analyses examined whether distress-induced changes in risk behavior could be explained by corresponding changes in executive functioning. However, we failed to find a significant effect of affect on

executive functioning, so we did not proceed with a formal test of mediation (Judd, Kenny & McClelland, 2001). However, as a result of the small sample, the observed power to detect a main effect of affect on executive functioning was low, so we also examined associations among executive functioning variables and risk behavior to identify potential relationships within the BPD group for exploration in future studies. Two findings warrant mention. First, there was a modest, non-significant relationship between change in risk behavior and change in executive functioning ($r = .22$). This suggests that distress-induced deterioration in executive functioning was modestly associated with distress-induced increases in risk behavior. Of course, given the non-significant association between these variables, the relationship must not be over-interpreted, however, the observed relationship is consistent with the predictions of our theoretical model. Although the current findings do not provide support for executive functioning as a mediator of the effect of affect on risk behavior among participants with BPD, further research is warranted to determine the nature of the relationship between distress-induced decrements in executive functioning and distress-related increases in risk behavior.

In addition, inspection of our correlation table revealed an unexpectedly robust correlation between executive functioning at baseline and subsequent engagement in risk behavior in the neutral condition ($r = .70$) and the distress condition ($r = .57$) within the BPD group. Results suggest that, among participants with BPD, poorer executive functioning at baseline (i.e., nonaffective deficits) are associated with engagement in risk behavior, regardless of affective state. These findings are consistent with previous work conducted in healthy subjects, which has found that individuals with lower executive

functioning engage in more under-controlled behavior than those high in executive functioning (Ochsner & Gross, 2005; Yarkoni & Braver, 2010; Brand et al., 2006). That is, individuals with higher executive functioning are better able to act in accordance with their long-term goals, whereas those with poorer executive functioning are more likely to behave in accordance with their immediate impulses and desires (Hofmann, Gschwendner, Friese, Wiers & Schmitt, 2008). It has been suggested that this occurs because individuals with poor executive functioning are less able to keep two simultaneous goals active in working memory, which leads them to pursue whichever goal is active in that moment (Strack & Deutsch, 2004). Although preliminary, these findings suggest that individual differences in non-affective executive functioning may have important implications for understanding engagement in risk behavior or individuals with BPD.

Our next set of analyses focused on neurocognitive factors associated with group differences in risk behavior in the distress condition. That is, we attempted to understand whether the relationship between diagnosis and distress-induced risk behavior could be explained by executive functioning deficits, either at baseline or when distressed. Results did not support the executive functioning variables as mediators of the relationship between diagnosis and distress-induced risk behavior. However, unexpectedly, the BPD group had a significantly lower rate of perseverative errors than the non-BPD group at baseline (BPD: 17%; non-BPD: 30%), and a marginally lower rate in the distress condition (BPD: 22%; non-BPD: 32%). This finding was surprising, as some studies have reported higher rates of perseverative errors among individuals with BPD, while others have reported an absence of group differences (Lenzenweger et al., 2004; Fertuck et al.,

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2006). To our knowledge this is the first study to find superior executive functioning among individuals with BPD. Furthermore, it is noteworthy that the groups did not differ significantly on any other performance indices (these findings were not presented because they were not directly relevant to the proposed analyses). Thus, findings suggest that the non-BPD group had a specific deficit in set-shifting relative to the BPD group, which could not be explained by demographic (e.g., age or education) or clinical variables measured in this study (Boone et al., 1993; Heaton et al., 1993; Compton et al., 1997). Additional work is needed to replicate this finding, and identify the sample characteristics or contexts in which individuals with BPD perform better than those without BPD.

In summary, the current study used two complementary approaches to identify neurocognitive mediators of the relationship between affect and risk behavior among individuals with and without BPD. First, we examined within-subject variability in executive functioning within the BPD group to determine whether intra-individual fluctuations correspond to distress-induced changes in risk behavior. Then, we examined group differences in executive functioning in relation to distress-induced risk behavior in the BPD and non-BPD groups. Overall, we did not find evidence that neurocognitive factors mediated any of the relationships of interest. Nonetheless, preliminary findings from the current study suggest the potential utility of these complementary approaches in understanding risk behavior in BPD. Future research is warranted to further explore the relationship of neurocognitive factors to risk behavior and other BPD symptom domains (Fertuck et al., 2006; Lenzenweger et al., 2004).

