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The Rewarding Nature of Anger Rumination in Borderline Personality Disorder: An fMRI Investigation

Jessica R. Peters
University of Kentucky, jrpeters@gmail.com

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Jessica R. Peters, Student

Dr. Ruth A. Baer, Major Professor

Dr. David T. Berry, Director of Graduate Studies

THE REWARDING NATURE OF ANGER RUMINATION IN BORDERLINE
PERSONALITY DISORDER: AN FMRI INVESTIGATION

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DISSERTATION

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A dissertation submitted in partial fulfillment of the
requirements for the degree of Doctor of Philosophy
in the College of Arts and Sciences at the University of Kentucky

By
Jessica Rachael Peters

Lexington, Kentucky

Director: Ruth A. Baer, Ph.D., Professor of Psychology

Lexington, Kentucky

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ABSTRACT OF DISSERTATION

THE REWARDING NATURE OF ANGER RUMINATION IN BORDERLINE
PERSONALITY DISORDER: AN FMRI INVESTIGATION

Anger rumination, or persistently dwelling on feelings of anger, is associated with
borderline personality disorder (BPD) and related features, such as aggressive behavior
and cognitive distortions. To develop more effective treatments, it is crucial to understand
why individuals with BPD engage in anger rumination despite its negative outcomes. The
activation of energy associated with anger, as well as feelings of justification and
validation, may be experienced in the short-term as rewarding. This may prevent
individuals with BPD from attempting to reduce their rumination.

Functional magnetic resonance imaging (fMRI) and behavioral methods were utilized to
examine this theory in a sample of women diagnosed with BPD (n=13) and healthy
controls (n=15). In an initial session, all participants were an administered a diagnostic
interview for BPD, as well a series of self-report measures. In a second session, all
participants completed an essay-writing task prior to the fMRI scan. All participants were
provided with identical, highly critical feedback about their essays from a supposed essay
evaluator. In response to this interpersonal provocation, participants with BPD
demonstrated higher activation in brain regions associated with self-conscious reactivity
to errors (insula, ventrolateral prefrontal cortex). Subsequent directed provocation-
focused thought, compared to neutral-focused thought, produced greater activation in
regions previously associated with anger rumination (dorsomedial prefrontal cortex,
lateral orbitofrontal cortex) across groups. As hypothesized, anger rumination, relative to
neutral-focused thought, produced greater activation in brain regions associated with
reward and pleasure (nucleus accumbens) for the BPD group only. No significant
differences were observed for self-focused thought. Following the directed rumination
task, participants completed a competitive reaction time task that provides an opportunity
for participants to act aggressively, supposedly against their essay evaluator. The BPD
group demonstrated significantly higher levels of aggressive behavior; however, no
significant group differences emerged in neural functioning during the task. These
findings suggest that anger rumination may be positively reinforcing for individuals with
BPD, which has implications for treatment approaches.

Jessica Rachael Peters

February 23, 2015

THE REWARDING NATURE OF ANGER RUMINATION IN BORDERLINE
PERSONALITY DISORDER: AN FMRI INVESTIGATION

By

Jessica Rachael Peters

Ruth A. Baer, Ph.D.
Director of Dissertation

David T. Berry, Ph.D.
Director of Graduate Studies

February 23, 2015
Date

"Anger is my comfort emotion"

Quoted with permission from a client. This dissertation is dedicated to my clients, who

have greatly shaped my understanding of anger and the theory examined here.

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Chapter One: Introduction

Borderline personality disorder (BPD) is characterized by affective instability, identity disturbances, problems in interpersonal relationships, and self-destructive impulsivity (American Psychiatric Association, 2013). Rumination, defined as repetitive, passive, unconstructive thinking about negative emotions and problems, may contribute to amplifying and maintaining these patterns of negative affect and dysfunctional behavior. Anger rumination in particular is associated with BPD features (Baer & Sauer, 2011; Peters, Eisenlohr-Moul, Upton, & Baer, 2013) and predicts characteristics of BPD, such as anger, aggression, and cognitive distortions (Bushman, Bonacci, Pedersen, Vasquez, & Miller, 2005; Peled & Moretti, 2009; Riva, Romero Lauro, Vergallito, DeWall, & Bushman, 2015). To develop more effective treatments, it is crucial to understand why individuals with BPD engage in anger rumination despite its negative outcomes. The current investigation will clarify central psychological processes contributing to BPD symptoms, with implications for new directions for BPD interventions.

Borderline Personality Disorder

BPD occurs in 1-4% of the general population. It is highly represented in psychiatric settings, with an estimate of 15% of inpatients meeting diagnostic criteria (Onoda et al., 2010; Widiger & Weissman, 1991). The majority of individuals who are diagnosed with BPD are female (American Psychiatric Association, 2013). Individuals with BPD demonstrate unstable, intense, and prolonged negative affect, including elevated levels of shame, anger, anxiety and depression. These difficulties in emotion regulation in BPD lead to a range of dysfunctional behaviors, including aggressive

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behavior, deliberate self-harm (such as cutting or burning), disordered eating, risky sex, and suicide (American Psychiatric Association, 2013). BPD has a 10% mortality rate by suicide (Paris & Zweig-Frank, 2001). Although several treatments have empirical support for their efficacy, many participants show only partial improvement, and more effective treatments are needed.

Rumination as a Factor in BPD

Rumination, or the tendency to think passively and repetitively about negative emotions, appears to be a major contributor to the difficulties in emotion and behavior regulation exhibited in BPD. Although many people assume that extended thinking about problems will lead to insight and solutions, rumination intensifies negative affect and reduces problem-solving ability. Anger rumination intensifies feelings of anger and leads to increases in displaced aggression (Bushman et al., 2005; Peled & Moretti, 2009). Anger rumination also facilitates the formation of distorted cognitions, in which negative beliefs and associations are incorporated into interpretations of ambiguous events (Rusting & Nolen-Hoeksema, 1998).

The outcomes of anger rumination, including increased anger, aggressive behavior, and distorted cognitions, are all characteristic of BPD. Studies support the theory that rumination, and anger rumination in particular, contribute to BPD features (see Baer, Peters, Eisenlohr-Moul, Geiger, & Sauer, 2012 for a review). Anger rumination shows large correlations with BPD features in several samples (Baer & Sauer, 2011; Peters et al., 2013; Peters, Geiger, Smart, & Baer, 2014). Other work has shown that anger rumination (but not depressive rumination) mediated the relationship between the general tendency to experience negative affect and BPD features (Baer & Sauer,

2011). Thus, anger rumination in particular is a dysfunctional strategy that represents a core component of BPD.

Function of Anger Rumination

Why do people engage in anger rumination, especially considering its negative consequences? A recently proposed explanation is that anger rumination may be a method of avoiding more aversive emotions and cognitions (Gardner & Moore, 2008). Ruminating on anger may reduce internally directed negative affect, such as shame, by focusing instead on external causes for distress, such as unfair situations and deplorable behavior of others. Shame proneness is common in BPD (Gratz, Rosenthal, Tull, & Lejuez, 2010; Rüsch et al., 2007; Schoenleber & Berenbaum, 2012); thus anger rumination may function to amplify anger as a preferred state to shame. While anger rumination successfully reduces the painful feelings of shame, it also contributes to the dysregulated behavior typical of BPD, such as aggression and interpersonal problems. In turn, these problems create conflict and turmoil in social relationships, triggering more feelings of shame and creating a vicious cycle. Consistent with this theory, self-reported anger rumination has been shown to mediate the relationship between shame-proneness and BPD features (Peters et al., 2014).

Anger is typically conceptualized as a negative emotion, but it also has immediate positive outcomes, such as increased energy and feeling justified. Most negative emotions induce avoidant behavior; however, like positive affect, anger increases approach motivation (C. Harmon-Jones, Schmeichel, Mennitt, & Harmon-Jones, 2011). Therefore, anger rumination may not only dampen BPD individuals' self-directed negative affect (negative reinforcement), but also provide them with feelings of

validation, empowerment, and pleasure (positive reinforcement). This rewarding effect of anger rumination may contribute to the difficulties with emotion regulation experienced individuals with BPD.

Use of Neuroimaging to Explore the Function of Anger Rumination

The present study explores this theory that anger rumination in response to interpersonal rejection and provocation is reinforcing for individuals with BPD, compared to control participants. Previous work on rumination in BPD largely relies on self-report and behavior laboratory tasks, both of which have limitations. People often lack access to information about their mental processes, and when asked to describe the motivation underlying their behavior, individuals tend to report what makes sense to them, rather than what necessarily happened (Nisbett & Wilson, 1977). The ability to recall emotional experiences may be particularly limited, especially as time passes (M. D. Robinson & Clore, 2002). It may be especially difficult for individuals with a disorder characterized by low awareness (Peters et al., 2013) to report accurately on their emotional and cognitive processes. This is the first study to use fMRI to better understand the neural correlates of anger rumination in BPD; by detecting activation in brain regions associated with the relevant cognitive processes, the approach complements the information provided by self-report methods of assessing cognitive processes, which can be subject to a variety of biases. A substantial affective neuroscience literature examines the neural correlates of social rejection and criticism, anger regulation and rumination, and reward processing. While little of this work is specific to BPD, it provides a context for understanding what neural patterns would be consistent with the hypothesized function of anger rumination in BPD.

Neural correlates of reactivity to social rejection and criticism. Social pain and rejection results in activation in neural regions involved in affective pain, including the dorsal anterior cingulate cortex (dACC) and the right ventrolateral prefrontal cortex (rVLPFC), as well as the anterior insula (Eisenberger, 2003; Eisenberger & Lieberman, 2004). The ACC generally functions as "neural alarm system" that responds to inconsistencies between stimuli and goals (Carter et al., 2000), and pain is one indicator of likely problems that triggers this ACC activation (Sawamoto et al., 2000). The dACC in particular is sensitive to affective distress, rather than sensory pain, and plays a role in the detection of social rejection (Eisenberger, 2003; Eisenberger & Lieberman, 2004). This activation has been shown to be specific to negative social feedback such as exclusion, not simply violation of expectancies in social interactions (Kawamoto, 2012).

While activation in the anterior insula has been demonstrated in response to social exclusion, the activation is not associated with self-reported distress (Eisenberger, 2003). However, one potential component of interpersonal rejection, particularly criticism, is that individuals may think they have done something wrong to provoke this response from others. The insula does appear to play a role in determining the salience of stimuli, including error detection and processing. Bilateral insula activation has been shown to occur in reaction to indicators of response-inhibition failure, which may represent processing of the significance of errors (Ramautar, Slagter, Kok, & Ridderinkhof, 2006). Further research implicates the left anterior insula specifically in consciousness of errors, with this region activating selectively in response to aware, versus unaware, error commission (Klein et al., 2007). The anterior insula may produce an orienting response

that generates autonomic reactivity and the potential to respond to the committed error (Ullsperger, Harsay, Wessel, & Ridderinkhof, 2010).

While the dACC and anterior insula are co-activated in response to social rejection in healthy controls, this pattern may be disrupted within BPD. An activation-likelihood-estimation meta-analysis showed that when processing negative emotions, individuals with BPD demonstrate hyperactivity in the right insular cortex, compared to controls (Ruocco, Amirthavasagam, Choi-Kain, & McMain, 2012). Studies examining the effects of psychological pain on the ACC in BPD show mixed results. Some samples of individuals with BPD demonstrate the expected increased ACC activation in response to negative emotion and social pain inductions (Koenigsberg et al., 2009; Niedtfeld et al., 2012; 2010); however, in others, individuals with BPD show *deactivation* in the ACC during abandonment memories (Schmahl et al., 2003) or personalized scripts of childhood abuse (Schmahl, Vermetten, Elzinga, & Bremner, 2004), as well as during physical pain perception (Schmahl & Bremner, 2006). The intense insula response to rejection and distress consistently observed in BPD may, at times, serve to trigger dissociation from pain (Ducasse, Courtet, & Olié, 2014). Individuals with BPD may, however, be less successful than controls in reducing painful emotions and related neural activation if consciously trying to distance themselves from social pain (Koenigsberg et al., 2009).

