Published in final edited form as:
J Abnorm Psychol. 2009 August ; 118(3): 531–544. doi:10.1037/a0016392.

Disentangling Emotion Processes in Borderline Personality Disorder: Physiological and Self-reported Assessment of Biological Vulnerability, Baseline Intensity, and Reactivity to Emotionally-Evocative Stimuli

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Abstract

This study investigated Linehan's (1993) theory that individuals meeting criteria for borderline personality disorder (BPD) have high biological vulnerability to emotion dysregulation, including high baseline emotional intensity and high reactivity to emotionally-evocative stimuli. Twenty individuals with BPD, 20 age-matched individuals with generalized social anxiety disorder (SAD), and 20 age-matched normal controls (NC) participated in two separate emotion induction conditions, a standardized condition and a personally-relevant condition. Respiratory sinus arrhythmia (RSA), skin conductance response (SCR), and self-report measures were collected throughout the experiment. BPD participants displayed heightened biological vulnerability compared with NC as indicated by reduced basal RSA. BPD participants also exhibited high baseline emotional intensity, characterized by heightened SCR and heightened self-reported negative emotions at baseline. However, the BPD group did not display heightened reactivity as their physiological and self-reported changes from baseline to the emotion inductions tasks were not greater than the other two groups.

Keywords

borderline personality disorder; emotion regulation; vulnerability; intensity; reactivity; psychophysiology; vagal tone

Introduction

Borderline Personality Disorder and Emotion Dysregulation

Borderline personality disorder (BPD) is a life-threatening disorder characterized by severe cognitive, behavioral, and emotional dysregulation. The prevalence of BPD in the general

population is between 1% to 5.9% (Samuels et al., 2002; Torgerson, Kringlen, & Cramer, 2001; Swartz, Blazer, George, & Winfield, 1990; Grant et al., 2008) and up to 40% of high utilizers of inpatient mental health care services (Geller, 1986) and approximately 15% of outpatients are diagnosed with BPD (Koenigsberg, Kaplan, Gilmore, & Cooper, 1985; Widiger & Weissman, 1991, Torgerson et al., 2001). The implications of these statistics are compelling in view of the extremely high lifetime prevalence of self-injurious acts (up to 84%, Clarkin, Widiger, Frances, Hurt, & Gilmore, 1983; McGlashan et al. 2005), the rate of suicide attempts (over 70% of BPD patients, Soloff et al., 2000; Zisook, Goff, Sledge, & Schucter, 1994; Soloff, Lynch, & Kelly, 2002), and the 10% suicide rate (Frances, Fyer, & Clarkin, 1986; Paris, Brown, & Nowlis, 1987).

Dialectical Behavior Therapy (DBT) is at present the most empirically-supported treatment for BPD (see Lieb, Zanarini, Linehan, & Bohus, 2004) and is based on Linehan's (1987) theory of emotion dysregulation as the core characteristic of the disorder. Linehan (1993) proposes a biosocial developmental model which states that BPD criteria are caused jointly by a *biological vulnerability* to emotion dysregulation and an invalidating environment (one that arbitrarily negates, rejects, or dismisses an individual's behaviors). According to Linehan's model, emotion dysregulation in BPD includes high *baseline negative emotional intensity* as well as high *emotional reactivity* which refers to changes in intensity of emotional responding after presentation of an emotionally-evocative cue. High emotional intensity and reactivity are proposed to be sequelae of emotional vulnerability and emotion dysregulation occurs as the individual is further unable to effectively modulate the intensity of the emotional response. When the emotionally vulnerable individual is placed in a chronically invalidating environment, the constant transaction between the two lead to the subsequent development of BPD.

There is a growing body of research examining Linehan's proposed factors of emotion dysregulation in BPD as well as the biosocial model in which it is based (see Rosenthal, et al., 2008 for review). Several studies have examined the *high intensity* and *high reactivity* components of emotion dysregulation. In contrast, fewer studies have examined the biological dimension of the biosocial model, which specifies that BPD individuals are *biologically vulnerable* to emotion dysregulation. Indeed, little is known about the biological factors that render a BPD individual vulnerable to emotional intensity and reactivity and/or more broadly, emotion dysregulation. The review below focuses on *biological vulnerability*, *baseline emotional intensity*, and *emotional reactivity* in BPD, the limitations of the research to date, and the rationale for the current study.

Linehan's Biosocial Theory: Biological Vulnerability

Over two decades of research has identified various biological factors that may influence the development of BPD. Studies of neurotransmitter dysfunction, particularly within the serotonergic system, suggest a link between reduced serotonergic activity and impulsivity in BPD (see Gurvits, Koenigsberg, & Siever, 2000 for review; Ni, et al., 2006). Electrophysiological studies in BPD (see Boutros, Torello, & McGlashan, 2003 for review) have reported abnormal brain electrical activity such as EEG slowing, lower EEG vigilance (Hegerl, et al., 2007), increased REM density (Battaglia, et al., 1999) decreased REM

latency (Battaglia, et al., 1993), and reduced right hemisphere gamma phase synchrony (Williams, Sidis, Gordon, & Meares, 2006), which together have been proposed to contribute to the impulsiveness and affective lability associated with the disorder. More recently, structural imaging studies have reported decreased hippocampal and amygdala volume in BPD (Driessen, et al., 2000; Tebartz van Elst, et al., 2003; Schmahl, Vermetten, Elzinga, & Bremner, 2003a). Functional imaging studies have identified altered baseline metabolism in prefrontal regions as well as dysfunctional frontolimbic networks in BPD (see Schmahl & Bremner, 2006) in response to stressful challenges. While these reported aberrations have been proposed as biological markers associated with BPD criteria, what remains unclear is whether these factors reflect a specific vulnerability to emotion dysregulation, the central mechanism proposed by Linehan's model.

Recent evidence has linked vagal tone, referring to parasympathetic influence on the heart, with emotion regulatory processes. Porges' polyvagal theory (Porges, Doussard-Roosevelt, & Maiti, 1994) suggests that activation of the myelinated vagal system terminating at the sinoatrial node of the heart is positively associated with various attentional and emotion regulatory processes. Substantial literature suggests that basal vagal tone is associated with individual differences in emotional vulnerability (Porges, 1995a; Porges, 1995b; Beauchaine, 2001; Butler, Wilhelm, & Gross, 2006). Additionally, studies of adult clinical samples have reported reduced basal vagal tone in several Axis I disorders (Thayer, Friedman, & Borkovec, 1996; Lyonfields, Borkovec, & Thayer, 1995; Friedman & Thayer, 1998; Cohen et al., 1998; Cohen et al., 2000) which has been conceptualized as indicating difficulties in effectively interacting emotionally with the environment. Collectively, these findings have led many investigators to conceptualize basal vagal tone as an index of *biological vulnerability* to emotion dysregulation. Despite such evidence, little to no research has investigated vagal tone in BPD.

Linehan's Emotion Dysregulation Theory: High Baseline Intensity and High Reactivity

High Intensity—The primary method for assessing heightened emotional intensity in BPD has been through self-report questionnaires. Consistent with Linehan's theory, these studies have reported higher negative emotional intensity in BPD compared with non-clinical controls, other Axis II individuals, as well as individuals with bipolar disorder (Levine, Marziali, & Hood, 1997; Koenigsberg et al., 2002; Henry et al., 2001). Other studies have reported a positive correlation between negative emotional intensity and BPD features (Yen, Zlotnick, & Costello, 2002; Rosenthal, Cheavens, Lejuez, & Lynch, 2005; Cheavens et al., 2005) as well as intense negative emotions following social interactions (Russell, Moskowitz, Zuroff, Sookman, & Paris, 2007). Ambulatory monitoring studies indicate that BPD participants report higher unpleasant emotional intensity (Stein, 1996; Ebner-Priemer et al., 2007) and higher aversive tension (Stiglmayr et al. 2005) than non-clinical participants in a naturalistic context.

The self-report studies provide substantial evidence for subjective, heightened negative emotional intensity in BPD. These data are commonly interpreted as evidence for heightened emotional reactivity in BPD. As noted above, however, Linehan's model defines high reactivity as *stimulus-related* changes in emotional intensity. Although self-report

studies provide evidence of high negative emotional intensity in BPD, these studies do not directly assess changes in emotional intensity after presentation of an emotionally-evocative stimulus (hence, *reactivity*). Indeed, a major limitation in the self-report literature is that baseline intensity and reactivity are often confounded. Thus, it remains unclear whether such self-reported intensity in BPD is indeed due to increased reactivity, increased baseline levels of emotional intensity, or both.

High Reactivity—In contrast to the self-report studies, physiological studies have assessed reactivity more directly. However, findings from this literature are mixed. Herpertz and colleagues (1999, 2000, 2001a) published three physiological emotion studies in which BPD participants viewed neutral, positive, and negative valenced slides while physiologically monitored (Herpertz, Kunert, Schwenger, & Sass, 1999; Herpertz et al. 2000, Herpertz et al., 2001a). There were no indications of heightened emotional reactivity in BPD. Interestingly, in the first two studies (Herpertz, et al., 1999; Herpertz et al., 2000), BPD individuals exhibited significantly *lower* skin conductance (SC) magnitude, a measure of sympathetic activity, across all slide valences compared with the control groups, suggesting lower intensity of emotional responding across the task. Similarly, in a study where abused women with BPD, PTSD, or neither disorder listened to personally-relevant abandonment and abuse scripts, Schmahl et al., (2004b) found a tendency towards greater skin conductance responses to the abandonment script in the BPD group, although this did not reach the level of significance.