<u>*4.2 Limitations and Future Directions*</u>

   Findings of the current study should be interpreted in light of its limitations.

4.2.1 Sampling Considerations

   A clear limitation of the current study is its modest sample size. In the initial

power analysis, it was determined that a sample of 80 participants would provide

adequate power to detect the presence of a between-group moderating variable and a

within-group mediating variable. Unfortunately, it was not possible to recruit a sample of

this size within the proposed timeframe. Despite low statistical power, the current sample

was large enough to detect an interaction of diagnosis and condition in predicting risk

behavior, however, both power and measurement issues precluded was not sufficient to

test a neurocognitive mediator of this effect.

   An additional limitation of the current study concerns the generalizability of

findings to other BPD samples. With regard to demographic characteristics, a number of

choices were made to limit within group variability, while maintaining external validity.

For instance, rates of BPD are equivalent among men in women in community samples

(Grant et al., 2006), suggesting the importance of studying emotional responding and risk

processes in both men and women. However, findings suggest that men tend to be riskier

than women on behavioral risk measures (Hunt, Hopko, Bare, Lejuez & Robinson, 2005;

Borghams, Golsteyn & Meijers, 2009; d'Acremont & Van der Linden, 2006), introducing

a possible source of variance unrelated to either diagnosis or the experimental

manipulation. Although we considered examining gender as a covariate, or even as a

moderator, we ultimately decided to focus our recruitment efforts on women given

previous difficulties recruiting a substantial number of men with BPD. Furthermore, we

limited our sample to women under the age of 35, in light of findings that the risky, self-destructive behavior characteristic of BPD peaks between the ages of 15 and 40 (Welch, 2001 Suyemoto, 1998; Stevenson, Meares & Comerford, 2003; Jarvis, Ferrence, Johnson & Whitehead, 1976). As a result, our findings do not necessarily generalize to men or older women with BPD, although the current sample does reflect a clinically meaningful subset of individuals with BPD.

Our next set of choices related to issues surrounding psychiatric comorbidity. Given high rates of co-occurring psychiatric disorders in BPD samples (Stein et al., 2007; Bender et al., 2001; Zanarini et al., 1998) we were concerned excluding these participants would reduce external validity, however, a number of psychiatric disorders are associated with impairment in neurocognitive functioning (Elliot et al., 1996; Schatzburg et al., 2000; Merriam et al., 1999), which complicate interpretation of findings (i.e., whether observed deficits result from BPD or impairment related to other psychiatric disorders; Sprock et al., 2001). Therefore, excluded individuals with current (i.e., past month) major depressive disorder and current (i.e., past year) substance dependence as well as individuals with history of bipolar I/II disorder or psychotic symptoms in light of evidence of persistent neurocognitive impairment associated with these disorders (Porter et al., 2003; Moritz et al., 2002; Degl'Innocenti et al., 2007; Sax et al., 1999; Clark et al., 2002; Nieuwenstein et al., 2001; Krabbendam et al., 2000; Goldberg et al., 1993). We chose to include individuals with remitted major depression and substance dependence as findings suggest that neurocognitive dysfunction associated with these disorders subsides within weeks to months (Miller, 1985; Bates et al., 2005; Austin, Goodwin & Mitchell, 2001; Wilder-Willis et al., 2001). As a result of these exclusion criteria, our BPD

participants may have been somewhat less clinically severe than other BPD samples. Indeed, with the exception of substance use disorders, our BPD group reported substantially lower lifetime rates of psychopathology than what is typically reported in population-based studies of BPD (Grant et al., 2006). Findings from this community sample should not be considered representative of clinical BPD populations more generally. Additional research is needed to determine how psychiatric comorbidity influences emotional responding, cognitive functioning and risk behavior in BPD.