The rVLPFC, which co-activates with the dACC and insula in response to social exclusion, is associated with regulation of negative emotions, particularly the inhibition of pain (Wager, Davidson, Hughes, Lindquist, & Ochsner, 2008a). Increased activation in the rVLPFC has been linked to inhibition of the pain resulting from social exclusion, with

activation of this region negatively correlated with self-reported distress (Eisenberger, 2003; Kawamoto, 2012). Transcranial direct current stimulation (tDCS) of rVLPFC that amplified activation in this region prior to and during a social exclusion paradigm attenuated emotional reactivity to rejection, relative to sham stimulation (Riva et al., 2012), and reduced subsequent aggressive behavior (Riva et al., 2014). Conversely, inhibiting the rVLPFC with tDCS following social exclusion amplified the normative negative emotional response (Bushman et al., 2005; Peled & Moretti, 2009; Riva et al., 2015). Together, these findings suggest a key role for the rVLPFC in regulating painful reactivity to social rejection.

Several studies have examined BPD-relevant individual differences as moderators of regulatory neural responses to rejection. Low trait-level self-esteem predicted greater activation of the dACC in response to an experimental social exclusion paradigm, which correlated with higher levels of self-reported pain (Onoda et al., 2010). For these individuals with low-self esteem, this pain-related activation was positively associated with simultaneous activation of self-regulatory networks in the PFC, whereas participants with high self-esteem demonstrated a negative association between dACC and PFC activation. Strong efforts to regulate the pain of rejection may also result in subsequent self-regulatory deficits and emotionally-driven impulsive behavior; recruitment of the rVLPFC during a social exclusion paradigm was associated with greater impact of felt-rejection on alcohol cravings outside of the lab (Chester & DeWall, 2014).

Given these findings for individuals with difficulties with self-image and self-regulation, it seems likely that individuals with BPD both may have intense sensitivity to

rejection (heightened reactivity in the right anterior insula and possibly the dACC) and may engage in greater regulatory efforts (increased activation of the rVLPFC).

Neural correlates of anger, anger regulation, and anger rumination.
Numerous brain regions have been implicated in indirect experiences of anger, such as recalling angering life events or viewing angry faces, including the medial PFC (MPFC), the ventromedial PFC (vmPFC), the ACC, the posterior cingulate cortex (PCC), the lateral PFC (LPFC), and the thalamus, as shown through meta-analyses of these types of studies (Murphy, Nimmo-Smith, & Lawrence, 2003; Phan, Wager, Taylor, & Liberzon, 2002). Few studies, however, have examined responses in the scanner to angering events. Following unexpected interpersonal provocation, activation in the dACC was associated with subjective experiences of anger (Denson et al., 2009). This is consistent with the dACC's function as a "neural alarm system", described above.

Several neural mechanisms have been specifically linked to anger regulation. The orbitofrontal cortex (OFC) may play an important role in regulating anger generally. In a sample of healthy undergraduate men and women, recalling angry events while using assigned regulatory strategies, including reappraisal, analytical rumination, and anger rumination, involved OFC activation regardless of the strategy, although anger rumination resulted in more subsequent self-reported anger than the other two strategies (Fabiansson et al., 2012). Additionally, the extent of activation of the OFC when frustrated may relate to anger regulation abilities. In another sample of healthy adults, individuals who reported better control of anger demonstrate increased activation in the OFC compared to baseline when hearing the word "no," compared to individuals

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endorsing poor anger control, who demonstrated decreased OFC activation when hearing the word "no" compared to baseline (Alia-Klein et al., 2007).

Several studies have examined neural regulation of anger in BPD or clinical groups similar to BPD. Individuals with BPD, compared to healthy controls, demonstrated greater glucose metabolism during PET scans in the OFC and amygdala during provocation, whereas controls decreased metabolism in these regions. In contrast, controls demonstrated greater glucose metabolism in the anterior, dorsal, and mediolateral PFC (New et al., 2009). This, in combination with the previously discussed findings, suggests that compared to controls, individuals with BPD are experiencing more anger in response to provocation and engaging in some form of regulation strategy.

An additional study compared control participants with two clinical groups with major depressive disorder (MDD): one with anger episodes (a phenotype similar to BPD) and one without anger episodes (Dougherty et al., 2004). When the three groups were exposed to anger-related autobiographical scripts, the control group demonstrated greater levels of activation in left ventromedial prefrontal cortex (vmPFC) than the MDD with anger group, suggesting greater recruitment of regulation strategies. However, the MDD with anger group demonstrated a positive association between activation in the left vmPFC and the amygdala, whereas controls demonstrated a negative association and the MDD without anger group had no correlation. This study could reflect a difference in regulatory strategies, with the MDD plus anger group engaging in a cognitive strategy that enhances anger, such as rumination, rather than mitigates it. Ruminative thought in general may result in activation of both regulatory PFC structures and the amygdala. A composite rumination measure, including the Anger Rumination Scale (ARS;

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Sukhodolsky, Golub, & Cromwell, 2001), was associated with increased activation in the amygdala and the vlPFC during attempts to increase affect in response to negative images (Ray et al., 2005).

Denson and colleagues (2009) examined neural correlates of anger-specific rumination in an undergraduate sample. Following provocation, participants were given prompts to engage in various forms of thought: provocation-focused (e.g. "Think about whom you have interacted with in the experiment up to this point"), self-focused (e.g. "Think about what kind of person you are", "Think about why you respond to others the way you do"), and neutral-focused (e.g. "Think about a bus driving down the street"). Few differences emerged between the provocation- and self-focused conditions. Compared to distraction, both provocation- and self-focused conditions involved greater recruitment of regions related to anger and social pain (dACC), emotion regulation (LPFC), arousal (thalamus, insula), and self-referential thought (dMPFC). Activation of the dmPFC and right anterior insula across both rumination conditions, compared to the neutral-focused condition, correlated with scores on self-reported state rumination and scores on the displaced aggression questionnaire (DAQ; Denson, Pedersen, & Miller, 2006), a measure of anger rumination, revenge-planning, and the tendency to direct aggression toward targets other than initial causes of anger.

Based on these findings, it seems likely that individuals with BPD would demonstrate activation in a number of brain regions during anger rumination. First, given that anger rumination is one form of anger-related emotion regulation, it is likely that the OFC would be recruited, particularly the lateral OFC, which has been linked to regulatory function (Heatherton & Wagner, 2011; Wager, Davidson, Hughes, Lindquist,

& Ochsner, 2008b). Given that anger rumination is a regulatory strategy that increases, rather than diminishes, anger, this LOFC activation seems likely to be linked to increased amygdala activity. Regions previously linked to anger rumination specifically, namely as the dACC and dMPFC, are also expected to be recruited during anger rumination. If cues to anger ruminate produce a stronger effect for individuals with BPD than those without, activation in all of these regions would likely be more pronounced.

Neural correlates of reward. Positive reinforcement, such as monetary rewards, has been reliably associated with recruitment of the striatum, which incorporates the nucleus accumbens (NAcc), ventral tegmentum, caudate nucleus, putamen and globus pallidus (Elliott, Friston, & Dolan, 2000). Activation in the NAcc particularly has been linked to experiences of reward and subjective happiness (Knutson, Adams, Fong, & Hommer, 2001) and occurs in response to a range of appetitive cues and pleasurable activities, including receiving monetary rewards (Elliott et al., 2000; Elliott, Newman, Longe, & Deakin, 2003; Ernst et al., 2004; Knutson et al., 2001), exposure to appetizing food (O'Doherty, Deichmann, Critchley, & Dolan, 2002; Wang et al., 2004), shopping for preferred objects (Knutson, Rick, Wimmer, Prelec, & Loewenstein, 2007), experiencing orgasm (Komisaruk et al., 2004), and viewing attractive faces (Aharon et al., 2001) and positive emotional expressions (Rademacher et al., 2010). These naturally occurring rewards activate many the same regions as drugs of abuse (Volkow, Fowler, Wang, & Swanson, 2004).

One study specifically examined reward activation in response to emotional mental imagery (Costa, Lang, Sabatinelli, Versace, & Bradley, 2010). Healthy undergraduates were asked to engage in pleasant (e.g. winning the lottery), aversive (e.g.

a car accident), and neutral (e.g. reading the newspaper) mental imagery in a scanner and to rate their subjective experiences of the imagery. Pleasant imagery selectively activated the NAcc and the MPFC, with the degree of NAcc activation correlated with the extent of pleasure endorsed. In contrast, amygdala activation occurred for both negatively and positively valenced imagery.

Various forms of addictive or habitual behavior result in increased NAcc activation in response to anticipation of relevant appetitive cues. While individuals addicted to substances demonstrate baseline hypoactivity of reward networks, these regions, including the NAcc, are hyperactive when presented with drug-related stimuli (see Volkow et al., 2004 for a review). This NAcc sensitization to reward has been theorized to create a learned motivational response in the brain that facilitates addiction even in the absence of withdrawal symptoms (T. E. Robinson & Berridge, 2008), suggesting that this process could also facilitate non-drug habits. For example, the anticipation of food, but not food consumption, produces higher levels of NAcc activation in obese individuals (Stice, Spoor, Ng, & Zald, 2009). Similarly, women with bulimia demonstrated NAcc activation while planning a binge-eating episode (Pearson et al., 2012), and repeated sexual experiences result in increased NAcc reactivity to later sexual encounters (Kohlert & Meisel, 1999).

Altered emotion processing has been posited to affect neural processing of reward in BPD, particularly in the striatum (Enzi et al., 2013). Individuals with and without BPD performed in a monetary reward task that produced reward and punishment anticipation and feedback, while simultaneously being presented with images of varying emotional valence (positive, negative, neutral). When reward and punishment information were

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presented alongside neutral emotional content, striatal regions function similarly in individuals with BPD and controls in differentiating between reward and non-reward; however, in the context of emotional pictures, individuals with BPD demonstrated reduced reward differentiation and less deactivation of reward circuitry following cue exposure, paired with increased reactivity in the amygdala. One possibility is that emotional reactivity disrupts reward systems for individuals with BPD (Enzi et al., 2013). However, an alternate possibility is that for emotionally reactive individuals, such as those with BPD, emotional cues have great impact and thus more potency as a potential reward or punishment than small amounts of money. These findings hint at the possibility that emotionally evocative stimuli and activities, such as anger rumination, might function as BPD-relevant appetitive cues.

Reward systems also have important implications for understanding the links between sensitivity to interpersonal rejection and criticism and maladaptive behavior. Increased attempts to regulate the pain of rejection may increase subsequent impulsive behavior by increasing reward reactivity to subsequent appetitive cues. Greater rVLPFC recruitment during social rejection not only predicted self-regulatory failures, as described previously, but was also associated with stronger NAcc activation and less functional connectivity between the NAcc and rVLPFC in response to appetitive cues, such as images of alcohol (Chester & DeWall, 2014). If individuals with BPD demonstrate similar higher levels of rVLPFC recruitment when criticized or rejected, this may relate to subsequent amplified NAcc activation when engaging in or contemplating potentially rewarding activities, such as ruminating about the provocation, and subsequent behavioral dyscontrol, such as more aggressive behavior.