Studies by Ebner-Priemer, in contrast, (Ebner-Priemer et al., 2005, 2007) offer support for physiological reactivity in BPD. Ebner-Priemer et al., (2007) found that non-medicated BPD individuals exhibited a heightened degree of emotional heart rate (i.e. changes in heart rate that is devoid of movement influences) compared with healthy controls in an ambulatory monitoring paradigm. Ebner-Priemer et al., (2005) also reported larger startle response magnitude (i.e., higher reactivity) and slower habituation in BPD compared with psychologically healthy controls. In this study, the investigators also compared differences in physiological responding between BPD participants high and low in present-state dissociation. Results indicated that BPD participants with low present-state dissociation exhibited higher EMG responses than BPD participants high in present-state dissociation.

One study to date has assessed vagal withdrawal as an indicator of emotional reactivity in BPD. According to Porges' polyvagal theory (Porges, Doussard-Roosevelt, Portales, & Greenspan, 1996), the appropriate use of the "vagal brake" is essential for proper engagement with tasks that require attention, social interaction, and emotion regulation. Consistent with this theory, recent evidence suggests that excessive vagal withdrawal reflects heightened emotional *reactivity* or emotionally dysregulated states such as panic (Yeragani et al., 1990), anger (Donzella, Gunnar, Krueger, & Alwin, 2000), worry (Thayer et al., 1996; Lyonfields et al., 1995) and hyperarousal in response to traumatic memories (Sack, Hopper, & Lamprecht, 2004).

Austin, Riniolo, & Porges (2007) monitored respiratory sinus arrhythmia (RSA), an index of vagal tone, and heart period (HP) while BPD participants and non-clinical controls watched film clips. Although there were no baseline differences, the BPD group displayed a decrease

in RSA and HP while the control group displayed the opposite pattern throughout the course of the experiment. The authors interpreted the vagal withdrawal exhibited by the BPD participants to indicate heightened emotional reactivity. However, it is unclear whether the vagal withdrawal exhibited by the BPD subjects was stimulus-linked (i.e. in response to the films) or due to the passage of time. Additionally, the authors excluded BPD participants with any additional disorders. Given that the rates of comorbidity between BPD and other disorders is as high as 98% (see Skodol et al., 2002 for review), generalizability of these results is limited.

In summary, although the physiological literature provides some evidence for heightened emotional reactivity in BPD, the findings within this literature are mixed and fraught with limitations. The mixed findings can be accounted for by the variability in the stimuli employed across investigations. Some studies included standardized stressors (e.g., slides) and others idiographic stressors (e.g., abandonment scripts). The discrepant methodologies make it difficult to determine whether BPD individuals are more reactive to certain cues or whether other methodological factors are influencing the results. Incorporating both standardized and idiographic stimuli under one methodological umbrella is needed to address this question. A second limitation is that very few of these studies have included clinical comparison groups. Given the recent movement towards a unifying theory of emotion dysregulation in Axis I disorders (Barlow, Allen, & Choate, 2004), additional comparisons of the emotion dysregulation profile in BPD versus other Axis I disorders is warranted. A majority of studies have also failed to control for the impact of dissociation on results, despite evidence indicating that present-state dissociation mitigates autonomic output in BPD (Ebner et al., 2005). Finally, there has been a lack of comprehensive autonomic assessments in the physiological studies to date. Contemporary views of autonomic activity emphasize that contributions of the sympathetic (SNS) and parasympathetic systems (PNS) may vary reciprocally, coactively, or independently (Berntson, Cacioppo, & Quigley, 1991) and therefore, simultaneously assessing SNS and PNS activity is crucial to understanding emotional processing systemically.

The Current Study

The study presented here examined three core components of Linehan's model (*biological vulnerability*, *baseline emotional intensity*, and *emotional reactivity*) and addressed the limitations of previous research. Three groups of participants, BPD, generalized social anxiety disorder (SAD), and normal controls (NC) engaged in a standardized (films) and idiographic (imagery) paradigm while physiologically monitored. The rationale for selecting SAD as a clinical control was as follows: First, given the substantial evidence for emotion dysregulation in anxiety disorders, selecting an anxiety disorder control group would allow for comparisons between BPD and other groups where emotion dysregulation processes have been indicated. Second, given the high (approximately 56%) comorbidity of BPD with PTSD (Zanarini, et al., 1998), selecting a PTSD control group would limit the specificity between the two samples. Further, given the existing theoretical debate around GAD as a separate diagnostic category as indicated by its high comorbid rates with depression as well as other anxiety disorders (See Nutt, Argyropoulos, Hood, & Potokar, 2006), selecting this group as a clinical control would also limit the specificity between the two samples. Indeed,

the “diagnostically unclean” GAD group would render it difficult to ascertain what clinical conditions BPD is distinct from. SAD was decidedly the most appropriate clinical control group because 1) It would allow for comparability with the current data on emotion processes in SAD and anxiety disorders in general, 2) Its rate of comorbidity with BPD (45–50%, Zanarini, et al., 1998; Zanarini, Frankenburg, Hennen, Reich, & Silk, 2003) is lower than that of PTSD and depression, and 3) its diagnostic profile is more distinctive than that of GAD.

The primary hypotheses were as follows: 1) BPD individuals would exhibit *biological vulnerability* to emotion dysregulation as indicated by reduced basal RSA compared with the SAD and NC groups, 2) BPD individuals would demonstrate heightened *baseline negative emotional intensity* compared with the SAD and NC groups as indicated by higher electrodermal responding and higher self-reported negative emotions at baseline and 3) BPD individuals would exhibit heightened *emotional reactivity* compared with the SAD and NC groups as indicated by greater changes from baseline to emotion-eliciting tasks in physiological and self-report measures.

The limitations reviewed above were addressed by first examining Linehan’s largely untested notion of *biological vulnerability* in BPD by comparing baseline differences in vagal tone. Second, baseline negative emotional *intensity* and stimulus-induced *reactivity* were separately assessed, thus allowing the two constructs to be delineated. Emotional *reactivity* was assessed across three core emotions (sadness, anger, and fear) and across both standardized and personally-relevant conditions. The addition of a clinical control group, SAD, allowed for comparisons of the BPD emotion dysregulation profile with that of another clinical population. Additionally, dissociative state was controlled for and both sympathetic and parasympathetic indices were measured.

Method

Participants

Three groups of participants were recruited to take part in the study: individuals with BPD, individuals with SAD, and individuals with no current Axis I disorders or BPD (NC). Participants were recruited through flyers and internet postings throughout the community, local hospitals, and clinics, and by outreach to BPD participants in existing treatment studies at the University of Washington Behavioral Research and Therapy Clinics (BRTC). Only females were recruited because of indicated differences in emotional reactions (Fischer, 2000) as well as differences in cardiovascular control and vagal activity between genders (Fyan et al., 1994). Given the challenges of diagnosing young children with personality disorders, only participants over the age of 18 were included. Exclusion criteria included schizophrenia, schizophreniform, and schizoaffective disorders, psychosis NS, bipolar disorder, current substance dependence, as well as the following physical conditions: epilepsy or seizure disorder, heart disease, and asthma. Participants were also excluded if they were taking any psychotropic medication other than SSRIs, major tranquilizers, antihistamies, or beta blockers. Color-blindness was also an exclusion criterion as one of the baseline tasks in the study required the identification of colors. Finally, SAD and NC participants meeting four or more BPD criteria or the impulsivity and self-harm/suicidality

criteria from the *Structured Clinical interview for DSM-IV Axis II (SCID-II)- BPD* (First, Spitzer, Gibbons, Williams, & Benjamin, 1996) were excluded to ensure the three samples remained distinct. Seventeen SAD and NC participants were therefore excluded after screening based on these criteria. All participants were between ages 18–45 and were matched on age (±2 years). The final sample resulted in 20 BPD, 20 SAD, and 20 NC participants. Effect sizes from pilot data as well as other physiological studies with similar designs suggested that, with a sample of n=20 in each group, we would have a power of .68 to detect baseline physiological differences as well as a power of .85 to detect differences in reactivity.

Participant Demographics—Mean age for the three groups were: BPD=23.55, SAD=23.90, NC=23.30, indicating successful matching. See Table 1 for ethnic and marital demographics and Table 2 for rates of Axis I comorbidiy for the BPD and SAD groups. A Mann-Whitney test indicated that there was not a significant difference in the number of comorbid diagnoses between the BPD and SAD groups, with an average rank of 21.42 for the BPD group and 17.7 for the SAD group ($p > .05$). Four (20%) of the BPD participants were on SSRIs (one on citalopram, two on fluoxetine, one on sertraline), one (10%) SAD participant was on citalopram, and none of the NC participants were on psychotropic medications. There were no significant differences in the number of medications with an average rank of 22.00 for the BPD group and 19.00 for the SAD group, ($p > .05$).