4.2.2. Measurement Considerations

In addition to issues related to the small sample size and low power, measurement issues may account for the lack of findings with regard to executive functioning. Although we had a number of reasons for choosing this measure of executive functioning, including consistency with our theoretical model and precedent in earlier research (Barcelo & Nyhus, 2009 Fertuck et al., 2006; Ruocco, 2006), the WCST may not have been the optimal measure for assessing executive functioning in this study. The WCST is limited by of relatively low test-retest stability and significant practice effects over multiple administrations (Spreen & Strauss, 1997; Paolo et al., 1996). Although we attempted to control for practice effects experimentally (Collie et al., 2003), the low test-retest stability of WCST makes it difficult to determine what effects are due to the experimental manipulation, and which are due to measurement error. For example, because test-retest instability is high, it has been suggested that a reliable deterioration in performance would require an increase of 21 perseverative errors from baseline (Herman et al., 1996). In our sample, this corresponds to a 3-fold increase in perseverative errors. Thus, the WCST may not have been sensitive enough to detect subtle affect-related

changes in cognitive functioning. In light of this issue, our failure to identify distress-induced changes in executive functioning is not surprising.

A related concern is that set shifting ability (as measured by perseverative errors on WCST) is not an ideal operationalization of cognitive control, the construct we intended to assess. Recent research suggests that cognitive control may be better-assessed by specific measures of working memory capacity (Botvinick, Braver, Barch, Carter & Cohen, 2001; Derrfuss, Brass & von Cramon, 2004; McDonald, Cohen, Stenger & Carver, 2000; Baddeley & Logie, 1999), rather than by global indicators of prefrontal functioning such as WCST. Conceptually, low working memory capacity may interfere with an individual's ability represent two simultaneous goals in working memory (e.g., the goal of alleviating hunger vs. the goal of remaining slim; the goal of emotion regulation vs. the goal of sobriety). Indeed, individual differences and experimentally induced fluctuations in working memory capacity both have been shown to correspond to increased engagement in dysregulated behavior among healthy participants (e.g., Robinson, Schmeichel & Inzlicht, 2010; Kopetz et al., *in preparation*). Therefore, future research on neurocognition in BPD should employ well-established measures of working memory capacity, which have been shown to reliably detect modest changes over multiple administrations (e.g., the N-back task; Xu, Mendrek, Cohen, Monterosso, Simon et al., 2006; Fitzsimons & Bargh, 2004).

In addition, it is noteworthy that our approach to measure affective neurocognition was somewhat atypical. Our study examined processing of neutral stimuli in an affectively aroused state, whereas previous work has assessed processing of emotionally-valenced cues (e.g., abandonment words), without considering affect more

generally (e.g., Yen et al., 2002; Fertuck et al., 2006). Although both approaches have

shown that negative affect or negative emotion cues are associated with decrements in

executive functioning (compared to neutral affect/cues; Fertuck et al., 2006; Chepenik,

Cornew & Farah, 2006), our approach may not have been sufficient to induce significant

changes, particularly using the WCST. This task involves lower order cognitive processes

(e.g., working memory, behavioral inhibition) as well as higher-order cognitive processes

(e.g., conceptual reasoning), which may be affected differently by emotion (Nyhus &

Barcelo, 2009). Therefore, future research should employ more conventional

experimental paradigms for assessing affective neurocognition, and should use a

combination of tasks that assess higher- and lower-order executive functions.

4.2.3. Experimental Considerations

Finally, although the mood manipulation was sufficient to produce the

hypothesized changes in risk behavior in the BPD group, several limitations warrant

mention. First, despite the significant change in negative affect from pre- to post-distress

script, the distress induction produced only mild levels of distress. For both groups, the

negative affect composite score corresponded to feeling "a little" distressed following the

distress manipulation. These effects are comparable to other studies that used

personalized distress imagery scripts with participants with BPD (e.g., Schmal, Elzinga,

Ebner, Simms, Sanislow et al., 2004; Kraus, Valerius, Seifritz, Ruf, Bremner et al.,

2010), however, these relatively modest effects may not be sufficient to produce

meaningful changes executive functioning (Williams, Suchy & Rau, 2009; Pessoa, 2009).

One possible explanation for the modest observed effect is that the study protocol

may have inadvertently included an opportunity for participants to regulate their

emotions before engaging in the tasks. As a manipulation check, participants completed a questionnaire that asked them to rate the intensity of 20 different emotion words immediately after hearing the distress script (PANAS). This questionnaire, which asked participants to observe, differentiate and label their emotions, may have functioned as an exercise in emotion regulation (Gratz & Tull, 2010). While the emotion regulating properties of the PANAS remain an empirical question, it is possible that this aspect of the design made the distress induction less effective, which limited our ability to detect affect-linked changes in risk behavior and executive functioning. Future research may consider dispensing with subjective mood ratings in favor of objective measures of emotional responding (e.g., psychophysiological measures such as heart rate variability, blood pressure, skin conductance; Schmal et al., 2004).