A Novel Approach to Clarifying the Function of Anger Rumination in BPD

To examine the function of anger rumination in BPD, we utilized fMRI to compare blood oxygen level dependent (BOLD) activation changes in specific brain regions among BPD patients and control participants across the experience of interpersonal provocation, ruminative responding, and subsequent opportunity for aggressive behavior. Individuals in both groups were scanned as they experienced an interpersonal provocation (negative feedback about a writing task from a fictitious evaluator). During the critical feedback, it was hypothesized that participants with BPD (vs. healthy controls) would demonstrate higher activation in brain regions associated with social pain (dACC, vLPFC) and self-conscious reactivity to errors (AI, vlPFC).

Three scans then took place, in counterbalanced order across participants: one with instructions to ruminate about the provocation (provocation-focus), one with instructions to ruminate focusing on themselves (self-focus), and one with instructions to think about neutral topics (neutral-focus). These methods have been validated in prior fMRI research on anger rumination (Denson et al., 2009). All participants were expected to demonstrate greater activation in regions previously associated with anger rumination (dACC, dMPFC) during subsequent provocation-focused thought compared to neutral-focused thought; however, this effect was expected to be greater for participants with BPD. All participants were also expected to demonstrate LOFC activation during anger rumination; however, this effect was expected to be stronger for participants with BPD and positively associated with activation of the amygdala for the BPD group only. Participants with BPD (vs. controls) were predicted to experience greater reward during

provocation-focused thought, as indicated by greater activation in brain regions associated with reward and pleasure (NAcc, MOFC).

A final scan was conducted while the participants engaged in an aggression paradigm that offered an opportunity to retaliate against the individual the participant believed provided the initial provocation. The primary outcomes of interest were differences in BOLD activation changes in individuals with BPD compared to healthy controls during these episodes in regions associated with reward, rumination, social pain, anger, and emotion regulation. The BPD group was expected to display greater levels of aggression in the final task, and the increased NAcc activation during provocation-focused thought for the BPD group was expected to mediate this effect.

Chapter Two: Methods

Participants

Participants (n=31) were right-handed women who were at least 18 years old. Thirteen of them met the DSM-V criteria for BPD. The other eighteen were age-matched healthy controls. All participants were screened for suitability for MRI research. Individuals were excluded who reported neurological pathology or injury, developmental disorders, substance use disorders, psychotic symptoms, and claustrophobia. Control participants were required to meet no criteria for BPD and to have never received any other psychological diagnosis or treatment and not to be using psychoactive medication or substances. Of the BPD group, 11 were not on any psychoactive substances at the time of the study, and 2 were taking SSRI medication. Recruitment occurred from contacts with local clinics and psychotherapists, craigslist advertisements, study flyers, and introductory psychology classes at a large, public university. Participants received either $100 for participating or course credit.

Measures

Structured Clinical Interview for the DSM-IV (SCID-II; First & Gibbon, 1997). The SCID-II is a standardized, semi-structured, clinician administered interview for diagnosing DSM-IV Axis II mental disorders.

Personality Assessment Inventory Borderline Features Scale (PAI-BOR; Morey, 2007). The PAI-BOR has 24 items measuring four aspects of BPD pathology: affective instability, identity problems, negative relationships, and self-harm. Responses range from 0 ("false, not at all true") to 3 ("very true"). Elevated scores on the PAI-BOR have been shown to differentiate BPD patients from those with other diagnoses, including

anxiety, mood, and psychotic disorders, antisocial personality disorder, and substance abuse disorders (Morey, 2007). Scores above 37 (T>70) are considered to be in the clinical range and predict BPD-specific dysfunction in clinical, community, and student samples (Morey, 2007; Trull, Useda, Conforti, & Doan, 1997). These findings suggest that high scores on the PAI-BOR are likely to reflect BPD-specific pathology rather than general distress or other disorders. In the present study, PAI-BOR total score and subscales demonstrated good internal consistency (α =.85-.92).

Center for Epidemiological Studies—Depression (CES-D; Radloff, 1977). The CES-D is a 20-item inventory of depressive symptoms. The CES-D asks participants to rate their mood, thoughts, and behavior during the previous week on a 4-point Likert scale, ranging from 0 ("rarely or none of the time") to 3 ("most or all of the time"). The CES-D has been well validated in both general and psychiatric populations (Radloff, 1977; Roberts, Rhoades, & Vernon, 1990). In the present study, the CES-D demonstrated excellent internal consistency (α = .94).

PTSD Checklist—Civilian Version (PCL-C; Blanchard, Jones-Alexander, Buckley, & Forneris, 1996). The PCL-C is a 17-item questionnaire that asks participants to rate the extent they have been bothered by PTSD symptoms over the past month. Responses range from 1 ("not at all") to 5 ("extremely"). The PCL-C has demonstrated good internal consistency and test-retest reliability and convergent and discriminant validity (Blanchard et al., 1996). In the present study, the PCL demonstrated excellent internal consistency (α = .95).

Positive and Negative Affect Schedule—Expanded Form (PANAS-X; Watson & Clark, 1999). The PANAS-X is a 60-item measure that asks participants to rate the

extent to which they feel a variety of emotions (e.g. cheerful, disgusted, attentive) on a 5-point Likert scale. Responses range from 1 ("very slightly or not at all") to 5 ("extremely"). Instructions for this instrument can be adjusted to assess multiple time frames; in the current study, participants will be asked to rate the extent to which they have been feeling each emotion "on average." Convergent and discriminant validity was supported by correlations in the expected directions with a variety of other constructs. In the present study, the Anger, Sadness, Guilt, and Fear subscales of the measure demonstrated excellent internal consistency ($\alpha = .90 - .92$).

Anger Rumination Scale (ARS; Sukhodolsky et al., 2001). The ARS has 19 items assessing the tendency to focus attention on angry moods, recall past anger episodes, and think about the causes and consequence of anger episodes. It has four subscales: angry afterthoughts; thoughts of revenge; angry memories; and understanding causes (e.g., "When something makes me angry I turn this matter over and over again in my mind"). Responses range from 1 ("almost never") to 4 ("almost always"). Sukhodolsky et al. (2001) reported moderate correlations between ARS scores and anger-related constructs such as anger expression and suppressed anger. Factor analysis indicated that items representing anger constructs loaded on separate factors from the anger rumination items, which all loaded on a single factor, supporting the discriminant validity of anger rumination as distinct from anger. The ARS total score demonstrated excellent internal consistency in the present study ($\alpha = .96$).

Displaced Aggression Questionnaire (DAQ; Denson et al., 2006). The DAQ consists of 31 items, assessing aggressive behavior directed at human targets other than the initial sources of provocation (displaced aggression) and contributing cognitive traits

to this behavior. The DAQ is comprised of three distinct factors: anger rumination (DAQ-AR; e.g., "I keep thinking about events that angered me for a long time"), revenge planning (DAQ-RP; e.g., "When someone makes me angry, I can't stop thinking about how to get back at this person"), and behavioral displaced aggression (DAQ-DA; e.g., "When something or someone makes me angry, I am likely to take it out on another person."). Six of the 10 items in the DAQ-AR subscale are from the ARS, as are two of the 11 items in the DAQ-RP. The DAQ and its subscales have demonstrated good reliability and predict both self-report aggression and displaced aggression in the laboratory (Denson et al., 2006). In the present study, the DAQ subscales demonstrated excellent internal consistency (α = .95 - .98).

Procedure

Preliminary screening. A phone screen was administered to all potential participants in which a brief clinical diagnostic interview based on the SCID-II BPD module was administered to determine eligibility for the study. Individuals who met at least five criteria for BPD were recruited for the BPD group; individuals who met no criteria for BPD and had never received any psychological diagnoses or treatment were recruited for the control group. Participants were also screened for safety and comfort in the MRI environment and administered a risk assessment. No individuals endorsed present risk of harm to self or others. These phone interviews and all subsequent clinical interviews and risk assessments were conducted by an advanced clinical psychology doctoral student who was trained in risk assessment procedures and clinical interviewing and had experience as a therapist for individuals with BPD.

Assessment session. Eligible individuals were asked to attend an assessment session. Participants were assessed for risk of harm to self and others at the beginning of the session; no participants endorsed current risk. Participants completed self-report measures of BPD symptoms, depressive symptoms, PTSD symptoms, negative affectivity, anger rumination, and aggression. The SCID II for BPD was then administered to all participants. Any participants who do not meet inclusion criteria (no BPD criteria met for the control group; at least five BPD criteria fully endorsed for the BPD group) were excluded from the second study session.

Scanning session. The scanning session took place between 2-10 days after the assessment visit. Participants arrived at the University of Kentucky's Magnetic Resonance Imaging and Spectroscopy Center and were administered a risk assessment for suicide and harm to others. After passing a final screening for MRI-related safety and comfort concerns, participants began the experimental procedure.

Essay-Writing Paradigm: Participants were asked to write a short essay about a time in which someone else angered them. In accordance with a previously validated provocation paradigm (Bushman & Baumeister, 1998), they were told that a research assistant would evaluate it on several key criteria and that this feedback would be provided while they are in the MRI scanner. Each participant's essay was given the same harsh criticism, regardless of what they had written.

Scanning Procedure: Each MRI scanning session included 3 experimental tasks. First, participants completed the Provocation Task, lasting four minutes. Adopting a modified version of Denson and colleagues' (2009) procedure, we acquired 2 minutes of baseline neural activation from participants. Next, participants viewed a series of nine

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ratings of various characteristics of their essay (10 seconds each; e.g., "clarity of expression", "writing style"). Finally, participants viewed their reviewer's 'comments' on their essay for 30 seconds, which were: "Horrible! One of the worst essays I have ever read!" Afterwards, participants completed the 340 second long *Directed Rumination Task*, which involved three inductions, presented in counter-balanced order across participants within groups. In all inductions, participants viewed a series of 6 statements (15 seconds each), which they were asked to think about. In the provocation-focused part of the rumination task, participants read rumination prompts with statements instructing them to engage in anger rumination, reflecting on the provoking incident encountered earlier in the study (e.g., "Think about how you have been treated" "Think about why people treat you the way they do" "Think about whether your treatment was unfair or unreasonable," see Appendix 1 for all prompts for all inductions). In the self-focused part of the rumination task, participants read statements instructing individuals to think about themselves (e.g., "Think about what kind of a person you are." "Think about why you respond to others the way you do."). In the neutral-focused part of the rumination task, participants read prompts with statements instructing individuals to reflect on neutral statements unrelated to the study (e.g., "Think about the layout of the local post office", 'Think about a bus driving down the street"). Between blocks of the DRT, participants were given a 30 second rest period with a fixation cross, followed by a 5-second prompt to get ready for the next set of statements.