Measures

Screening and Descriptive—The *Structured Clinical interview for DSM-IV Axis I (SCID-I)* (First, Spitzer, Gibbon, & Williams, 1995) and *Structured Clinical interview for DSM-IV Axis II (SCID-II)- BPD* (First, et al., 1996) were administered to screen for Axis I disorders and BPD, respectively. All SCIDs were conducted by the principal investigator of the study, who was trained to reliability with assessors from the larger treatment trials within the UW BRTC. The *Demographic Data Survey (DDS)* (Linehan, unpublished, 1982) was administered to obtain a wide range of demographic data including age, height, weight, and ethnicity.

Self-Report measures of emotion and emotion regulation

Trait Measures: The *Difficulties in Emotion Regulation Scale (DERS)* (Gratz & Roemer, 2004), was administered as a trait measure of emotion regulation. The DERS is a 36-item measure that assesses individuals' typical levels of emotion dysregulation across six domains: non-acceptance of negative emotions, inability to engage in goal-directed behaviors when experiencing negative emotions, difficulties controlling impulsive behaviors when experiencing negative emotions, limited access to emotion regulation strategies perceived as effective, lack of emotional awareness, and lack of emotional clarity. The DERS has high internal consistency (α=.93) and good test-retest reliability (ρ_I=.88). The *Acceptance and Action Questionnaire (AAQ)*-16 item (Hayes et al, 2004), a measure of experiential avoidance was also administered. Finally, the *State-Trait Anger Inventory (STAXI), Trait Anger* subscale (Spielberger, Krasner, & Solomon, 1988), was administered to measure trait levels of anger (Cronbach's alpha for women = 0.75; re-test correlation for women between 0.70 and 0.76).

State Measures: After every baseline and emotion induction, participants reported their emotional states using the *Visual Analogue Scale (VAS)* (Haines J., Williams C., Brain K., & Wilson, 1995), a measure of subjective reactions on a scale of 1–100 on the following bipolar dimensions: *relaxed-tense*, *calm-angry*, *unafraid-afraid*, *happy-sad*, *normal-unreal*, *relieved-uptight*, *contented-ashamed*, and *accepting-punishing*. After every emotion induction, participants also completed the *Dissociative State Scale (DSS)* (Stiglmayr, Shapiro, Stieglitz, Limberger, & Bohus, 2001), a 21-item self-report inventory assessing the duration and intensity of acute somatic and psychological dissociation, which was included as a covariate. The scale was developed at the Borderline Research Unit, University of Freiburg. Test retest reliability (1-week daily data recording, k= .8) and internal consistency estimates (Cronbach's alpha = 0.9) are high.

Physiological Measures

Respiratory sinus arrhythmia (RSA): RSA was used as the index of vagal tone and was measured by assessing the high-frequency (HF) band of spectral analysis (Berntson et al., 1997), which decomposes ECG R-wave time series into three heart rate variability frequency ranges through Fast-Fourier transformations. Spectral analyses were conducted using Mindware Technologies HRV 2.33 software (Westerville, OH) which detects questionable R-R intervals based on the overall R-R distribution using a validated algorithm to aid artifact detection and editing (Berntson, Quigley, Jang, & Boysen, 1990) The low-frequency range is less than 0.04 Hz, mid-frequency from 0.04–0.15 Hz, and the high-frequency range is greater than .15 Hz. Research on heart rate and heart rate variability suggest that parasympathetic/vagal activity influences all frequencies of <0.5, while sympathetic activity affects frequencies of <0.15 (Berntson et al., 1997). High-frequency spectral densities were calculated across 30-second epochs for each baseline and emotion induction.

Skin Conductance Response (SCR): Skin conductance was measured as an index of sympathetic responding. Nonspecific skin conductance responses (SCRs) were scored using the Mindware Technologies EDA 2.40 program which calculated the number of fluctuations exceeding 0.05_{μ}S across one-minute epochs. A programmable rolling filter was employed for artifact detection and editing.

All physiological measurements were collected using a BIOPAC 5-channel data acquisition system (Biopac Technologies, Model MP100). Data were digitized at 1000 samples per second and set for a gain of 1000 using low (35Hz) and high (.05Hz) pass filters. ECG data were collected at the left and right wrists via disposable Ag-AgCl snap electrodes (Biopac Technologies Model EL503) with a third electrode attached to the ankle for ground reference. SCR was measured by 2 6-mm electrodes (Biopac model TSD203) with electrolyte gel attached to the medial phalanges of two fingers on the non-dominant hand. All SC application was done according to field standards (Fowles et al., 1981).

Emotion Induction Conditions

Standardized Condition/Emotion Films—Films known to reliably elicit targeted emotions were selected from a sample developed by Gross & Levenson (1995). Each film

lasted approximately 166 seconds. The sad film was a 2:51 minute clip from the movie "The Champ," which showed a young boy crying at his father's death. The fear film was a 3:29 minute clip from the movie "Silence of the Lambs," which depicted a basement chase scene. The anger film was a 4:06 minute scene from the movie, "My Bodyguard" which showed a young teenage boy being bullied by another young man. Finally, the Neutral film was a silent 1:30 minute clip depicting colored bars. All films were presented from on a 16.5" ×12.5" computer monitor.

Personally-relevant Condition/Imagery—Development of the imagery task followed procedures described by Pitman and colleagues (Pitman, Orr, Forgue, de Jong, & Claiborn, 1987). Two to five days prior to the experimental procedures, participants were interviewed by a trained assessor who asked them to describe in writing the most recent or vivid event in which they felt sad, afraid, angry, and emotionally neutral (control). The assessor prompted the participant when needed in order to ensure the described event was sufficient to elicit an emotional response. Only events where the intensity of the emotion experienced was rated as an eight or higher on a scale of 1–10 were used. Each participant's script was then rewritten to take approximately two minutes when read out loud. In accordance with the Pitman et al., (1987) protocol, scripts were also evaluated by the trained assessor and the principal investigator to ensure that each script (with the exception of the neutral script) consisted of five emotions, five sensations, and five thoughts. The rewritten scripts maintained the exact phrases of the participants and were recorded by the principal investigator in a neutral voice tone. On the day of the experiment, the recording was played back to the participant and they were instructed to imagine themselves back in the situation described.

Procedure

The experimental procedures were divided into two separate sessions, one for each condition. Counterbalancing procedures were applied to the order of the condition (standardized vs. personally-relevant) and type of emotion induction (sadness, fear, anger, neutral). At the start of each experimental session, participants engaged in a four-minute "True Baseline" where they were instructed to maintain wakefulness and sit quietly and still. Participants then engaged in a four-minute "Vanilla Baseline" period (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992), where they engaged in a non-stressful, non-demanding cognitive task requiring them to count the number of times a specified color appeared on a screen. The purpose of using the vanilla baseline was to control for any negative emotions that the participant may experience during a true baseline period.

Following the first True and Vanilla Baselines, participants engaged in either the standardized or personally-relevant condition, depending on the counterbalancing method. Between each emotion induction, participants engaged in a five-minute vanilla baseline. Participants were physiologically monitored throughout the entirety of the experimental session.

All were instructed to avoid ingestion of caffeine and tobacco on the day of and refrain from taking any over the counter medications 24 hours prior to the physiological procedures.

Data Reduction and Analysis

Analyses were conducted using SPSS, version 13.0. A visual inspection of the physiological data indicated no distinct linear or polynomial relationship, therefore, a Mixed-Model ANOVA (MMANOVA) approach was employed for all physiological analysis. The mixed model (Littell, Milliken, Stroup, & Wofinger, 1996) is an extension of the standard general linear regression and is appropriate when the data consist of independent subjects or clusters, and the regression model for each subject or cluster can be assumed to be a random deviation from some population parameter estimate. The MMANOVA models separate means per group over time, where our interest focuses on (a) group by time interaction, which assesses whether the differences between groups vary over time and (b) group effect which assesses the difference between groups pooled across the longitudinal period. The MMANOVA is similar to repeated measures ANOVA in that it models the clustering of the repeated data but unlike repeated measures ANOVA, offers more flexibility to model various covariance structures and accommodate missing data. In addition, the MMANOVA framework, as implemented in SPSS, allows within-group and between-group contrasts to be investigated over various ranges of time.

A priori hypotheses involving multiple comparisons are specified below. Adjustments were not employed for planned comparisons. However, for all tasks where a priori hypotheses were not stated, omnibus F-tests, followed by post-hoc comparisons were conducted. All significance tests were two-tailed.

Sample Descriptives and Trait Measures of Emotion and Emotion Regulation

—Sample descriptives and self-report questionnaires of trait emotion and emotion regulation were analyzed using One-way ANOVA.

Biological Vulnerability

RSA: A 3×2 Repeated-Measures ANOVA investigating the relationship between Group and the two true baselines (Group: BPD, SAD, NC; Task: Films true baseline, Imagery true baseline) indicated no Group*Task interactions, F=.72 (2,56) p=.49. Similarly, 3×2 Repeated-Measures ANOVA investigating the relationship between Group and the two vanilla baselines (Group: BPD, SAD, NC; Task: Films vanilla baseline, Imagery vanilla baseline) indicated no Group*Task interactions F=1.41 (2,56) p=.25. Therefore, true baselines were combined across the Films and Imagery conditions as well as the vanilla baselines across the Films and Imagery conditions. Differences between the three groups were then analyzed using Mixed-Model ANOVA with “Epoch” (i.e., eight epochs/thirty seconds each) as the within-subjects factor and “Group” (BPD, SAD, and NC) as the between-subject factor.