Another potential limitation of our affect manipulation was that it aimed to increase distress generally, rather than targeting specific emotional states that are known to be associated with engagement in risk behavior. For instance, recent research suggests that interpersonal distress (e.g., feelings of abandonment, disapproval or rejection) has a unique association with engagement in risk behavior for those with BPD (Stanley, 2010; Sadikaj, Russel, Moskowitz & Paris, 2010). Thus, future research may employ affect manipulations that specifically target stressful interpersonal experiences instead of other distressing events. Along these lines, several studies of emotional responding in BPD have used personalized abandonment scripts (e.g., Schmal et al., 2003, 2004), however evidence does not suggest that interpersonally-oriented distress scripts are *more* effective than general distress scripts for individuals with BPD (e.g., Schmal et al., 2004). Another alternative may be affect manipulations that are not script-based but are interpersonal in

nature, such as Cyberball (Williams, 2006), a computerized ball-tossing game in which

the participant is systematically excluded from a group interaction. Addressing these

methodological concerns related to the affect induction procedure may provide a more

robust test of the effect of affect on risk behavior and executive functioning.

4.2.4. Future Directions

The preceding sections we identified a number of methodological factors that may

have interfered with our ability to detect the presence of mediating variables. However, in

the absence of conclusive findings to support the neurocognitive model of risk behavior,

it is also appropriate to consider alternative theories of risk behavior in BPD.

Our findings regarding affect and risk behavior are consistent with prominent

behavioral theories of risk behavior in BPD, which emphasize that the motivational basis

of risk behavior is the regulation of negative affect (e.g., Linehan, 1993; Chapman et al.,

2006; Selby & Joiner, 2009). These theories suggest that individuals with BPD engage in

risk behavior because they experience intense negative affect, and have low ability or

willingness tolerance of distress, which leads them to engage in behaviors that decrease

distress in the short-term, even though these behaviors may also lead to negative

consequences and interfere with other goals (Linehan, 1993; Gratz & Roemer, 2004).

Compared to individuals with BPD, those without BPD may experience less intense

negative affect, feel less need to reduce distress when it occurs, or have access to more

adaptive means of emotion regulation (Levine, Marziali & Hood, 1997; Gratz et a., 2006;

Bornovalova, Gratz, Daughters, Nick, Delaney-Brumsey, Lynch et al., 2008;

Bornovalova, Matusiewicz & Riggs, *in press*), and therefore engage in less risk behavior

(Sansone, Wiederman & Sansone, 1998). Any combination of these variables may have

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accounted for the pattern of findings that we observed, yet the current study was not designed to test these hypotheses. Future research could examine these hypotheses using the current experimental paradigm in combination with existing self-report, behavioral and physiological measures of emotion regulation (e.g., Watson et al., 1988; Lejuez et al., 2002; Gratz & Roemer, 2004).

A central tenet of emotion regulation theories of risk behavior is that risk behavior is maintained through negative reinforcement, yet there is there is considerable evidence that risk behavior is also maintained by positive reinforcement processes. As example, self-harm may function to reduce negative affect (e.g., feelings of tension, shame, sadness) or to escape an aversive situation (Brown et al., 2002; Nock & Prinstein, 2005). However, the same behavior may also be used to generate feelings (e.g., terminate dissociative states) or influence others' behavior (e.g., elicit support, increase compliance, or even as an act of self-validation (Brown et al., 2002). Positive and negative reinforcement functions of other risk behaviors (e.g., substance use, bulimic behavior, impulsive sexual behavior, suicidal behavior) have also been identified (Baker, McCarthy, Piper McCarthy, Majeskie & Fiore, 2004; Koob & Kreek, 2007; Wedig & Nock, 2010; Ikemoto & Panksepp, 1999). Furthermore, within individuals, different risk behaviors may be maintained by distinct motivational processes, and the function of any specific risk behavior may change over time (Brown et al., 2002). Accordingly, future should explore a combination of positive and negative reinforcement processes in attempting to model actual risk behavior, acknowledging that the functions of any particular behavior may be contextually-based.