Participants then completed a well-validated behavioral measure of aggression, the Taylor Aggression Paradigm (TAP; S. P. Taylor, 1967). Participants were told they would play a computerized game against their essay evaluator. This game took the form

of a competitive reaction-time task in which the winner could deliver aversive noise to the loser through headphones. The aggression task consisted of nine trials. Prior to each trial, participants set the volume of the noise blast their partner would receive if the participant won the round. Specific volumes could not be calibrated, as decibel readers are not MRI-safe; therefore, volumes were calibrated by the subjective appraisal of the experimenter as quiet (1), noticeably loud (2), loud (3), and uncomfortably loud (4), as has been done in previous fMRI work utilizing the TAP (Krämer, Jansma, Tempelmann, & Münte, 2007). After each trial, participants saw whether they won or lost, as well as the volume settings their partners had ostensibly set for them. Participants won five trials and lost four trials (determined randomly, despite being told that their performance was what determined the outcome of each trial). Trials were also split into two categories: high provocation (following a 3 or 4-level volume setting by the opponent on the previous trial) and low provocation (following a 1 or 2-level blast volume setting by the opponent on the previous trial). Three scores were generated for each participant: mean volume setting across all trials, mean settings following high provocation, and mean settings following low provocation. This task provides an ethical way to evaluate how participants utilize the opportunity to blast their essay evaluator with unpleasant noise. The construct validity of this task is well established (Anderson & Bushman, 1997; Bernstein, Richardson, & Hammock, 1987).

Debriefing: After exiting the scanner, participants were escorted to a private room where they were told of the deception involved in the writing task and provocation. A risk assessment was administered, and no participants endorsed elevated risk of harm to self or others.

Data Acquisition and Analyses

fMRI data acquisition. All images were collected on a 3T Siemens Magnetom Trio scanner using a Siemens 32-channel head coil. Functional images were acquired with a T2-weighted gradient echo sequence, with a 3D shim applied before functional data acquisition (matrix size = 64 × 64, field of view = 224 mm, echo time = 28 ms, repetition time = 2.5 s, slice thickness = 3.5 mm, 40 interleaved axial slices, flip angle = 90°). These parameters allowed for whole- brain coverage with 3.5mm cubic voxels. A high-resolution, T1-weighted image was also acquired from each participant so that functional data could be registered to native anatomical space and then normalized to the Montreal Neurological Institute (MNI) atlas space.

fMRI preprocessing. All preprocessing and statistical analyses were conducted using FSL (Oxford Center for Functional Magnetic Resonance Imaging [FMRIB]; (Smith, Jenkinson, Woolrich, & Beckmann, 2004; Woolrich, Jbabdi, Patenaude, & Chappell, 2009)). Functional volumes were reconstructed from k-space using a linear time interpolation algorithm to double the effective sampling rate, the first of which was removed to allow for signal equilibration. Remaining functional volumes were corrected for head movement to the median volume using MCFLIRT (Jenkinson, Bannister, Brady, & Smith, 2002), corrected for slice-timing skew using temporal sinc interpolation, pre-whitened using FILM and smoothed with a 5mm FWHM Gaussian kernel. To remove drifts within sessions, a high-pass filter with a cutoff period of 120 s was applied. Non-brain structures were stripped from functional and anatomical volumes using FSL's Brain Extraction Tool (Smith, 2002).

fMRI data analyses. We modeled within-subjects, between-subjects and between-groups (BPD vs. control) variance in brain activation utilizing a 2-stage summary statistics approach to multi-level modeling via FSL. A fixed-effects analysis modeled event-related responses for each run of each participant using a canonical double-gamma hemodynamic response function with a temporal derivative. Motion parameters were modeled as nuisance regressors for all analyses. For the essay feedback task, response to feedback was modeled as percent-change from pre-task baseline, with pre-block instructions were modeled as a nuisance regressor. For the DRT task, provocation-focus, self-focus, and neutral-focus blocks were modeled as percent-change from unmodeled, implicit baselines. Pre-block instructions were modeled as a nuisance regressor. Within the DRT task, we contrasted provocation-focus with both self-focus and neutral-focus blocks, as well as self-focus contrasted with neutral-focus, to assess activation specific to each of those conditions. For the TAP, aggression trials were contrasted to the implicit, unmodeled baseline, with three variables created: one averaging across all trials, one across trials following high provocation from the opponent, and one across trials following low provocation from the opponent.

To model these variables, performed top-level, mixed-effects analysis were performed, which created group average maps for contrasts of interest. Z (Gaussianized T/F) statistic images were thresholded using clusters determined by $Z > 2.3$ and a (corrected) cluster significance threshold of $p < .005$ across the whole brain and contrained to out *a priori* regions-of-interest (ROI). Parameter estimates were extracted (in units of percent signal change) from activated clusters from both whole-brain and a priori ROIs.

The resulting activation from those contrasts were compared between controls and participants with BPD.

Construction of ROI. Each participant's contrast volumes were fed into a group-level, mixed-effects analysis that created group average maps. Cluster-based thresholding (Heller, Stanley, Yekutieli, Rubin, & Benjamini, 2006; Worsley, 2001) was applied to each image (cluster Z statistic threshold: 2.3). Region of interest (ROI) masks were constructed for the rVLPFC, LOFC (left, right), MOFC (left, right), and amygdala (left, right) from the Automated Anatomical Labeling (AAL) atlas using MNI coordinates (Tzourio-Mazoyer et al., 2002). The ROI mask for the Nacc constructed from the Wake Forest Pickatlas toolkit (Maldjian, Laurienti, Kraft, & Burdette, 2003). Family-wise error correction was then applied to all voxels within the ROI masks (cluster significance threshold: p b .005).

Four ROIs in the dMPFC (left superior dMPFC, right superior dMPFC, left medial dMPFC, and right medial dMPFC) and two in the dACC (right dACC, left dACC) were based on an activation clusters found in previous research on activation in these regions during anger rumination, compared to neural thought (Denson et al., 2009). Each ROI was constructed using a 8mm-radius sphere around the MNI coordinates based on the previous functional data.

Chapter Three: Results

Data Screening

Data was screened for outliers on all measures. One participant was removed from analyses due to excess movement during the scan. One participant was removed from analyses due to values greater than 3 SD above the mean for the entire sample for activation of the right and bilateral NAcc during the provocation > neutral contrast during the DRT. One control participant was removed from analyses due to partial endorsement of one of the DSM BPD criteria. The final sample analyzed included 28 participants (BPD group = 13; control group = 15).

Demographics

Groups did not significantly differ by age (see Table 1). Groups also did not demonstrate significant differences in race (χ^2 = .59, p = .746) or education level (χ^2 = 4.14, p = .126). Accordingly, these demographic variables were not controlled for in subsequent analyses.

Clinical Interview and Self-Report Measures

To confirm validity of SCID II diagnoses, t-tests were computed comparing PAI-BOR scores for the BPD group to the control group, as well as scores on the CES-D, PANAS-NA, PCL, AQ, ARS, and DAQ (see Table 1). As expected, the BPD group reported significantly higher levels of BPD symptoms on all subscales of the PAI-BOR, as well as significantly higher scores on the CES-D, PCL, PANAS-NA subscales, all AQ subscales, ARS, and all subscales of the DAQ.

For the clinical scales, the groups' mean scores fell into appropriately different levels of impairment. The control group reported a mean level of PAI-BOR total scores

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in the low symptoms category (raw score < 18; T < 30), whereas the BPD group's mean is clinically elevated (raw score > 37; T >70). The control group reported a mean level of CES-D total scores in the low symptoms category (<16), whereas the BPD group's mean is in the "probable depression" range (>23). The control group also demonstrated a mean level of PCL scores in the little to no symptom category (17-29), whereas the BPD group's mean falls into the moderate to moderately high range (30-44).

Despite the association of BPD diagnosis with BPD, depressive, and PTSD symptoms, the association between BPD diagnosis and PAI-BOR total scores was significantly stronger than the association between BPD and both PCL total scores (t[25] = 3.99, p <.001) and CES-D total scores (t[25] = 2.89, p <.01). While the BPD group demonstrated symptoms of a range of psychopathology, as is typical of individuals with BPD (Tomko, Trull, Wood, & Sher, 2013), their diagnostic status was particularly associated with BPD-specific symptoms. This suggests the appropriateness of using this sample to investigate BPD-specific hypotheses; however, it is not possible to eliminate the possibility that other symptoms contribute to findings. The association between BPD diagnosis and DAQ-AR scores was also significantly stronger than the association between BPD and both DAQ-DA (t[25] = 3.97, p <.001) or DAQ-RP (t[25] = 5.32, p <.001). This finding highlights the relevance of anger rumination, over and above other anger-related constructs, to BPD.

Table 1. *Differences between control and BPD groups on self-report measures of BPD symptoms, depression, PTSD symptoms, negative affect, anger rumination, aggression, and age (N = 28).*

	HC Mean (SD)	BPD Mean (SD)	t	*p*-value	r
PAI-BOR AI***	.30 (.29)	1.99 (.38)	13.40	<.001	.87
PAI-BOR ID***	.62 (.39)	2.09 (.50)	8.68	<.001	.74
PAI BOR NR***	.39 (.34)	2.08 (.47)	10.96	<.001	.82
PAI-BOR SH**	.37 (.25)	1.21 (.67)	4.33[1]	.001	.45
PAI-BOR Tot***	10.07 (5.13)	44.23 (8.75)	12.81	<.001	.86
CES-D***	7.73 (6.78)	28.23 (9.27)	6.74	<.001	.64
PCL***	23.73 (8.96)	49.31 (13.21)	6.07	<.001	.59
PANAS Anger***	1.21 (.23)	2.47 (.74)	5.92[1]	<.001	.60
PANAS Sad***	1.28 (.36)	3.06 (.62)	9.13[1]	<.001	.77
PANAS Guilt***	1.20 (.34)	2.64 (.92)	5.35[1]	<.001	.55
PANAS Fear***	1.37 (.33)	2.84 (.95)	5.32[1]	<.001	.55
ARS***	1.27 (.21)	2.55 (.37)	11.13[1]	<.001	.84
DAQ-AR***	1.49 (.62)	4.86 (1.04)	10.55	<.001	.81
DAQ-DA***	1.89 (1.03)	3.77 (1.37)	4.14	<.001	.40
DAQ-RP*	1.16 (.32)	2.64 (1.93)	2.74[1]	.017	.25
AQ Phys Agg*	1.49 (.44)	2.28 (1.24)	2.19[1]	.045	.17
AQ Verb Agg***	2.29 (.68)	4.08 (1.00)	5.42[1]	<.001	.54
AQ Anger***	1.60 (.58)	3.85 (.92)	7.83	<.001	.70
AQ Hostility***	1.44 (.52)	4.54 (1.14)	8.99[1]	<.001	.77
Age	22.07 (4.03)	21.23 (3.30)	-.60	.56	.01

*p <.05, ** p<.01, ***p<.001
Note: PAI-BOR = Personality Assessment Inventory-Borderline Features Subscale; AI = Affective Instability; ID = Identity Disturbances; NR = Negative Relationships; SH = Self-Harming; CES-D = Center for Epidemiological Studies Depression Scale; PCL = PTSD Checklist; PANAS-NA = Positive and Negative Affect Schedule; ARS = Anger Rumination Scale; DAQ = Displaced Aggression Questionnaire; AR = Anger Rumination; DA = Displaced Aggression; RP = Revenge Planning; AQ = Aggression Questionnaire; Phys Agg = Physical Aggression; Verb Agg = Verbal Aggression.
t-tests conducted with equal variances assumed except where denoted by ([1]).
r values denote correlations between BPD status and self-report measures.

Behavioral Results

BPD status significantly predicted higher overall aggression scores on the TAP (t = 2.06, *p* = .049). This effect appears to be driven more by responses to low provocation (receiving a 1 or 2 noise blast from opponent; t = 2.12, *p* = .044) compared to responses following high provocation (receiving a 3 or 4 noise blast from opponent; t = 1.86, *p* = .074).