Baseline Negative Emotional Intensity: SCR and Self-report/VAS

SCR: A 3×2 Repeated-Measures ANOVA investigating the relationship between Group and the two true baselines (Group: BPD, SAD, NC; Task: Films true baseline, Imagery true baseline) indicated no Group*Task interactions, F=2.22 (2,54) p=.12. Similarly, 3×2 Repeated-Measures ANOVA investigating the relationship between Group and the two vanilla baselines (Group: BPD, SAD, NC; Task: Films vanilla baseline, Imagery vanilla

baseline) indicated no Group*Task interactions F=.05 (2,54) p=.95. Therefore, true baselines were combined across the Films and Imagery conditions as well as the vanilla baselines across the Films and Imagery conditions. Differences between the three groups were then analyzed using Mixed-Model ANOVA with "Epoch" (i.e., eight epochs/thirty seconds each) as the within-subjects factor and "Group" (BPD, SAD, and NC) as the between-subject factor.

Self-report/VAS: A composite score of negative emotion was computed (i.e., "VASNeg," consisting of items *relaxed-tense, relaxed-anxious, calm-angry, unafraid-afraid, happy-sad, relieved-uptight and contented-ashamed*). Square root transformations were employed where violations of normality were violated. Differences in combined Films and Imagery true baseline "VASNeg" scores as well as combined Films and Imagery vanilla baseline "VASNeg" scores were then examined using independent samples t-tests.

Emotional Reactivity: RSA, SCR, and Self-report/VAS

RSA and SCR: Emotional reactivity was operationalized as the difference between the mean RSA/SCR during the baseline prior to the emotion induction and the mean RSA/SCR during the emotion induction. As skin conductance data are frequently positively skewed, square-root transformations were employed as necessary (Venables & Christie, 1980). Mixed-model ANCOVA (with total DSS score as the covariate) was run separately for the each paradigm, with "Phase" (baseline or film/imagery) as the within-subjects factor and "Group" (BPD, SAD, or NC) as the between-subject factor.

Self-report/VAS: Emotional reactivity was operationalized as the difference between the VAS rating during the baseline prior to the emotion induction and the VAS rating during the emotion induction. For the Neutral tasks, emotional reactivity was operationalized as the difference between "VASNeg" during the baseline before the neutral task and "VASNeg" during the neutral task. The VAS data from the Films and Imagery procedures were analyzed using Mixed-Model ANOVA, with "Phase" (Baseline or Film/Imagery) as the within-subjects factor and "Group" (BPD, SAD, and NC) as the between-subjects factor.

Results

Self-Report Measures

A summary of the results for self-report measures are in Table 3. One SAD participant was missing her Difficulties in Emotion Regulation Scale (DERS) and State-Trait Anger Scale (STAXI) data and, therefore, was not included in the analysis. Omnibus One-way ANOVAs indicated significant between-group differences in the total DERS score as well as all six DERS subscales, the Acceptance and Action Questionnaire (AAQ), and the STAXI trait anger subscale. The BPD group scored higher than NC on all scales and subscales, and higher than SAD on all but the AAQ awareness subscale. A summary of the Dissociative State Scale (DSS) is in Table 4. As expected, the BPD group scored significantly higher on the DSS than the NC across all film and imagery inductions and significantly higher than the SAD group in the sad and anger imagery inductions. There were no significant DSS differences between the SAD and NC groups on any film or imagery inductions.

Biological Vulnerability: RSA True and Vanilla Baselines

A summary of the results for the baseline analyses are in Table 5. As predicted, the BPD group exhibited reduced RSA in the true and vanilla baselines compared with the NC group. Additionally, the BPD group demonstrated reduced RSA in the true and vanilla baselines compared with the SAD group. There were no significant differences in the true or vanilla baselines between the SAD and NC groups.

Baseline Negative Emotional Intensity: SCR and Self-report/VAS True and Vanilla Baselines

SCR—Results indicated that the BPD group exhibited a higher number of SCRs in the vanilla baseline and a trend toward a higher number of SCRs in the true baseline compared with the NC group. The BPD group did not exhibit significantly higher SCR compared with the SAD group in any of the baselines. The SAD group exhibited a higher number of SCRs in the true and vanilla baselines compared with the NC group.

VAS—As predicted, the BPD group reported significantly higher levels of negative emotion in the true and vanilla baselines compared with the NC group. There were no other significant differences.

Emotional Reactivity: RSA, SCR, and Self-report/VAS

RSA—Results of analysis of all Films and Imagery Group*Phase interactions are reported in Tables 6 and 7. Only one significant difference emerged (See Figure 1). There was a significant difference in slopes between the BPD and SAD group in the *sad* film (M=−.43, SE=.15), t(103.60)=−2.89, p<.01) such that the BPD group exhibited a significant increase in RSA from baseline to *sad* film (M=.25, SE=.11, t(103.18)=−2.37, p<.05), while SAD group exhibited a decrease in RSA, although this change did not reach statistical significance (M=−.18, SE=.11, t(104.05)=1.71, p>.05). All other Group*Phase interactions in the films and imagery tasks were non-significant.[1]

SCR—Similar to the RSA data, results indicated that there were two significant Group*Phase interactions in the *sad* film (See Figure 1). There was a significant difference in slopes between the BPD and NC group (M=−.41, SE=.19, t(93.68)=2.16, p<.05) such that the BPD group did not exhibit a significant change from Baseline to *sad* film (M=−.12, SE=.13, t(93.62)=.94, p>.05) while the NC exhibited a significant increase from Baseline to *sad* film (M=.28, SE=.13, t(93.73)=−2.10, p<.05). Similarly, while the BPD group did not exhibit a significant change from Baseline to *sad* film (M=−.12, SE=.13, t(93.62)=.94, p>.05) the SAD group exhibited a significant increase from Baseline to *sad* film (M=.27, SE=.13), t(91.94)=2.01, p<.05. All other Group*Phase interactions in the films and imagery tasks were non-significant. [1]

VAS—There were no significant Group*Phase interactions any of the films or imagery tasks.

[1]When analyses were rerun without controlling for dissociative state, findings did not change (data available upon request)

DISCUSSION

Findings from this investigation offer partial support for Linehan's (1987) theory of emotion dysregulation in BPD. Three major findings emerged. First, BPD individuals exhibited lower basal RSA compared with the SAD and NC, indicating biological vulnerability to emotion dysregulation. Second, BPD individuals displayed high baseline negative emotional intensity, characterized by higher baseline electrodermal responding and self-reported negative emotions compared with the other two groups. Third, there were no indications of heightened reactivity in BPD on any index or in either emotion condition. Taken together, the baseline differences between the BPD and control groups coupled with the *lack* of differences in slope from baseline to emotional stress suggest that 1) BPD individuals are biologically vulnerable to emotion dysregulation and 2) emotion dysregulation in BPD is accounted for by high baseline emotional intensity and not by high reactivity. Indeed, these data seemingly conflict with clinical observations of emotional reactivity in BPD. While clinical observations of high emotional intensity in BPD are often assumed to reflect high reactivity, these findings suggest that individuals meeting criteria for BPD are, in fact, not more "reactive" than non-clinical and socially anxious individuals. Rather, the extreme intensity of negative emotions often seen in BPD appears to be accounted for by the high starting point likely associated with persistent difficulties in regulation of negative affect.

Biological Vulnerability

Consistent with our hypothesis, BPD participants consistently demonstrated low basal RSA compared with the NC group in both the true and vanilla baselines. Interestingly, this finding conflicts with the null findings reported by Austin et al. (2007) who reported no significant basal RSA differences between BPD and NC. Reasons for this may be due to differences in the BPD participants between the two studies. While the current study excluded participants with specific comorbid mental disorders (e.g., bipolar disorder, psychotic disorders, current substance dependence), Austin and colleagues excluded BPD participants with any additional mental disorders. The BPD sample in the current study was likely drawn from a much more complex and severe population.

Additionally, the BPD group exhibited lower RSA compared with the SAD group in both the true and vanilla baselines, and there were no RSA differences in either of the baselines between the SAD and NC group. The differences in RSA between the BPD and SAD group is particularly compelling in that it suggests that reduced vagal tone is not a mere marker of psychopathology. Rather, these findings suggest that compromised parasympathetic functioning may be reflective of dimensional differences in emotion regulation capabilities between BPD and other clinical groups.

Baseline Negative Emotional Intensity

The BPD group also demonstrated high baseline negative emotional intensity as indicated by higher baseline SCR and self-reported negative emotions compared with the NC but not the SAD group. This finding was more robust for the self-report than electrodermal data. While there were significant differences in self-reported negative emotion between the BPD and NC across both baselines, the BPD group exhibited higher SCR than the NC in the

vanilla baseline and a trend toward higher SCR in the true baseline. Inspection of the mean SCR values showed that the BPD group had a higher number of SCRs in the vanilla baseline than the true baseline, while the NC stayed relatively the same. Given that the vanilla baseline always followed the true baseline, it may be that the increased SCRs in the BPD group reflected increased distress from sitting for an extended period of time. Although the purpose of the vanilla baseline was to mitigate any such potential distress, it is possible that it was not effective in doing so. This may account for why the heightened baseline EDA reported in this study conflicts with the previous reports of reduced EDA across slide viewing (Herpertz et al., 1999, 2000). It is possible that the slides functioned as a distraction from any potential distress induced by sitting over an extensive period, thus reducing EDA.