Finally, our results indicate that the BPD and non-BPD groups exhibited different patterns of behavior in response to distress: the BPD group engaged in more risk behavior when distressed, while the non-BPD group showed less risk behavior. This finding is consistent with previous research, which suggests that healthy female participants engage in less risk behavior in the context of distress than under no-stress conditions (Lighthall, Mather & Gorlick, 2009; Preston, Tansfield, Bechanan & Bechara, 2007). The pattern of behavior observed in the BPD group is noteworthy, because not only did they show an increase in the distress condition, but they actually showed the opposite behavioral pattern than what is observed in healthy samples. The factors that contribute to these distinct patterns of responding remain unclear. One possibility is that the BPD group may have experienced an intense desire to decrease distress, and so may have attempted to repair their mood by seeking rewards. On the other hand, non-BPD may have been motivated to avoid a further deterioration in mood, and behaved more conservatively to avoid incurring losses. Continued research is warranted to determine the motivational and neurocognitive processes involved in the tendency to become risk seeking versus risk averse in the context of distress.

4.3. Conclusions

The application of neuropsychological and biobehavioral findings to clinically relevant outcomes has been identified as a priority in research on BPD (Bohus, 2010). Despite a number of limitations, the current study represents a unique contribution to the empirical literature on risk behavior in BPD. This study extends previous research by inducing affect and behavior change in real-time, which permits examination of a wider

range of potential processes associated with risk behavior than has been allowed by retrospective self-report studies. Along these lines, an exploratory aim of this study was to examine affective deficits in executive functioning as they related to engagement in distress-induced risk behavior. Although we did not find support for our proposed neurocognitive mediators, our results indicate several avenues for future research. In addition, the experimental paradigm used in this study may have applications in future research examining the relationship of neurocognitive vulnerabilities to clinically important outcomes among individuals with BPD.