Imaging Results

Essay Feedback. In whole-brain analyses, BPD diagnosis predicted increased activation in the insula (R^2 = .19, F(1, 26) = 7.34, β = .44, *p* = .019) in response to the essay feedback (Figure 1, feedback > pre-feedback contrast). ROI analyses also demonstrated BPD status predicting increased activation in the vlPFC (R^2 = .22, F[1, 26] = 6.25, β = .47, *p* = .012). No between-group differences in activation in response to the essay feedback were found for the dACC (R^2 = .04, F[1, 26] = .04, *p* = .838) or the vACC (R^2 = .14, F[1, 26] = .54, *p* = .470).
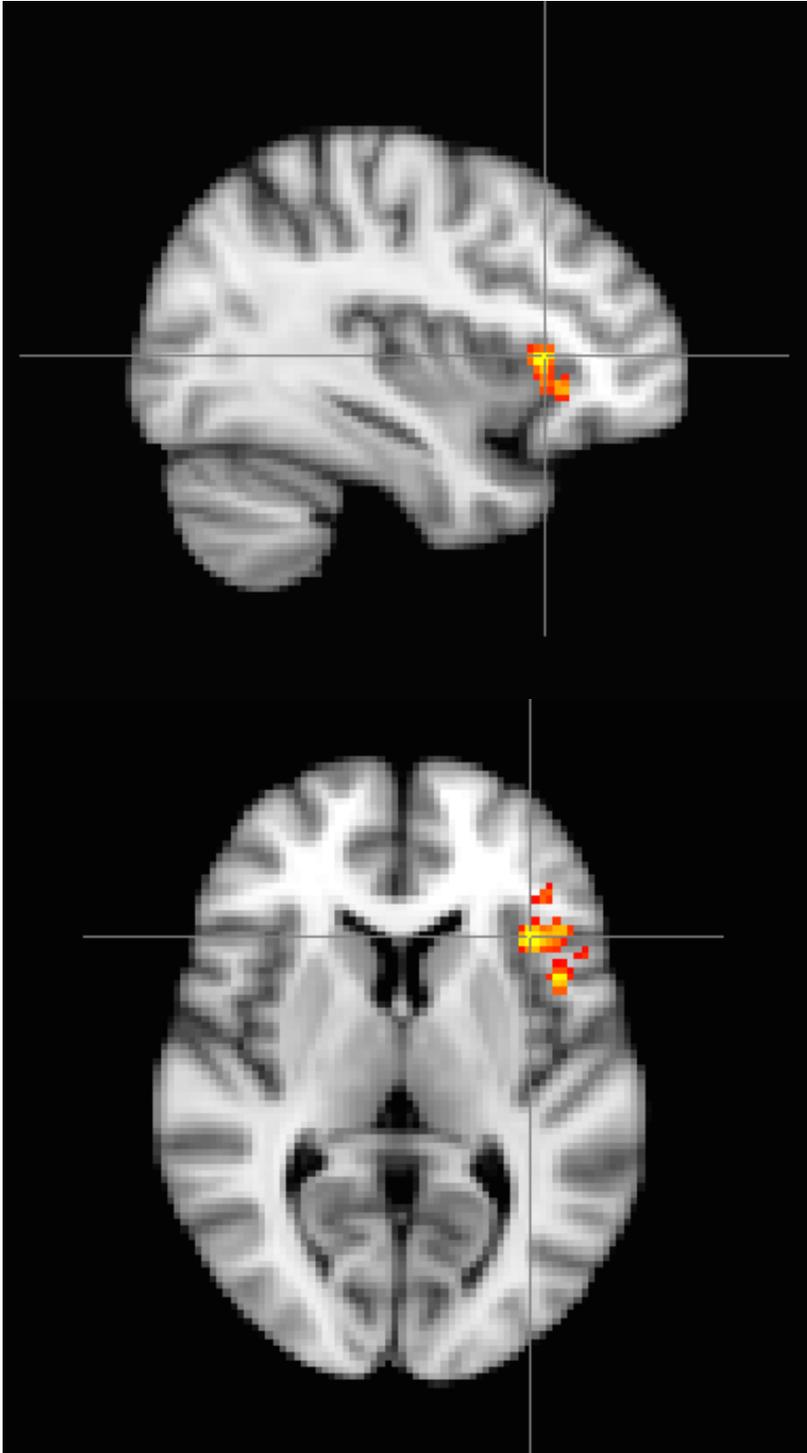
Figure 1. Neural activation during the essay feedback task, contrast between BPD group and controls demonstrating increased activation in the right insula for the BPD group.

Directed Rumination Task. Provocation-focus, compared to neutral-focus (provocation > neutral contrast), produced significantly greater activation in four ROIs of the dMPFC and trends toward greater activation in two ROIs in the dACC (see Table 2 for all neural activation means and comparisons for the DRT). Relative to the neutral condition, self-focus also produced significantly greater activation in the four dMPFC ROIs, but no differences were observed in the dACC (self > neutral contrast). There were no differences between the provocation-focus and self-focus condition in any ROIs (provocation > self contrast). No significant between-group interactions were found for any of the dMPFC ROIs (F[1,26] = .14-.69, p = .415-.709), the left dACC ROI (F[1,26] = 2.12, p = .158), or right dACC ROI (F[1,26] = .80, p = .381). However, when these regions were compared separately for each group (see Table 2), significant differences in the dMPFC for provocation-focus compared to neutral-focus were more consistently observed for the BPD group, and only the BPD group demonstrated significant differences in dMPFC activation in the self-focus compared to neutral-focus. Given the small sample size, the present study may be underpowered to detect these between-group differences.

Across the full sample, greater activation was demonstrated in the LOFC (both right and left) during provocation focus relative to neutral focus; no other significant differences emerged for either the LOFC or MOFC. No significant between-group effects were found when BPD status was used to predict these contrasts in activation in the LOFC right (F[2,52] = .30, p = .741) or LOFC left (F[2,52] = .53, p = .590). None of the contrasts were significant for activation in the amygdala (right or left) across the full sample or for either group. Notably, the amygdala also had higher variance than other

Table 2. *Comparisons between levels of neural activation in all regions of interest for conditions of the Directed Rumination Task, across the full sample and by diagnostic group.*

Total (N=28)	Prov (p) Mean (SD)	Self (s) Mean (SD)	Neutral (n) Mean (SD)	t(p>n)	t(p>s)	t(s>n)
NAcc R	.027 (.110)	-.046 (.183)	-.045 (.111)	2.57*	1.75^	-.21
NAcc B	.035 (.111)	-.035 (.169)	-.028 (.092)	3.01**	1.73^	-.03
NAcc L	.046 (.124)	-.017 (.174)	-.001 (.115)	1.42	1.60	-.411
dMPFC LS	.046 (.073)	.041 (.076)	-.026 (.083)	4.61***	.25	3.39**
dMPFC LM	.046 (.062)	.044 (.057)	-.012 (.075)	4.00***	.14	3.46**
dMPFC RS	-.005 (.048)	.006 (.060)	-.046 (.073)	3.34**	-.74	2.96**
dMPFC RM	.029 (.074)	.010 (.062)	-.031 (.060)	4.26***	1.17	2.32*
dACC L	.040 (.061)	.021 (.066)	-.003 (.128)	1.86^	1.24	1.12
dACC R	.030 (.050)	.011 (.059)	.006 (.100)	1.37	1.50	.31
LOFC R	.094 (.083)	.063 (.091)	.025 (.160)	2.20*	1.38	1.38
LOFC L	.034 (.073)	.001 (.070)	-.005 (.098)	2.08*	1.84^	.33
MOFC R	.008 (.071)	.007 (.064)	-.019 (.100)	1.48	.08	1.23
MOFC L	-.011 (.121)	-.005 (.099)	-.038 (.103)	1.13	-.19	1.16
Amyg R	-.102 (.405)	-.023 (.636)	.010 (.447)	-1.15	-.61	-.26
Amyg L	.033 (.517)	-.095 (.455)	-.095 (.356)	1.40	.91	.00
Controls (N= 15)	Prov (p) Mean (SD)	Self (s) Mean (SD)	Neutral (n) Mean (SD)	t(p>n)	t(p>s)	t(s>n)
NAcc R	.028 (.092)	-.034 (.127)	.000 (.067)	1.03	1.36	-1.00
NAcc B	.035 (.100)	-.037 (.131)	.004 (.077)	.98	1.54	-1.21
NAcc L	.047 (.123)	-.042 (.155)	.010 (.136)	.73	1.67	-1.06
dMPFC LS	.033 (.070)	.025 (.052)	-.028 (.110)	2.56*	.40	2.03^
dMPFC LM	.037 (.064)	.030 (.046)	-.020 (.102)	2.49*	.39	2.05^
dMPFC RS	-.004 (.051)	.004 (.033)	-.034 (.093)	1.56	-.62	1.66
dMPFC RM	.025 (.070)	-.001 (.052)	-.019 (.068)	2.24*	1.23	.88
dACC L	.028 (.044)	.003 (.072)	-.032 (.165)	1.44	1.06	.90
dACC R	.025 (.048)	-.007 (.053)	-.009 (.129)	1.08	1.87	.09
LOFC R	.093 (.083)	.047 (.088)	.027 (.214)	1.17	1.67	.45
LOFC L	.022 (.070)	-.007 (.053)	.000 (.126)	.71	1.41	-.25
MOFC R	.018 (.072)	.011 (.042)	-.014 (.120)	1.20	.38	.82
MOFC L	.011 (.126)	-.005 (.061)	-.035 (.120)	1.43	.44	.83
Amyg R	-.048 (.341)	.021 (.758)	.082 (.425)	-.90	-.34	-.26
Amyg L	.117 (.598)	-.190 (.558)	-.071 (.379)	1.43	1.37	-.62

Table 2. (continued)

BPD (N=13)	Prov (p) Mean (SD)	Self (s) Mean (SD)	Neutral (n) Mean (SD)	t(p>n)	t(p>s)	t(s>n)
NAcc R	.026 (.130)	-.060 (.237)	-.100 (.131)	3.31**	1.13	.57
NAcc B	.034 (.127)	-.032 (.211)	-.064 (.097)	2.76*	.97	.55
NAcc L	.046 (.129)	.011 (.196)	-.014 (.089)	1.35	.57	.41
dMPFC LS	.060 (.077)	.059 (.096)	-.024 (.034)	4.28**	.03	2.73*
dMPFC LM	.056 (.060)	.059 (.066)	-.003 (.022)	3.32**	-.13	2.92*
dMPFC RS	-.006 (.046)	.007 (.082)	-.060 (.037)	3.69**	-.48	2.51*
dMPFC RM	.035 (.081)	.023 (.071)	-.045 (.047)	3.96**	.44	2.32*
dACC L	.053 (.076)	.042 (.054)	.030 (.054)	1.72	.62	.73
dACC R	.036 (.057)	.032 (.060)	.023 (.052)	1.01	.20	.47
LOFC R	.095 (.087)	.083 (.094)	.022 (.064)	3.21**	.35	1.80^
LOFC L	.047 (.076)	.010 (.086)	-.012 (.056)	3.01*	1.21	.80
MOFC R	-.003 (.071)	.003 (.085)	-.024 (.076)	.83	-.31	.89
MOFC L	-.035 (.115)	-.006 (.133)	-.041 (.085)	.16	-.65	.78
Amyg R	-.165 (.475)	-.075 (.484)	-.075 (.475)	-.67	-.60	.00
Amyg L	-.063 (.406)	.014 (.280)	-.123 (.339)	.46	-.52	1.56

^$p<.10$, *$p<.05$, ** $p<.01$, ***$p<.001$

Note: Prov = Provocation, R =right, B = bilateral, L= left, NAcc = nucleus accumbens, dMPFC = dorsomedial prefrontal cortex, dACC = dorsal anterior cingulate cortex, LOFC = lateral orbitofrontal cortex, MOFC = medial orbitofrontal cortex, Amyg = amygdala

regions. Five participants in addition to those already excluded based on other measures

had values for single conditions that were over 1 SD from the mean. This variability was

taken to indicate problems with imaging of this region; accordingly, further analyses with

the amygdala were not conducted.