Emotional Reactivity

Our hypothesis of high emotional reactivity in BPD was not supported. Across both emotion conditions, the BPD group did not demonstrate greater self-reported or physiological changes in slope compared with the control groups. Inspection of the raw data also discounts the possible influence of a ceiling or floor (for RSA) effect as self-reported negative emotion in the BPD group was less than 25 on a 0–100 scale at baseline, and baseline SCR and RSA values for the BPD group were within the average range of reported scores in the literature (Dawson, Schell, & Filion, 2007; Fukusaki, Kawakubo, &Yamamoto, 2000).

A further unexpected finding also emerged. In the *sad* film, the BPD group exhibited a significant *decrease* in physiological reactivity from baseline to film compared with the NC and SAD groups. These findings are particularly interesting in that they suggest that the BPD group was not only less reactive than the SAD and NC groups, but that they were more regulated and/or less aroused during the *sad* film, despite their self-reported increase in sadness. This was not the case for the SAD and NC groups, where increases in self-reported sadness were coupled with significant increases in electrodermal responding. A possible interpretation of the decoupling exhibited by the BPD group may be that, while this group did experience an increase in sadness during the *sad* film, they were employing implicit emotion regulation strategies to mitigate their emotional experiencing. Data from the emotion regulation literature suggest that within-subject changes in RSA may be reflective of emotion regulatory efforts, which may or may not correspond with increased positive emotional experiencing (Butler, Wilhelm, & Gross; Ingjaldsson et al., 2003). Therefore, the increase in RSA demonstrated by the BPD group may reflect engagement of emotion regulation strategies during the sad film.

Limitations and Future Directions

Some limitations are noted. First, controlling for medications, necessary due to possible dampening of physiological reactivity, limits the generalizability of our findings. In the present study, 80% of the BPD participants were not on medications and 20% were only taking SSRIs. In a large longitudinal study of BPD individuals, 69% reported medication consultations in the first year of the study (Bender et al., 2006). In a recent randomized-controlled study on DBT (Linehan et al., 2006), 30% of the BPD participants took more than just SSRI medication. Future larger-scale studies should include participants with a wider range of medication use and compare differences in emotional processes between various

medicated subgroups. Second, the study did not exclude BPD participants with comorbid diagnoses. Given the high comorbid rates of BPD with other disorders, this decision increased the external validity, yet simultaneously decreased the internal validity of the study. However, while acknowledging the trade-off of compromising internal validity in the service of increasing external validity, adding the SAD control group mitigated some of the threat to internal validity.

A third limitation is that the personally-relevant imagery condition was retrospective in nature. The participants knew how the script would unfold and this knowledge likely influenced their emotional responding. This is of particular concern for the fear script, where, in the actual event, being oblivious to the outcome is central to the experience of fear. Therefore, recalling the event retrospectively, where the outcome was already known, likely mitigated the emotional responding. Nonetheless, the mere retrospective recall of an emotional event may not accurately reflect the actual intensity of emotional response during the time of the event.

The findings from this investigation, while informative, raise additional questions that will be important to address in future investigations. First, the relationship between emotional vulnerability and baseline emotional intensity warrants theoretical and empirical attention. Given that the current investigation found evidence of emotional vulnerability and high baseline intensity in BPD, a question that follows is how these two constructs are related. Although Linehan's (1987) theory proposes that high emotional intensity is an outcome of vulnerability, direct empirical investigation of the conceptual and temporal relationship between these two constructs is needed. For instance, is intensity indeed an independent vulnerability factor? Does vulnerability lead to high baseline intensity (as proposed by Linehan's theory), or could it be that individuals experiencing high baseline intensity are more emotionally vulnerable? Addressing these questions in future investigation is crucial to further the understanding the emotion dysregulation profile in BPD.

Some future directions are proposed. As indicated in the Method section, participants were not asked to report what strategies they might have implemented during the induction. Although dissociation was assessed, the possibility exists that participants engaged in other emotion regulation strategies during the induction period. Therefore, investigators of emotion processes in BPD may want to consider assessing for regulation strategies after employing emotion inductions. However, although asking participants to report any strategies they might have used would allow for a better understanding of self-report and physiological patterns, doing so could potentially prompt participants to engage in various emotion regulation strategies that they otherwise would not have known to implement. If the purpose of the study is to investigate natural emotional processes, then this would pose a potential confound. Therefore, the decision whether to assess for emotion regulation strategies needs to be carefully weighed and considered in the context of the primary research question.

In conclusion, these findings have significant implications for the etiological and treatment-relevant models of BPD. As reviewed earlier, Linehan's (1993) theory encompasses a biosocial, developmental model of BPD which proposes that BPD is the result of a

transactional relationship between biological vulnerability to emotion dysregulation and an invalidating environment. It appears, therefore, that BPD individuals may not be more reactive to the invalidating environment but, rather, start off with a higher level of emotional intensity. Further, while this study provides evidence for biological vulnerability in BPD and substantial literature report high rates of childhood sexual abuse (see Lieb, et al., 2004; Murray, 1993 for review) in this group (i.e. the epitome of an invalidating environment), the transactional relationship between these two factors on the subsequent development of BPD has not been directly examined. As such, it remains unclear whether the baseline intensity observed in this study is indeed a sequelae of vulnerability (as proposed by Linehan's model) or, if such intensity is the outcome of the chronic transaction between an emotionally vulnerable individual and an invalidating environment. Prospective, longitudinal studies are important to more rigorously substantiate Linehan's etiological model of BPD. In regards to clinical intervention, findings from this study offer specificity in the targeting of emotional reactions in BPD. While emotional reactivity is often a primary treatment target in BPD, these findings suggest that skills that target emotional vulnerability and baseline intensity may be more useful.

References

Austin M, Riniolo T, Porges. Borderline personality disorder and emotion regulation: Insights from the polyvagal theory. Brain and Cognition. 2007; 65:69–76. [PubMed: 17659821]

Barlow D, Allen L, Choate L. Towards a unified treatment for emotional disorders. Behavior Therapy. 2004; 35:205–230.

Battaglia M, Ferini Strambi L, Bertella S, Bajo S, Bellodi L. First-Cycle REM density in never-depressed subjects with borderline personality disorder. Society of Biological Psychiatry. 1999; 45:1056–1058.

Battaglia M, Ferini-Strambi L, Smirne S, Bernardeschi L, Bellodi L. Ambulatory polysomnography of never-depressed borderline subjects: A high-risk approach to rapid eye movement latency. Biological Psychiatry. 1993; 33:326–334. [PubMed: 8471690]

Beauchaine T. Vagal tone, development, and Gray's motivational theory: toward an integrated model of autonomic nervous system functioning in psychopathology. Development and Psychopathology. 2001:183–214. [PubMed: 11393643]

Bender D, Skodol A, Pagano M, Dyck I, Grilo C, Shea M, Sanislow C, Zanarini M, Yen S, McGlashan T, Gunderson J. Prospective assessment of treatment use by patients with personality disorders. Psychiatric Services. 2006; 57:254–257. [PubMed: 16452705]

Berntson G, Bigger J, Eckberg D, Grossman P, Kaufmann P, Malik M, et al. Heart rate variability: origins, methods, and interpretive caveats. Psychophysiology. 1997; 34:623–648. [PubMed: 9401419]

Berntson G, Cacioppo J, Quigley K. Autonomic determinism: The modes of autonomic control, the doctrine of autonomic space, and the laws of autonomic constraint. Psychological Review. 1991; 98:459–487. [PubMed: 1660159]

Bernston G, Quigley K, Jang J, Boysen S. An approach to artifact identification: Application to heart period data. Psychophysiology. 1990; 27(5):586–598. [PubMed: 2274622]

Boutros N, Torello M, McGlashan T. Electrophysiological aberrations in borderline personality disorder: State of the evidence. Journal of Neuropsychiatry and Clinical Neurosciences. 2003; 15(2):145–154. [PubMed: 12724454]

Butler E, Wilhelm F, Gross J. Respiratory sinus arrhythmia, emotion, and emotion regulation during social interaction. Psychophysiology. 2006; 43:612–622. [PubMed: 17076818]

Cheavens J, Rosenthal M, Daughters S, Novak J, Kosson D, Lynch T, et al. An analogue investigation of the relationships among perceived parental criticism, negative affect, and borderline personality

disorder features: The role of thought suppression. Behaviour Research and Therapy. 2005; 43:257–268. [PubMed: 15629754]

Clarkin J, Widiger T, Frances A, Hurt S, Gilmore M. Prototypic typology and the borderline personality disorder. Journal of Abnormal Psychology. 1983; 92:263–275. [PubMed: 6619404]

Cohen H, Benjamin J, Geva A, Matar M, Kaplan Z, Kotler M. Autonomic dysregulation in panic disorder and in post-traumatic stress disorder: application of power spectrum analysis of heart rate variability at rest and in response to recollection of trauma or panic attacks. Psychiatry Research. 2000; 96:1–13. [PubMed: 10980322]

Cohen H, Kotler M, Matar M, Kaplan Z, Loewenthal U, Miodownik H, et al. Analysis of heart rate variability in posttraumatic stress disorder patients in response to a trauma-related reminder. Society of Biological Psychiatry. 1998; 44:1054–1059.