Figure i. Flowchart of study recruitment, retention and inclusion in analyses.
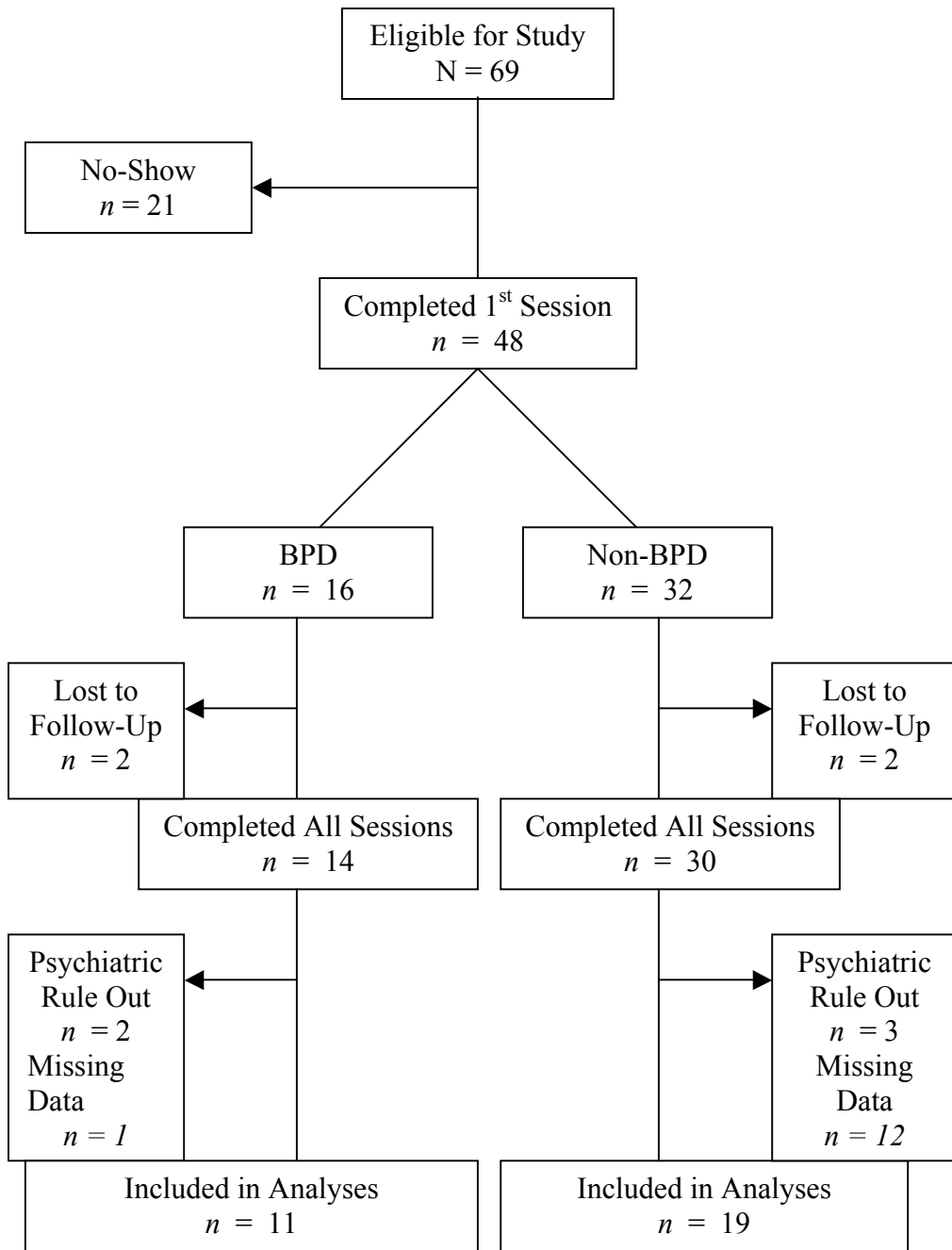
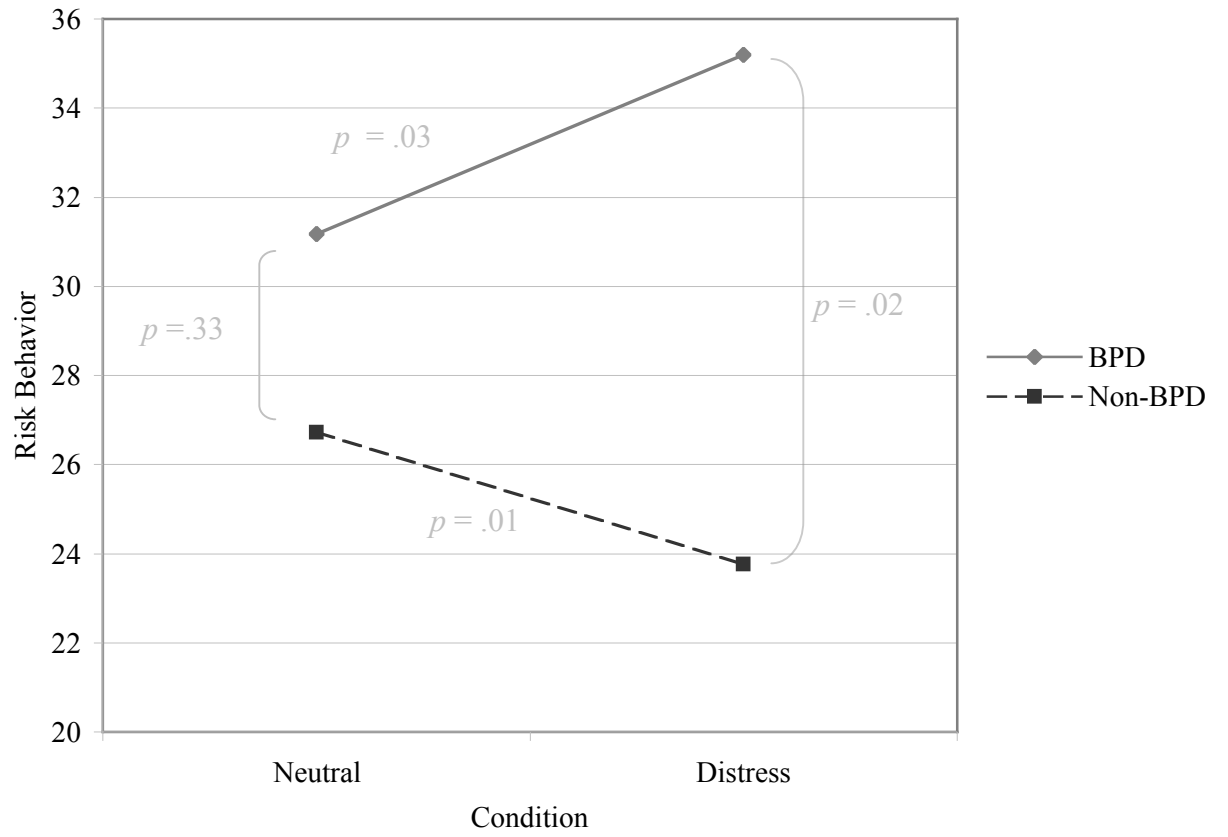Figure ii. Risk behavior as a function of condition and diagnosis.