When analyzed across the full sample, a significant difference in activation in

both the right and bilateral NAcc between the provocation and neutral-focus conditions of

the DRT emerged, showing higher levels of activation during the provocation-focus

condition. Findings were not significant for the left NAcc. To test the hypothesis that

BPD would explain this finding, BPD status was entered into a regression analysis

predicting increased NAcc activation during provocation-focus relative to neutral-focus
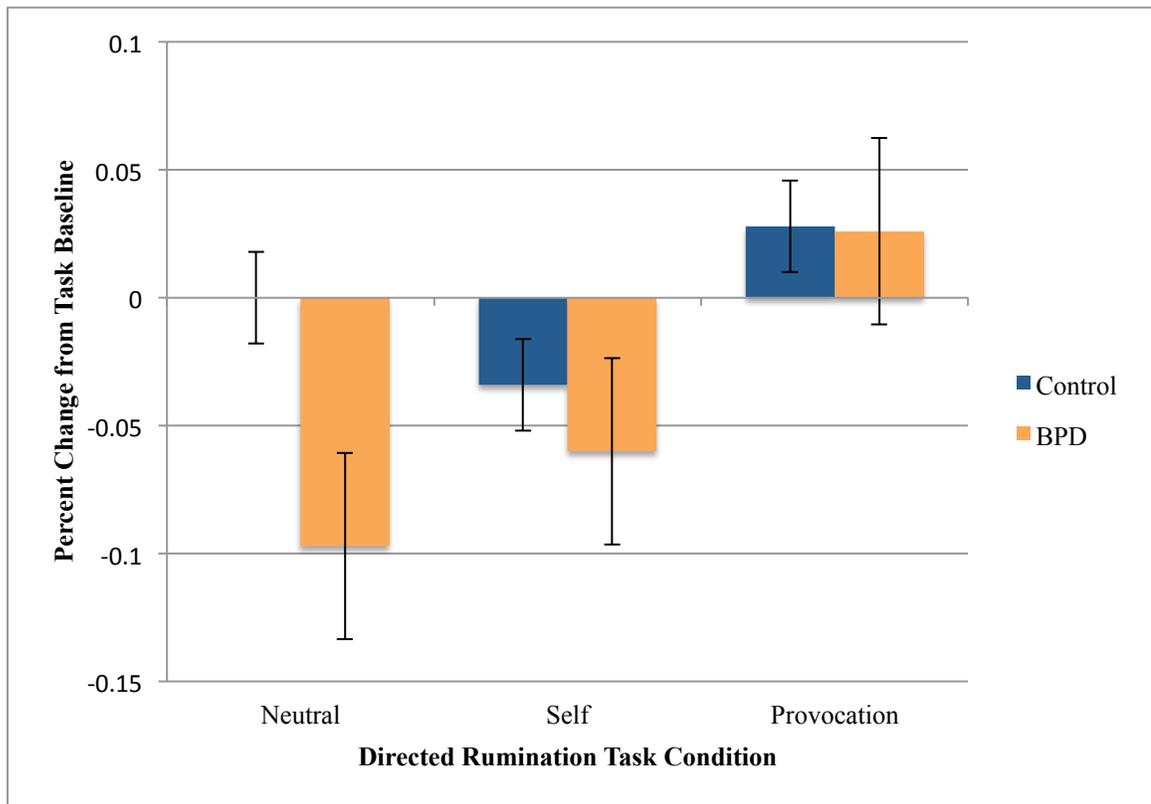
Figure 2. Nucleus accumbens activation during provocation-focus, self-focus, and neutral-focus, for group with borderline personality disorder and controls.

(provocation > neutral contrast). A significant effect of BPD status on right NAcc activation was observed ($R^2 = .14$, $F[1, 26] = 4.33$, $\beta = .38$, $p = .047$). As hypothesized, for individuals with BPD, the provocation-focus condition, compared to neutral focus, led to increased activation in the right NAcc ($t[12]= 3.31$, $p = .006$), whereas for controls, no significant differences between these two conditions were observed ($t(14) = 1.03$, $p = .319$; see Figure 2). BPD status had a similar but nonsignificant effect on bilateral NAcc activation (provocation > neutral: $R^2 = .07$, $F[1, 26] = 1.90$, $\beta = .26$, $p = .180$). The self-focus condition demonstrated no significant differences from either provocation-focus or neutral focus for the full sample in activation for either the right or bilateral NAcc. BPD status was not a significant predictor for either contrast of right NAcc activation

(provocation > self: R^2 = .06, F[1,26] = .08, p = .782; self > neutral: R^2 = .04, F[1,26] =

1.01, p = .324) or bilateral NAcc (provocation > self: R^2 = .00, F[1,26] = .01, p = .938;

self > neutral: R^2 = .05, F[1,26] = 1.24, p = .276).

Taylor Aggression Paradigm. Correlations between BPD status and activation

of ROIs relating to anger rumination and reward were computed for all TAP trials, trials

following high provocation, and trials following low provocation (see Table 3). Contrary

to hypotheses, BPD status predicted lower activation of the right dACC, across all TAP

trials, with a trend toward lower activation in the high provocation condition. BPD status

was not significantly associated with activation during any of the TAP trial sets in any of

the following ROIs: right, bilateral, or left NAcc, the four regions of the dMPFC, and left

dACC.

Table 3. *Correlations between borderline personality disorder status and neural*
activation during Taylor Aggression Paradigm trials (aggression>baseline contrast)
(N=28).

	All trials	High Provocation	Low Provocation
NAcc B	.01	.00	.01
NAcc R	.08	.10	.06
NAcc L	-.11	-.13	-.08
dMPFC, L superior	-.06	-.11	.02
dMPFC, L medial	-.13	-.15	-.04
dMPFC, R superior	-.26	-.25	-.17
dMPFC, R medial	-.19	-.16	-.13
dACC L	-.24	-.26	-.12
dACC R	-.43*	-.36^	-.29

^p<.10, *p<.05, ** p<.01, ***p<.001

Note: R =right, B = bilateral, L= left, NAcc = nucleus accumbens, dMPFC = medial
prefrontal cortex, dACC = dorsal anterior cingulate cortex

Mediation of Aggressive Behavior

Bootstrapping was used to examine the indirect effect of BPD on aggression following low provocation on the TAP via increases in right NAcc activation during provocation-focus during DRT (provocation > neutral contrast). Right NAcc activation did not mediate the association between BPD status and TAP aggression (95% CI = -.784 - .010).

Moderation of Association Between Reactivity to Criticism and Reward Activation

A post-hoc analysis was conducted to test whether the relationship between reactivity to criticism and reward activation during anger rumination is moderated by BPD status. Insula activation during the EF task was not correlated with right NAcc activation during the DRT (provocation > neutral contrast) across the full sample ($r(27)=.11$, $p =.580$). AI activation during EF was mean-centered and entered with BPD status in step 1 of a hierarchical regression model predicting increases in NAcc activation during the provocation-focus condition relative to neutral-focus (provocation > neutral contrast) of the DRT. Together, AI and BPD did not predict significant variance in NAcc activation ($R^2 = .15$, $F[2, 25] = 2.15$, $p = .137$). In step 2, the cross-product of BPD and AI was added to the model. This step did not predict significantly greater amount of variance in NAcc activation; however, a trend toward an interaction was demonstrated ($\Delta R^2 = .06$, $F[1, 24] = 1.91$, $\beta = .53$, $p = .179$). Probing this finding shows that, although the effects are not significant, there is a trend with greater insula activation predicting less NAcc R activation for the controls ($\beta = -.41$, $p = .125$), with this trend absent in the BPD group ($\beta = .10$, $p = .753$) (see Figure 3).
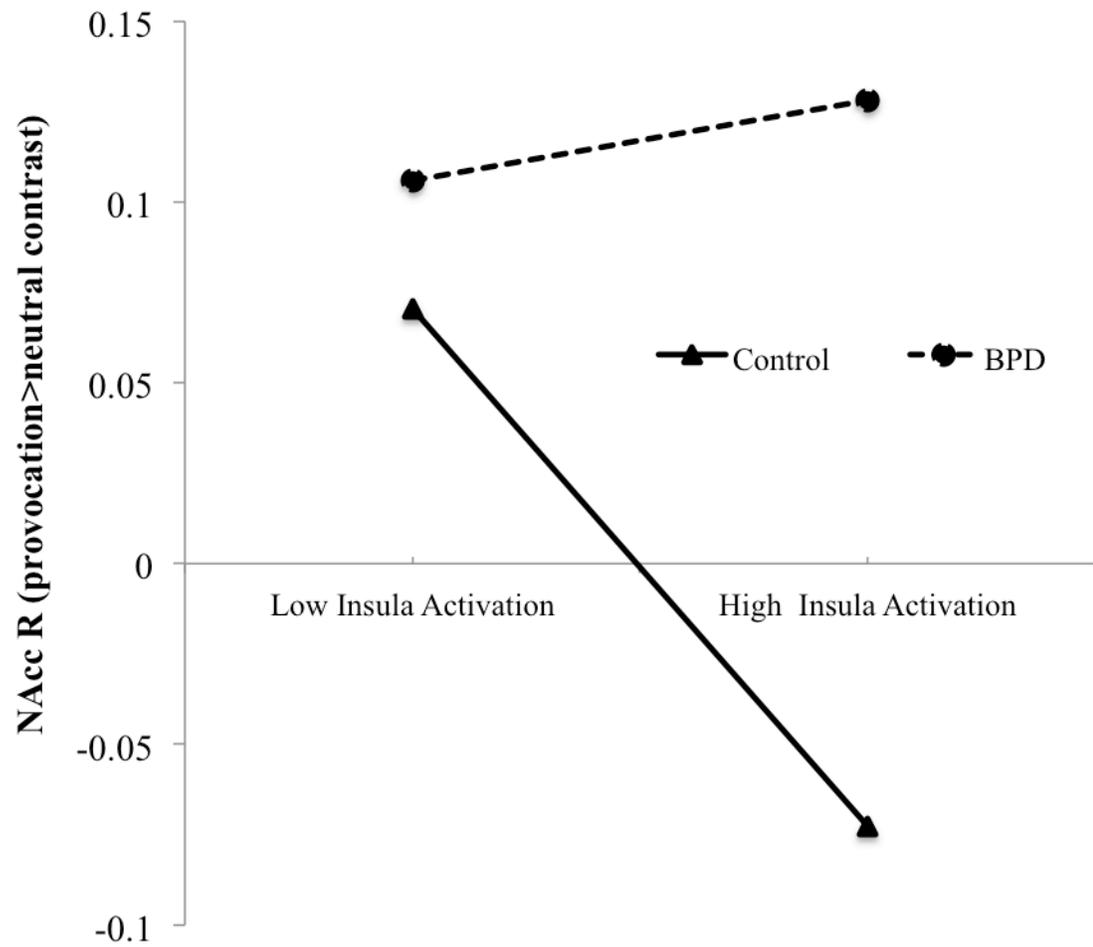
Figure 3. BPD status as a moderator of the effect of insula activation during the essay feedback test on nucleus accumbens activation during provocation-focused thought.