Dawson, M.; Schell, A.; Filion, D. The Electrodermal System. In: Cacioppo, J.; Tassinary, L.; Berntson, G., editors. Handbook of Psychophysiology. 3rd Edition. New York, NY: Cambridge University Press; 2007. p. 159-181.

Donzella B, Gunnar M, Krueger W, Alwin J. Cortisol and vagal tone responses to competitive challenge in preschoolers: associations with temperament. Developmental Psychobiology. 2000; 37:209–220. [PubMed: 11084602]

Driessen M, Hermann J, Stahl K, Zwann M, Meier S, Hill A, Osterheider M, Petersen D. Magnetic resonance imaging-based measurement of the hippocampus and the amygdala in women with borderline personality disorder and early traumatization. Archives of General Psychiatry. 2000; 57:1115–1122. [PubMed: 11115325]

Ebner-Priemer U, Badeck S, Beckmann C, Wagner A, Feige B, Weiss I, et al. Affective dysregulation and dissociative experience in female patients with borderline personality disorder: a startle response study. Journal of Psychiatric Research. 2005; 39:85–92. [PubMed: 15504426]

Ebner-Priemer UW, Shaw-Welch S, Grossman P, Reisch T, Linehan MM, Bohus M. Psychophysiological ambulatory assessment of affective dysregulation in borderline personality disorder. Psychiatry Research. 2007; 150(3):265–275. [PubMed: 17321599]

First, M.; Spitzer, R.; Gibbon, M.; Williams, J. Structured Clinical Interview for Axis I DSM-IV Disorders - Patient Edition (SCID-I/P). New York: Biometrics Research Department, NY State Psychiatric Institute; 1995.

First, M.; Spitzer, R.; Gibbons, M.; Williams, J.; Benjamin, L. User's guide for the Structured Clinical Interview for DSM-IV Axis II Personality Disorders (SCID-II). New York: Biometrics Research Department, New York State Psychiatric Institute; 1996.

Fischer, A. Gender and Emotion: Social psychological perspectives. New York NY, US: Cambridge University Press; 2000.

Fowles D, Christie M, Edelberg R, Grings W, Lykken D, Venables P. Publication recommendations for electrodermal measurements. Psychophysiology. 1981; 18:232–239. [PubMed: 7291438]

Frances A, Fyer M, Clarkin J. Personality and suicide. Annals of the New York Academy of Sciences. 1986; 487:281–293. [PubMed: 2882718]

Friedman B, Thayer J. Autonomic balance revisited: panic anxiety and heart rate variability. Journal of Psychosomatic Research. 1998; 44:133–151. [PubMed: 9483470]

Fyan S, Goldberger A, Pincus S, Mietus J, Lipsitz L, Goldberger A. Gender- and age-related differences in heart rate dynamics: are women more complex than men? Journal of the American College of Cardiology. 1994; 24:1700–1707. [PubMed: 7963118]

Fukusaki C, Kawakubo K, Yamamoto Y. Assessment of the primary effect of aging on heart rate variability in humans. Clinical Autonomic Research. 2000; 10:123–130. [PubMed: 10954070]

Geller J. In again, out again: preliminary evaluation of a state hospital's worst recidivists. Hospital Community Psychiatry. 1986; 37:386–390. [PubMed: 2870978]

Grant B, Chou S, Goldstein R, Huang B, Stinson F, Saha T, et al. Prevalence, correlates, disability, and comorbidity of DSM-IV borderline personality disorder: results from the Wave 2 National Epidemiologic Survey on Alcohol and Related Conditions. Journal of Clinical Psychiatry. 2008; 69(4):533–545. [PubMed: 18426259]

Gratz K, Roemer L. Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the Difficulties in Emotion Regulation Scale. Journal of Psychopathology and Behavioral Assessment. 2004; 26:41–54.

Gross J, Levenson R. Emotion elicitation using films. Cognition and Emotion. 1995; 9:87–108.

Gurvits I, Koenigsberg H, Siever L. Neurotransmitter dysfunction in patients with borderline personality disorder. The Psychiatric Clinics of North America. 2000; 23:27–40. [PubMed: 10729929]

Haines J, Williams C, Brain K, Wilson G. The psychophysiology of self-mutilation. Journal of Abnormal Psychology. 1995; 104:471–489. [PubMed: 7673571]

Hayes S, Strosahl K, Wilson K, Bissett R, Pistorello J, Toarmino D, et al. Measuring experiential avoidance: A preliminary test of a working model. The Psychological Record. 2004; 54:553–578.

Hegerl U, Stein M, Mulert C, Mergl R, Olbrich S, Dichgans E, Rujescu D, Pogarell O. EEG-vigilance differences between patients with borderline personality disorder, patients with obsessive-compulsive disorder and healthy controls. European Archives of Psychiatry and Clinical Neuroscience. 2007; 258(3):137–143. [PubMed: 17990048]

Henry C, Mitropoulou V, New A, Koenigsberg H, Silverman J, Siever L. Affective instability and impulsivity in borderline and bipolar II disorders: similarities and differences. Journal of Psychiatric Research. 2001; 35:307–312. [PubMed: 11684137]

Herpertz S, Kunert H, Schwenger U, Sass H. Affective responsiveness in borderline personality disorder: a psychophysiological approach. American Journal of Psychiatry. 1999; 156:1550–1556. [PubMed: 10518165]

Herpertz S, Schwenger U, Kunert H, Lukas G, Gretzer U, Nutzmann J, Schuerkens A, Sass H. Emotional responses in patients with borderline as compared with avoidant personality disorder. Journal of Personality Disorders. 2000; 14:339–351. [PubMed: 11204341]

Herpertz S, Werth U, Lucas G, Qunaibi M, Schuerkens A, Kunert H, et al. Emotion in criminal offenders with psychopathy and borderline personality disorders. Archives of General Psychiatry. 2001a; 58:737–745. [PubMed: 11483139]

Ingjaldsson J, Laberg J, Thayer J. Reduced heart rate variability in chronic alcohol abuse: Relationship with negative mood, chronic thought suppression, and compulsive drinking. Society of Biological Psychiatry. 2003; 54:1427–1436.

Jennings J, Kamarck T, Stewart C, Eddy M, Johnson P. Alternate cardiovascular baseline assessment techniques: vanilla or resting baseline. Psychophysiology. 1992; 29:742–750. [PubMed: 1461961]

Koenigsberg H, Harvey P, Mitropoulou V, Schmeidler J, New A, Goodman M, et al. Characterizing affective instability in borderline personality disorder. American Journal of Psychiatry. 2002; 159:784–788. [PubMed: 11986132]

Koenigsberg H, Kaplan R, Gilmore M, Cooper A. The relationship between syndrome and personality disorder in DSM-III: experience with 2,464 patients. American Journal of Psychiatry. 1985; 142(2):207–212. [PubMed: 3970245]

Levine D, Marziali E, Hood J. Emotion processing in borderline personality disorders. Journal of Nervous and Mental Disease. 1997; 185:240–246. [PubMed: 9114809]

Lieb, K.; Zanarini, MC.; Linehan, MM.; Bohus, M. Seminar Section: Borderline Personality Disorder. London: The Lancet; 2004.

Linehan M. Dialectical behavior therapy for borderline personality disorder: Theory and method. Bulletin of the Menninger Clinic. 1987; 51:261–276. [PubMed: 3580661]

Linehan, M. Demographic Data Schedule (DDS). University of Washington, Seattle, WA: 1982. Unpublished manuscript

Linehan, M. Cognitive-behavioral treatment of borderline personality disorder. New York, NY: Guilford Press; 1993.

Linehan M, Comtois K, Murray A, Brown M, Gallop R, Heard H, et al. Two-year randomized controlled trial and follow-up of dialectical behavior therapy vs. therapy by experts for suicidal behaviors and borderline personality disorder. Archives of General Psychiatry. 2006; 63:757–766. [PubMed: 16818865]

Littell, R.; Milliken, G.; Stroup, W.; Wofinger, R. SAS System for Mixed Models. Cary, NC: SAS Institute Inc.; 1996.

Lyonfields J, Borkovec T, Thayer J. Vagal tone in generalized anxiety disorder and the effects of aversive imagery and worrisome thinking, Behavior Therapy. 1995; 26:457–466.

McGlashan T, Grilo C, Sanislow C, Ralevski E, Morey L, Gunderson J, et al. Two-Year Prevalence and Stability of Individual DSM-IV Criteria for Schizotypal, Borderline, Avoidant, and Obsessive-Compulsive Personality Disorders: Toward a Hybrid Model of Axis II Disorders. American Journal of Psychiatry. 2005; 162(5):883–889. [PubMed: 15863789]

Murray J. Relationship of childhood sexual abuse to borderline personality disorder, posttraumatic stress disorder, and multiple personality disorder. The Journal of Psychology. 1993; 127(6):657–676. [PubMed: 8301618]

Ni X, Chan K, Bulgin N, Sicard T, Bismil R, McMain S, Kennedy J. Association between serotonin transporter gene and borderline personality disorder. Journal of Psychiatric Research. 2006; 40:448–453. [PubMed: 16690085]

Nutt D, Argyropoulos S, Hood S, Potokar J. Generalized anxiety disorder: A comorbid disease. European Neuropsychopharmacology. 2006; 16:5109–5118.