Table i. Demographic and Clinical Characteristics of Participants by Diagnostic Group

	BPD (n = 11)	Non-BPD (n = 19)
Age	$M = 21.5 \pm 3.6$	$M = 22.6 \pm 4.6$
Racial/ethnic background		
White	9.1%	36.8%
Black or African American	54.5%	42.3%
Asian or Asian American	9.1%	10.5
Other	27.3%	10.4%
Highest Educational Attainment		
High school	9.1%	10.5%
Some college	72.7%	63.2%
College	0%	15.8%
Masters or doctoral degree	5.5%	10.6%
Psychiatric Treatment (Lifetime)	27.3%	31.6%
Axis I disorders		
Mood disorder (Lifetime)*	63.6%	26.3%
Substance use disorder (Lifetime)	27.3%	10.5%
Anxiety disorder (Lifetime)	36.3%	15.8%
Anxiety disorder (Current)	33.3%	6.2%

Note. BPD = borderline personality disorder

* p < .05

Table ii. Ratings of Negative Affect and Positive Affect Before and After the Affect

Manipulation in Each Condition, as a Function of Diagnosis.

	Condition	Time	BPD (N = 11) M (SD)	Non-BPD (N = 19) M (SD)
Negative Affect	Neutral	Pre	1.66 (.80)	1.22 (.29)
		Post	1.75 (.95)	1.39 (.65)
	Distress	Pre	1.53 (.77)	1.25 (.37)
		Post	2.35 (1.16)	1.69 (.69)
Positive Affect	Neutral	Pre	2.42 (.65)	2.77 (.74)
		Post	2.47 (.75)	2.41 (.75)
	Distress	Pre	2.87 (1.05)	2.86 (.88)
		Post	2.33 (1.01)	2.32 (1.01)

Note. BPD = borderline personality disorder

* $p < .05$

Table iii. BART Scores by Diagnostic Group and Condition.

	BPD (n = 11)	Non-BPD (n = 19)
	_M (SD)_	_M (SD)_
BART: Neutral	31.17 (11.8)	26.72 (11.9)
BART: Distress	35.19 (12.9)*	23.77 (10.95)

Note. BART = Balloon Analogue Risk Task adjusted average pumps per balloon; BPD = borderline personality disorder

* p < .01

Table iv. Descriptive Data for Executive Functioning as a Function of Diagnostic Group and Condition.

	BPD (n = 11)	Non-BPD (n = 19)
	M (SD)	*M (SD)*
Persev. Errors (%): Baseline	16.7 (13.9)	30.0 (18.4)
Persev. Errors (%): Neutral	22.9 (15.4)	34.4 (13.2)
Persev. Errors (%): Distress	22.5 (13.8)	32.2 (17.7)

Table v. Point Biserial and Pearson Correlations Between Diagnostic Status, Risk Behavior and Executive Functioning in Participants with BPD (below the diagonal) and without BPD (above the diagonal)

	1	2	3	4	5	6	7
Diagnostic status (combined)	.18	.44*	.56**	-.39*	-.41*	-.27	.05
1. BART: Neutral		.94**	-.40	.15	.25	.20	.01
2. BART: Distress	.87**		-.05	.14	.27	.08	-.11
3. ΔBART	-.10	.40		.05	.01	-.34	-.30
4. Persev. Errors (%): Baseline	.71*	.57	-.19		.38	.38	.04
5. Persev. Errors (%): Neutral	.22	.17	-.06	.21		.31	-.45
6. Persev. Errors (%): Distress	-.05	-.02	.04	.43	.60		.68**
7. ΔPersev. Errors (%)	-.36	-.22	.22	.14	-.55	.32	