Chapter Four: Discussion

The present study provides preliminary support for the theory that anger rumination is rewarding for individuals with BPD. While engaging in provocation-focused thought, all participants demonstrated greater activation in most of the regions previously associated with anger rumination (dMPFC; Denson et al., 2009), as well as greater recruitment of regions associated with general anger regulation including rumination (LOFC; Fabiansson et al., 2012), suggesting both groups engaged in the task. While controls demonstrated no differences in reward-activation (right or bilateral NAcc ROI activation) between the neutral-focused and provocation-focused conditions, the provocation-focused condition produced significantly more activation in both the right and bilateral NAcc than neutral-focused thought for the BPD group. Combined with the finding of greater activation in the AI and rVLPFC during the prior critical feedback, these findings suggest that individuals with BPD are more sensitive to criticism, try harder to regulate their responses to it, and find the experience of anger ruminating about the provocation more rewarding than controls. This sequence of reactions could explain why individuals with BPD endorse both high trait levels of internally directed negative affect (shame) and externally directed negative affect (anger) and aggression.

No significant findings emerged for the MOFC for any analyses. While this region is also associated with reward, its role may involve valuation of reward, compared to the NAcc which responds to the prescence or absence of rewarding stimuli (Elliott et al., 2003). This component of reward response may be less relevant for anger rumination. Further work should continue to explore this and other reward-relevant regions.

As hypothesized, the BPD group demonstrated greater recruitment of the AI and rVLPFC when receiving critical feedback than controls; however, contrary to hypotheses, there were no significant differences between groups in dACC activation during this task. Some previous findings demonstrate deactivation of the ACC in BPD in response to social rejection, and the present results are consistent with the theory that a strong response in the right insula may lead to suppression of ACC activation and pain reactivity (Ducasse et al., 2014). Alternatively, the critical feedback may have been experienced as both notice of having done poorly on the task and also potentially unfair, but not as an incident of social rejection. It is possible that different effects might be achieved if a more explicitly interpersonal critique had been levied, such as critical feedback regarding the person's potential as a friend after meeting them.

Although there was not a significant correlation between the recruitment of the AI or the rVLPFC during the essay feedback task with the contrast in NAcc activation during provocation-focused from neutral-focused thought, there was a trend toward a significant moderation of the relation between AI recruitment and NAcc activation by BPD status. For controls, greater reactivity to criticism showed a trend toward *inhibiting* reward response to anger rumination, whereas for individuals with BPD, the effect was much smaller and in the opposite direction. Sensitivity to criticism for people very low in BPD features may make those individuals less inclined to think about the provocation, perhaps because rather than becoming angry in response, they simply continue to think about their own mistakes. This is consistent with exit interviews with individuals from both groups, with participants from the BPD group tending to talk about how the task was unfair or their evaluator unreasonable prior to debriefing, whereas controls more often

commented on their own lack of performance on the task. This difference might reflect cognitive biases common to BPD, such a belief that the world is unjust and dangerous (Arntz, Klokman, & Sieswerda, 2005; Young, Klosko, & Weishaar, 2003). The absence of these biases may give controls less fodder for anger rumination, and thus make it less likely to function as a form of immediate emotion regulation.

The values obtained for the amygdala had high variability and numerous outlying values. Due to these difficulties obtaining reliable activation estimates for the amygdala, the hypothesis that increased LOFC activation would be positively associated with increased amygdala activity for the BPD group only was not tested. Further work should examine connectivity between these regions.

Similar to previous research (Denson et al., 2009), the self-focus condition did not produce significantly different levels of reward activation from the other conditions for either group. This may indicate that for nonclinical individuals, none of these forms of thought produce different levels of reward, whereas for individuals with BPD, focusing on the self falls at an indistinguishable midpoint between neutral-focused and provocation-focused. One possible explanation for this finding is that the self-focused prompts may invoke components of anger, particularly when following an angering experience, for the BPD group. Future research utilizing other more specific affective inductions, such as a clear depressive-focus condition or worry-focus, may clarify the extent to which the reward responses demonstrated in this study are specific to anger.

While the present study was able to demonstrate differences between individuals with BPD and healthy controls, it is not clear the extent to which these effects are specific to BPD, especially given the high levels of symptoms of depression, anxiety, and PTSD

endorsed by the sample. Extending this work with clinical comparison groups as well as non-clinical controls would clarify to what extent these findings are due to BPD and not other, comorbid psychopathology. In particular, examining social phobia as an example of individuals who are rejection-sensitive but not prone to the same anger-related symptoms as individuals with BPD might help clarify what neural processes lead to the presence or absence of anger rumination as a coping mechanism.

The BPD group demonstrated more aggression than controls on the TAP, particularly following trials with low provocation. Both groups tended to immediately respond to their opponent's show of aggression (loud noise blast settings) with immediate aggression themselves on the following trial; however, the BPD group persisted more with elevated volumes after the opponent had backed off. It may be adaptive to respond to escalating aggression with aggression, as might be required to defend one's self, but maladaptive not to respond reciprocally to de-escalation. These difficulties returning to baseline are consistent with the biosocial theory of BPD (Linehan, 1993).

This difference in aggressive behavior was largely not associated with differences in neural activation between groups, with only one region of the dACC showing a *lower* level of activation for the BPD group than controls. Contrary to hypotheses, aggressive behavior was also not associated with the BPD group's increased reward activation during provocation-focused thinking. Physical aggression may have less external validity for this sample than verbal aggression, based on the self-report data. Also, in addition to providing the participants with opportunities to aggress, the TAP generates feedback about their performance (wins and losses on the reaction time task) and potential interpersonal threat (the opponent's noise blast settings). If individuals with BPD are

hypersensitive to criticism and threat as suggested by the provocation task findings and previous research (e.g. Sieswerda, Arntz, Mertens, & Vertommen, 2007), the anticipation of potential loss and subsequent noise blast may interfere with aggression-related neural responses. Using a paradigm where the individual aggresses without any critical, performance-based feedback may provide clearer data. A next step may also be to examine how neural findings relate to behavior outside of the lab, such as predicting interpersonal conflict, emotional stability, and impulsive behaviors, perhaps using ecological momentary assessment following a scanning session.

One major limitation of this study is the small sample size. Additionally, the BPD sample, while meeting criteria for the disorder, endorsed a level of BPD-related dysfunction on the dimensional measure that was in the clinical range, but below levels typical for a treatment-seeking sample. BPD symptoms are correlated with many of the MRI-related exclusion criteria (e.g., substance use, obesity, ADHD, multiple medication use), limiting the ability to recruit a sample with more severe psychopathology. As a result of these factors and the inherent variability in BPD, a diagnosis met by one of many possible combinations of symptoms, the present study may lack adequate power to detect some of the potential effects studied. Future studies with larger samples that are able to recruit participants endorsing more BPD-related dysfunction should be conducted to explore these theories with greater power.

These findings have potential clinical implications for the treatment of BPD. If anger rumination following interpersonal criticism and provocation is a rewarding experience specifically for these individuals, that may explain why they do it despite the long-term negative consequences. It also may make it difficult for individuals to stop

engaging in anger rumination, even if they are aware of its detrimental effects. This reward-sensitization could also have effects on other addictive tendencies. Bidirectional cross-sensitization has been demonstrated between substances and naturally occurring rewards, such as food and sex (Avena & Hoebel, 2003a; 2003b; Fiorino & Phillips, 1999), with sensitization to one stimuli increasing responses to the other due to common neural mechanisms (Antelman, Eichler, Black, & Kocan, 1980). Individuals with BPD demonstrate elevated rates of impulsive behaviors such as substance abuse, binge-eating, and risky sexual behavior (American Psychiatric Association, 2013); early sensitization to anger rumination-related reward could contribute to these vulnerabilities.

Interventions targeting anger rumination may need to utilize techniques used to treat other behaviors that are rewarding in the short term such as substance abuse. Motivational interviewing (W. R. Miller & Rollnick, 2013), for example, might help individuals increase their acknowledgment of the effects of their behavior and readiness to make changes. Current approaches to BPD treatment, such as dialectical behavior therapy (DBT; Linehan, 1993; 2014) teach mindfulness skills for increasing awareness of thoughts and emotions and skills for managing urges and tolerating distress without engaging in risky behaviors. Applying these skills specifically to anger rumination may help patients to identify when they feel distress from interpersonal interactions, recognize when they are engaging in anger rumination, and to substitute less harmful behaviors for managing those emotions. Increasing acceptance of initial emotional reactivity to criticism may also reduce the drive to inhibit these emotions and the reward value of externalizing blame. Cognitive emotion regulation strategies have been shown to affect striatal responses to reward cues in a non-clinical sample (Delgado, Gillis, & Phelps,

2008). Further research should examine whether interventions could attenuate the reward activation found in the present study during anger rumination for individuals with BPD, or whether any strategies may help with self-control despite maintained reward activation.

Appendix 1.

Directions and Prompts for Directed Rumination Task

<u>Directions (presented once at the beginning of the task)</u>
Screen 1: In this task, you will be asked to imagine various objects, scenarios and events.
Screen 2: Specific instructions will show up on the screen to tell you exactly what to remember or imagine. Each trial will last for 15 seconds.
Screen 3: Whenever you see a cross, like below, just clear your mind and relax.
Screen 4: Any questions? [Wait for participant response.]
Screen 5: Please lie still as the task will start soon...

<u>Provocation-focus</u>
Think about the feelings and emotions you had during each part of the MRI scan thus far.
Mentally describe the essay evaluator.
Think about the thoughts that you have towards the essay evaluator.
Think about the feelings and emotions you have towards the essay evaluator.
Think about your thoughts during the essay feedback.
Think about your feelings during the essay feedback.

<u>Self-focus</u>
Think about what kind of a person you are.
Think about how other people react to you.
Think about how you interact with people.
Think about the kinds of conversations you have with others.
Think about why you respond to others the way you do.
Think about how you're treated by other people.

<u>Neutral-focus</u>
Think about a bus driving down the street.
Think about the details of a baseball diamond.
Think about pigeons pecking at the ground.
Think about the layout of grocery store aisles.
Think about how a ball point pen works.
Think about the layout of a local coffee shop.

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Riva, P., Romero Lauro, L. J., DeWall, C. N., Chester, D. S., & Bushman, B. J. (2014). Reducing aggressive responses to social exclusion using transcranial direct current stimulation. *Social Cognitive and Affective Neuroscience*. doi:10.1093/scan/nsu053

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Schmahl, C. G., Vermetten, E., Elzinga, B. M., & Bremner, J. D. (2004). A positron emission tomography study of memories of childhood abuse in borderline personality disorder. *Biological Psychiatry*, *55*(7), 759–765. doi:10.1016/j.biopsych.2003.11.007

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informant-reported personality disorder symptoms. *Personality Disorders: Theory, Research, and Treatment, 3*(3), 294-304. doi: 10.1037/a0025654

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Volkow, N. D., Fowler, J. S., Wang, G.-J., & Swanson, J. M. (2004). Dopamine in drug abuse and addiction: results from imaging studies and treatment implications. *Molecular Psychiatry*, *9*(6), 557–569. doi:10.1038/sj.mp.4001507

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Wager, T. D., Davidson, M. L., Hughes, B. L., Lindquist, M. A., & Ochsner, K. N. (2008b). Prefrontal-subcortical pathways mediating successful emotion regulation. *Neuron*, *59*(6), 1037–1050. doi:10.1016/j.neuron.2008.09.006

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Worsley, K. J. (2001). Statistical analysis of activation images. In P. Jezzard, P. M.