Paris J, Brown R, Nowlis D. Long-term follow-up of borderline patients in a general hospital. Comprehensive Psychiatry. 1987; 28:530–535. [PubMed: 3691077]

Pitman R, Orr S, Forgue D, de Jong J, Claiborn J. Pschophysiologic Assessment of Posttraumatic Stress Disorder Imagery in Vietnam Combat Veterans. Archives of General Psychiatry. 1987; 44:970–975. [PubMed: 3675137]

Porges S. Cardiac vagal tone: A physiological index of stress. Neuroscience and Biobehavioral Reviews. 1995a; 19:225–233. [PubMed: 7630578]

Porges S. Orienting in a defensive world: Mammalian modifications of our evolutionary heritage. A Polyvagal Theory. Psychophysiology. 1995b; 32:301–318. [PubMed: 7652107]

Porges S, Doussard-Roosevelt J, Maiti A. Vagal tone and the physiological regulation of emotion. Monograms of the Society for Research in Child Development. 1994; 59:167–186.

Porges S, Doussard-Roosevelt J, Portales A, Greenspan S. Infant regulation of the vagal "brake" predicts child behavior problems: a psychobiological model of social behavior. Developmental Psychobiology. 1996; 29:697–712. [PubMed: 8958482]

Rechlin T, Weis M, Spitzer A, Kaschka W. Are affective disorders associated with alterations of heart rate variability? Journal of Affective Disorders. 1994; 32:271–275. [PubMed: 7897091]

Rosenthal M, Cheavens J, Lejuez C, Lynch T. Thought suppression mediates the relationship between negative affect and borderline personality disorder symptoms. Behaviour Research and Therapy. 2005; 43:1173–1185. [PubMed: 16005704]

Rosenthal M, Gratz K, Kosson D, Cheavens J, Lejuez C, Lynch T. Borderline personality disorder and emotional responding: A review of the research literature. Clinical Psychology Review. 2008; 28:75–91. [PubMed: 17544188]

Russell J, Moskowitz D, Zuroff D, Sookman D, Paris J. Stability and variability of affective experience and interpersonal behavior in borderline personality disorder. Journal of Abnormal Psychology. 2007; 116(3):578–588. [PubMed: 17696713]

Sack M, Hopper J, Lamprecht F. Low respiratory sinus arrhythmia and prolonged psychophysiological arousal in postrauamatic stress disorder: heart rate dynamics and individual differences in arousal regulation. Biological Psychiatry. 2004; 1:284–290. [PubMed: 14744470]

Samuels J, Eaten W, Bienvenu J, Clayton P, Brown H, Costa P, et al. Prevalence and correlates of personality disorder in a community sample. British Journal of Psychiatry. 2002; 180:536–542. [PubMed: 12042233]

Schmahl C, Bremner D. Neuroimaging in borderline personality disorder. Journal of Psychiatric Research. 2006; 40:419–427. [PubMed: 16239012]

Schmahl C, Elzinga B, Ebner U, Simms T, Sanislow C, Vermetten E, et al. Psychophysiological reactivity to traumatic and abandonment scripts in borderline personality and posttraumatic stress disorders: A preliminary report. Psychiatry Research. 2004b; 126:33–42. [PubMed: 15081625]

Schmahl C, Vermetten E, Elzinga B, Bremner J. Magnetic resonance imaging of hippocampal and amygdala volume in women with childhood abuse and borderline personality disorder. Psychiatry Research: Neuroimaging. 2003a; 122:109–115.

Skodol A, Siever L, Livesley W, Gunderson J, Pfohl B, Widiger T. The borderline diagnosis II: biology, genetics, and clinical course. Biological Psychiatry. 2002; 51:951–963. [PubMed: 12062878]

Soloff P, Lynch K, Kelly T. Childhood abuse as a risk factor for suicidal behavior in borderline personality disorder. Journal of Personality Disorders. 2002; 16:201–214. [PubMed: 12136678]

Soloff P, Lynch K, Kelly T, Malone K, Mann J. Characteristics of suicide attempts of patients with major depressive episode and borderline personality disorder: A comparative study. American Journal of Psychiatry. 2000; 157:601–608. [PubMed: 10739420]

Spielberger, C.; Krasner, S.; Solomon, E. The experience, expression and control of anger. In: Janisse, MP., editor. Health Psychology: Individual Differences and Stress. NY: Springer Verlag Publishers; 1988. p. 89-108.

Stein KF. Affect instability in adults with a borderline personality disorder. Archives of Psychiatric Nursing. 1996; 10:32–40. [PubMed: 8904033]

Stiglmayr C, Grathwol T, Linehan M, Ihorst G, Fahrenberg J, Bohus M. Aversive tension in patients with borderline personality disorder: a computer-based controlled field study. Acta Psychiatrica Scandinavica. 2005; 111:372–379. [PubMed: 15819731]

Stiglmayr C, Shapiro D, Stieglitz R, Limberger M, Bohus M. Experience of aversive tension and dissociation in female patients with borderline personality disorder -- a controlled study. Journal of Psychiatry Research. 2001; 35:111–118.

Swartz M, Blazer D, George L, Winfield I. Estimating the prevalence of borderline personality disorder in the community. Journal of Personality Disorders. 1990; 4:257–272.

Tebartz van Elst L, Hesslinger B, Thiel T, Geiger T, Haegele K, Lemieux L, Lieb K, Bohus M, Hennig J, Ebert D. Frontolimbic brain abnormalities in patients with borderline personality disorder. A volumetric MRI study. Biological Psychiatry. 2003; 54:163–171. [PubMed: 12873806]

Thayer J, Friedman B, Borkovec T. Autonomic characteristics of generalized anxiety disorder and worry. Biological Psychiatry. 1996; 39:255–266. [PubMed: 8645772]

Torgersen S, Kringlen E, Cramer V. The prevalence of personality disorders in a community sample. Archives of General Psychiatry. 2001; 58:590–596. [PubMed: 11386989]

Venables, P.; Christie, M. Electrodermal activity. Martin, I.; Venables, PH., editors. Chichester, UK: Wiley; 1980. p. 3-67.Techniques in Psychophysiology

Widiger T, Weissman M. Epidemiology of borderline personality disorder. Hospital & Community Psychiatry. 1991; 42(10):1015–1021. [PubMed: 1959892]

Williams L, Sidis A, Gordon E, Meares R. “Missing links” in borderline personality disorder: loss of neural synchrony relates to lack of emotion regulation and impulse control. Journal of Psychiatry & Neuroscience. 2006; 31(3):181–188. [PubMed: 16699604]

Yen S, Zlotnick C, Costello E. Affect regulation in women with borderline personality disorder traits. Journal of Nervous and Mental Diseases. 2002; 190:693–696.

Yeragani V, Balon R, Pohl R, et al. Decreased R-R variance in panic disorder patients. Acta Psychiatrica Scandinavica. 1990:554–559. [PubMed: 2378249]

Zanarini M, Frankenburg F, Dubo E, Sickel A, Tikha A, Levin A, Reynolds V. Axis I comorbidity of borderline personality disorder. The American Journal of Psychiatry. 1998; 155(12):1733–1739. [PubMed: 9842784]

Zanarini M, Frankenberg F, Hennen J, Silk K. The longitudinal course of borderline psychopathology: 6-year prospective follow-up of the phenomenology of borderline personality disorder. American Journal of Psychiatry. 2003; 160:274–283. [PubMed: 12562573]

Zanarini M, Williams A, Lewis R, Reich R, Vera S, Marino M, Levin A, Yong L, Frankenberg F. Reported pathological childhood experiences associated with the development of borderline personality disorder. American Journal of Psychiatry. 1997; 154:1101–1106. [PubMed: 9247396]

Zisook S, Goff A, Sledge P, Schuchter S. Reported suicidal behavior and current suicidal ideation in a psychiatric outpatient clinic. Annals of Clinical Psychiatry. 1994; 6:27–31. [PubMed: 7951642]

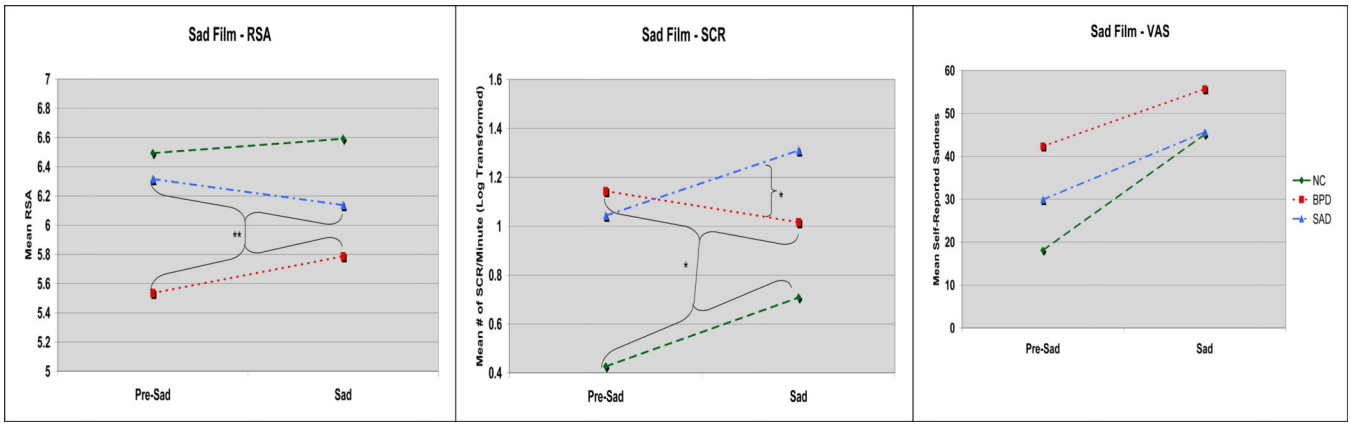

Figure 1.