Note: Correlations for participants with BPD are presented below the diagonal, and correlations for controls are above the diagonal. BART = Balloon Analogue Risk Task adjusted average pumps per balloon. ΔBART = the difference between BART score in the distress condition and in the neutral condition, with higher scores reflecting greater risk behavior in the distress condition. Persev. Errors (%) = Percentage perseverative errors out of total errors. ΔPersev. Errors (%) = the difference between rate of perseverative errors in the distress condition and in the neutral condition, with higher scores reflecting more errors in the distress condition
* $p < .05$
** $p < .01$

Appendices

You will soon hear a situation being described to you. Your task is to close your eyes and imagine yourself in the situation being described, 'as if' it were happening right now. Allow yourself to become completely involved in the situation, by involving your mind and body in actually doing what is being described. Continue imagining until you are asked to stop.

You are taking a shower when suddenly you realize that you are washing off the last remnants of your relationship with your ex-boyfriend. You get choked up as you think about the fact that you won't even be able to smell him anymore. There's a lump in your throat and you feel like crying. You can't stop missing him so much. There's a knot in your stomach. You two were perfect together and now it's all over. Your chest feels tight and it gets harder to breath. He might as well be on another planet because you're never going to see or talk to him again. Your breathing becomes fast and labored. You feel like Juliette, like everything that should have been right got ruined by circumstance. Your eyes burn with tears. Mormonism took him away from you. You could have been together except he couldn't convert for you and you couldn't convert for him. Your whole body is shaking. You're afraid to be alone now. It hurts to be alive. You can't stop the thoughts from coming. Your heart keeps beating faster. You're remembering how you two had to sneak around. You hated having to do that. You're remembering how you couldn't tell him you love him because you knew he wouldn't say "I love you" back. Now you're gasping for air. You're remembering how he kissed another girl. You felt like an idiot. Your fists clench. You think you might have been able to prevent everything

if only you had insisted on making the relationship public. But you feared his refusal. His parents never liked you because you're atheist. He tried to blame you for what he called "leading him astray". You want to scream or hit someone. The end was inevitable and now it's real. You knew all along that he was going to leave you. There's nothing you can do about it. But it still hurts so much. You grit your teeth. Being with him was like a drug. You can't stand it. You hate how nothing is more important than his religion. You don't understand why he won't break free from it. His sister did and it was fine, so why won't he? You know he has doubts about his religion, so why did he choose to leave you? You feel like you don't matter to him at all. He could always make you feel better, but now he's gone and the hurt won't stop. You feel empty and drained except for the intense pain deep inside.

Stop imagining now

Open your eyes and stop imagining

Now please complete the questionnaire on the top shelf to your right. When you are finished open the door.

You will soon hear a situation being described to you. Your task is to close your eyes and imagine yourself in the situation being described, 'as if' it were happening right now. Allow yourself to become completely involved in the situation, by involving your mind and body in actually doing what is being described. Continue imagining until you are asked to stop.

You are sitting on the beach on a bright summer day. You breathe in deeply as you notice a red kite against the cloudless blue sky. Your eyes trace the path of the kite as it whips up and down in spirals with the wind. The sun glares at you from behind the kite and makes the white sandy beach sparkle with reflection. You tense the muscles in your forehead and around your eyes, squinting to block out the bright sunlight. You follow with your eyes the long white tail, which dances from side to side beneath the soaring kite. You take in a few deep breaths of the fresh ocean air, noticing the smells of the fish and the salt water. The warm sun beats down against your skin and a light gentle breeze blows over you. You listen to the soothing sound of the ocean waves, roaring and splashing as it comes onto the sand, and quiet as the water goes back out to sea. You relax the muscles in your arms, back and legs as you lay back on the sand, feeling the soft fine granules of sand between your toes and fingers. The tension from your body goes down and you feel comfortable and at ease. Your breathing slows down and the worry thoughts seem to fade away. There is a sense of lightness and you want to hold time and capture this moment. A feeling of peace overcomes you.

Stop imagining now

Open your eyes and stop imagining

Now please complete the questionnaire on the top shelf to your right. When you are

finished open the door.

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