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UK: Oxford University Press.

Young, J. E., Klosko, J. S., & Weishaar, M. E. (2003). *Schema therapy: A practitioner's*

guide. New York: Guilford Press.

EDUCATION

Clinical Psychology Resident   2014–2015
Brown University—Alpert Medical School; Providence, RI
Mentor: Shirley Yen, Ph.D.

M.S. in Clinical Psychology   2012
University of Kentucky; Lexington, KY
Thesis: Shame and Borderline Personality Features: The Potential Mediating Role of Anger and
     Anger Rumination
Chair: Ruth A. Baer, Ph.D.

B.S. in Psychology, *Summa cum Laude*   2007
University of Massachusetts Boston; Boston, MA
Honors Thesis: A Preliminary Investigation of the Relationship Between Mindfulness and
     Impulsivity
Mentor: Lizabeth Roemer, Ph.D.

HONORS & AWARDS

Best Poster, Brown University Mind/Brain Research Conference   2015

University of Kentucky
Nietzel Award (Outstanding Psychology Doctoral Graduate)   2015
Exceptional Clinical Performance Award   2014
Presidential Fellowship (research-based Graduate School award; full tuition/stipend) 2013–2014
Excellence in Clinical Performance Award   2013
Research Challenge Trust Fund Fellowship (full tuition/stipend)   2011–2012
College of Arts & Sciences Outstanding Teaching Award   2011
Research Challenge Trust Fund Research Award ($750)   2010
Daniel R. Reedy Quality Achievement Fellowship ($3000 per year)   2009–2012

University of Massachusetts Boston
Psychology Department Honors   2007
Undergraduate Research Funding Award ($500)   2006
Psychological Association of Psi Chi (National Psychology Honor Society)   2006

PEER-REVIEWED PUBLICATIONS

1. **Peters, J.R.**, Vallie, B., Difronzo, M. & Donaldson, S.T. (2007). Role of dopamine D1 receptors in novelty seeking in adult female Long-Evans rats, *Brain Research Bulletin, 74*, 232-236.

2. **Peters, J.R.**, Erisman, S.M., Upton, B.T., Baer, R.A., & Roemer, L. (2011). A preliminary investigation of the relationships between dispositional mindfulness and impulsivity. *Mindfulness, 2,* 228-235.

3. Baer, R.A., Lykins, E.L.B., & **Peters, J.R.** (2012). Mindfulness and self-compassion as predictors of psychological wellbeing in long-term meditators and matched non-meditators. *Journal of Positive Psychology, 7*, 230-238.

4. Baer, R.A, **Peters, J.R.**, Eisenlohr-Moul, T.A., Geiger, P.J., & Sauer, S.E. (2012). Emotion-related cognitive processes in borderline personality disorder: A review of the empirical literature. *Clinical Psychology Review*, 32*,* 359-369.

5. **Peters, J.R.**, Upton, B.T., & Baer, R.A. (2013). Relationships between facets of impulsivity and borderline personality features. *Journal of Personality Disorders, 27,* 547-552.

6. Geiger, P.J., **Peters, J.R.**, Sauer, S.E., & Baer, R.A. (2013). Relationships between cognitive content, cognitive processes, and borderline personality features. *Journal of Personality Disorders, 27*, 457-464.

7. **Peters, J.R**., Eisenlohr-Moul, T.A., Upton, B.T., & Baer, R.A. (2013). Nonjudgment as a moderator of the relationship between present-centered awareness and borderline features: Synergistic interactions in mindfulness assessment. *Personality and Individual Differences, 55*, 24-28.

8. **Peters, J.R.**, Geiger, P.J., Smart, L.M., & Baer, R.A. (2014). Shame and borderline personality features: The potential mediating roles of anger and anger rumination. *Personality Disorders: Theory, Research & Treatment, 5*, 1-9.

9. Geiger, P.J., **Peters, J.R.**, & Baer, R.A. (2014). Using a measure of cognitive distortion to assess the relationship between thought suppression and borderline personality features: A multi-method investigation. *Personality and Individual Differences, 59*, 54-59.

10. Roberts, W., **Peters, J.R.**, Adams, Z.W., Lynam, D.R., & Milich, R. (2014). Identifying the facets of impulsivity that explain the relation between ADHD symptoms and substance use in a nonclinical sample. *Addictive Behaviors, 39*, 1272-1277.

11. **Peters, J.R.**, Eisenlohr-Moul, T.A., Pond, R.S., Jr., & DeWall, C.N. (2014). The downside of being sexually restricted: The effect of sociosexual orientation on relationships between jealousy, rejection, and anger. *Journal of Research in Personality, 51*, 18-22.

12. Derefinko, K.J., **Peters, J.R.,** Eisenlohr-Moul, T.A., Walsh, E.C., Adams, Z.W., & Lynam, D.R. (in press). Trait impulsivity, behavioral disinhibition, arousal, and risky sexual behavior. *Archives of Sexual Behavior.*

13. **Peters, J.R.**, Smart, L.M., & Baer, R.A. (in press). Dysfunctional responses to emotion account for relationships between rejection sensitivity and borderline personality features. *Journal of Personality Disorders.*

14. Chester, D.S., DeWall, C.N., Derefinko, K.J., Estus, S., **Peters, J.R.**, Lynam, D.R., & Jiang, Y. (in press). Monoamine oxidase A (MAOA) genotype predicts greater aggression through impulsive reactivity to negative affect. *Behavioural Brain Research.*

15. Smart, L.M., **Peters, J.R.**, & Baer, R.A. (in press). Development and validation of a measure of self-critical rumination. *Assessment.*

16. Lee, D.C., **Peters, J.R.**, Adams, Z.W., Lynam, D.R., & Milich, R. (in press). Specific dimensions of impulsivity are differentially associated with daily and non-daily cigarette smoking status in young adults. *Addictive Behaviors.*

17. Geiger, P.J., **Peters, J.R.**, & Baer, R.A. (in press). Cognitive distortions under cognitive load mediate the relationship between borderline personality feature severity and goal-interfering emotion dysregulation. *Journal of Experimental Psychopathology.*

18. **Peters, J.R.**, Smart, L.M., Eisenlohr-Moul, T.A., Geiger, P.J., Smith, G.T., & Baer, R.A. (in press). Anger rumination as a mediator of the relationship between mindfulness and

aggression: The utility of a multidimensional mindfulness model. *Journal of Clinical Psychology.*

BOOK CHAPTERS

1. Baer, R.A. & **Peters, J.R.** (2011) Assessment of acceptance and mindfulness in behavioral medicine. In: McCracken, L. M. (Ed.), *Acceptance and Commitment Therapy and Mindfulness-based Approaches in Behavioral Medicine: An Integrative Clinical and Research Guide.* Oakland, CA: New Harbinger Publications.

2. Eisenlohr-Moul, T.A., **Peters, J.R.**, & Baer, R.A. (2013). Using mindfulness effectively in clinical practice: Two case studies. In: Wedding, D. & Corsini, R. J. (Eds.) *Case Studies in Psychotherapy, 7th Edition.* Stamford, CT: Cengage Learning.

3. Eisenlohr-Moul, T.A., **Peters, J.R.**, & Baer, R.A. (2015). Assessment of mindfulness in clinical research. In: Ostafin, B.D., Robinson, M.D., & Meier, B.P. (Eds). *Handbook of Mindfulness and Self-Regulation*. New York, NY: Springer.

GRANTS AWARDED

Research Support Grant, University of Kentucky (PI: Baer, Ph.D.)   2013-2014
Title: "The Rewarding Nature of Anger Rumination in Borderline Personality
Disorder: An fMRI Investigation"
Role: Co-Investigator
Direct costs: $7550

RESEARCH APPOINTMENTS & POSITIONS

Research Assistant (Statistics/Data Analysis; Clinical Research)   2011–2014
NIDA (DA005312): "Center for Drug and Alcohol Research Translation"
University of Kentucky, Lexington, KY
PI: Michael Bardo, PhD; Richard Milich, PhD; Donald Lynam PhD

Graduate Student Researcher   2009–present
Baer Mindfulness Lab; University of Kentucky, Lexington, KY
Lab Director: Ruth Baer, PhD

Research Coordinator   2007–2009
Program for Psychotherapy, Cambridge Health Alliance, Cambridge, MA
NIMH (5R01MH078100-04): "Comparing Alternative Dimensional Approaches to
Personality Diagnosis"
PI: Drew Westen, PhD; Co-I/Site Supervisor: Rebecca Drill, Ph.D.

Research Assistant   2005–2007
Emotions Research Lab; University of Massachusetts Boston, Boston, MA
Lab Director: Lizabeth Roemer, PhD

CLINICAL POSITIONS

Clinical Psychology Resident, Adult Track   2014–present
Brown University Clinical Psychology Training Consortium, Alpert Medical School
Butler Hospital and Rhode Island Hospital
Rotations: DBT Partial Hospital; Alcohol and Drug Partial Hospital; Mood Disorders
Inpatient Unit

Graduate Student Therapist; Clinic Assistant Coordinator   2010–2014
Jesse G. Harris Psychological Services Center

Peer Supervisor   2013–2014
University of Kentucky Clinical Psychology Program

Primary Therapist   2011–2012
Chrysalis House Rehabilitation Center
Women's Residential Treatment Center for Substance Abuse

TEACHING & MENTORING EXPERIENCE

Invited Lecturer, University of Kentucky
    Dialectical Behavior Therapy Seminar (Graduate): Emotion Regulation Skills   2013
    Clinical Interviewing Seminar (Graduate): Diversity; Challenging Clients   2012, 2013
    Honors Psychology Seminar (Undergraduate): Applying to Graduate School   2012
    Clinic Assistant Seminar (Undergraduate): DBT; Mindfulness   2012; 2013

Undergraduate Honors Thesis Mentor, Baer Lab, University of Kentucky   2011–2013

Laboratory Instructor, University of Kentucky
Graduate Level
    ANOVA   2010
    Regression   2011
Undergraduate Level
    Introduction to Psychology   2009, 2010
    Harris PSC Clinic Assistant Seminar (Special Topics in Clinical Psychology)   2010–2011

Curriculum Development, University of Kentucky
    Committee to Restructure Psychology 100 Learning Objectives and Laboratory Course   2010

Teaching Assistant Training, University of Kentucky
    Co-Leader, Psychology Department Teaching Assistant Training   2010
    Teaching Assistant Coordinator for Introductory Psychology Course   2010

Individual Tutoring, University of Massachusetts Boston
    Undergraduate Psychology Statistics   2006–2007

PROFESSIONAL ACTIVITIES

Ad Hoc Reviewer: *Personality Disorders: Theory, Research, and Treatment; Assessment; Journal of Clinical Psychology; Psychiatry Research; Cognitive Therapy and Research; Journal of Nervous and Mental Disease; Behavioural Pharmacology; Journal of Psychopathology and Behavioral Assessment; Mindfulness; Spanish Journal of Psychology*

Clinical Psychology Residents' Representative: Training Committee   2014–2015
Department of Psychiatry, Alpert Medical School at Brown University

Fellow: Mind and Life Summer Research Institute   2013

Founding Member: Quantitative Psychology Interest Group   2012–2015
University of Kentucky

Founding Member: Diversity Task Force   2011–2015
Clinical Psychology Program, University of Kentucky
    Authored LGBT section of Diversity Manual   2012

Representative: Harris Psychological Services Center
    Clinical Psychology applicant interview presentation and Q&A   2011–2013
    Outreach and recruitment presentation to University of Kentucky Psychiatry residents   2010