Sad film group*phase interactions

Note. All significant differences and trends are in bold. BPD=borderline personality disorder, SAD=social anxiety disorder, NC=normal controls. RSA=respiratory sinus arrhythmia, SCR=skin conductance response, VAS=Visual Analogue Scale *p<.05*, p<.01**, p<.001***

Table 1

Participant Ethnic and Marital Demographics: Percent Breakdown

	BPD	SAD	NC
Caucasian	65.0	72.2	42.2
African-American	5.0	5.6	10.5
Asian-American	25.0	16.7	47.3
Other	5.0	5.5	0.0
Single	84.2	72.2	84.2
Married	10.5	22.2	10.5
Divorced	5.3	5.6	5.3

Note. BPD=borderline personality disorder, SAD=social anxiety disorder, NC=normal controls

Table 2

BPD and SAD Past and Current Axis I Diagnoses: Percent Breakdown

	BPD		SAD	
	Past	**Current**	**Past**	**Current**
Anorexia	10	0	0	0
Bulimia	10	5	5	0
Binge-eating	0	0	10	5
Depression	75	50	40	15
Dysthymia	N/A	5	N/A	0
Depressive NOS	5	0	0	0
Mood Disorder/Medic	5	0	5	0
Mood Disorder/Substance	0	0	0	0
Substance Dependence	50	10 (Abuse only)	30	5
PTSD	35	20	20	20
Generalized Anxiety Disorder	N/A	5	N/A	5
Obsessive Compulsive Disorder	15	10	15	5
Specific Phobia	10	10	10	5
Social Anxiety- General	30	30	100	100
Social Anxiety- Eating	5	5	0	0
Panic w/ Agoraphobia	0	0	0	0
Panic	25	15	5	0
Anxiety NOS	N/A	0	N/A	0

Note. BPD=borderline personality disorder, SAD=social anxiety disorder, NC=normal controls

Table 3

Mean Scores and Statistics on Self-report Emotion and Emotion Regulation Measures

	BPD	SAD[a]	NC	Omnibus test	BPD vs. NC	BPD vs. SAD	SAD vs. NC
	M (SD)	**M (SD)**	**M (SD)**	**F,df**	**t,df**	**t,df**	**t,df**
DERS total	114.65(20.62)	87.79(19.36)	71.00(13.19)	F=29.93 df=2,56***	t=7.67 df=56***	t=4.66 df=56***	t=2.91 df=56*
Nonacceptance	18.7(4.74)	15.53(6.08)	11.00(4.01)	F=11.88 df=2,56***	t=4.84 df=56***	t=1.98 df=56*	t=2.81 df=56*
Goal-directed	18.60(4.20)	15.05(4.70)	12.80(3.74)	F=9.60 df=2,56***	t=4.36 df=56***	t=1.89 df=56*	t=1.67 df=56
Impulse control	18.10(5.82)	10.05(3.54)	9.65(2.90)	F=24.54 df=2,56***	t=6.21 df=56***	t=5.87 df=56***	t=0.29 df=56
ER strategies	25.85(6.98)	17.89(5.65)	13.25(3.94)	F=25.73 df=2,56***	t=7.08 df=56***	t=4.42 df=56***	t=2.58 df=56*
Awareness	17.00(4.70)	16.63(5.13)	13.45(3.79)	F=3.69 df=2,56*	t=2.47 df=56*	t=0.25 df=56	t=2.18 df=56
Clarity	16.40(3.52)	12.55(3.55)	10.85(3.30)	F=13.55 df=2,57***	t=5.09 df=57***	t=3.49 df=57**	t=1.56 df=57
AAQ total	4.69 (.64)	4.07 (.62)	3.49 (.53)	F=19.75 df=2,57***	t=6.28 df=57***	t=3.20 df=57***	t=3.08 df=57**
STAXI- trait	24.80(5.28)	15.55(3.59)	17.63(1.16)	F=21.33 df=2,56***	t=6.23 df=56***	t=4.77 df=56***	t=1.38 df=56

Note. BPD=borderline personality disorder, SAD=social anxiety disorder, NC=normal controls. DERS=Difficulties in Emotion Regulation Scale, AAQ=Acceptance and Action Questionnaire, STAXI-trait=State-trait Anger Inventory. Higher scores indicate more difficulty in emotion regulation, experiential avoidance, and trait anger

[a]One SAD subject was missing her DERS and STAXI data and therefore, was excluded from the analysis.

p<.05,

**p<.01,

***p<.001 p-values are bonferroni-corrected.

Table 4

Mean Dissociation Scores and Statistics For Each Film and Imagery Task

	BPD	SAD	NC	Omnibus Test	BPD vs. NC	BPD vs. SAD	SAD vs. NC
	M (SD)	M (SD)	M (SD)	F, df	t,df	t,df	t,df
Sad Film	1.25 (1.63)	.36 (.43)	.16 (.28)	F=6.88 df=2,56**	t=2.87 df=56*	t=2.31 df=56	t=1.69 df=56
Anger Film	1.18 (1.52)	.45 (.69)	.15 (.22)	F=6.59 df=2,57**	t=3.20 df=57*	t=1.98 df=57	t=1.84 df=57
Fear Film	1.25 (1.63)	.36 (.43)	.16 (.28)	F=6.88 df=2,56**	t=2.87 df=56*	t=2.31 df=56	t=1.69 df=56
Neutral Film	1.21 (1.60)	.36 (.43)	.16 (.28)	F=6.59 df=2,57**	t=2.91df=57*	t=2.30 df=57	t=1.69 df=57
Sad Imagery	1.29 (1.48)	.37 (.51)	.20 (.30)	F=6.87 df=2,56**	t=3.16 df=56*	t=2.58 df=56*	t=1.29 df=56
Anger Imagery	1.81 (1.87)	.53 (.72)	.33 (.44)	F=9.16 df=2,56***	t=3.37 df=56**	t=2.79 df=56*	t=1.10 df=56
Fear Imagery	1.70 (1.78)	.66 (.85)	.38 (.53)	F=6.87 df=2,56**	t=3.10 df=56*	t=2.30 df=56	t=1.27 df=56
Neutral Imagery	1.70 (1.78)	.66 (.85)	.38 (.53)	F=6.87 df=2,56**	t=3.10 df=56*	t=2.30 df=56	t=1.27 df=56

Note. BPD=borderline personality disorder, SAD=social anxiety disorder, NC=normal controls. DSS=Dissociative State Scale

* p<.05*,

** p<.01,

*** p<.001 p-values are Games-Howell-corrected

J Abnorm Psychol. Author manuscript; available in PMC 2014 December 26.

Table 5

Baseline Film and Imagery Means and Statistics: RSA, SCR, and Self-report

	M(SE)	M(SE)	M(SE)	t, df, Cohen's d	t, df, Cohen's d	t, df, Cohen's d
[a]RSA (ms^2)	**BPD[d]**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
True Baseline	5.47(.24)	6.14(.24)	6.72(.24)	**t=−3.72 df=57** d=1.21**	**t=−1.99 df=57* d=.66**	t=1.73 df=57 d=.56
Vanilla Baseline	5.37(.23)	6.14(.23)	6.59(.23)	**t=−3.84 df=57*** d=1.23**	**t=−2.43 df=57* d=.78**	t=−1.41 df=57 d=.46
[ab]SCR	**BPD**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
True Baseline	1.69(.44)	2.17(.45)	.79(.45)	**t=1.89 df=54.38 p=.07** d=.47	t=−1.01 df=54.38 d=.25	**t=2.86 df=55** d=.72**
Vanilla Baseline	2.78(.37)	2.32(.38)	1.00(.38)	**t=3.40 df=53.92** d=1.10**	t=.87 df=53.97 d=.29	**t=2.50 df=53.80* d=.82**
[c]VAS	**BPD**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
True Baseline	4.77 (.51)	4.01(.46)	3.41(.31)	**t=2.27 df=38* d=.74**	t=1.11 df=38 d=.36	t=1.07 df=38 d=.35
Vanilla Baseline	4.69(.51)	3.81(.41)	2.78 (.34)	**t=3.15 df=38** d=1.01**	t=1.36 df=38 d=.44	t=1.96 df=38 d=.63

Note. All significant differences and trends are in bold. BPD=borderline personality disorder, SAD=social anxiety disorder, NC=normal controls. RSA=respiratory sinus arrhythmia, SCR=skin conductance response, VAS=Visual Analogue Scale

[a]Denominator dfs were derived from Sattherwaite approximation

[b]Values refer to number of non-specific fluctuations per minute

[c]Values are square root-transformed

*p<.05*,

**p<.01,

***p<.001

Table 6

Mean Changes and Statistics from Baseline to Film: RSA, SCR, and Self-report

	Phase Δ M(SE)	Phase Δ M(SE)	Phase Δ M(SE)	t, df, Cohen's d	t, df, Cohen's d	t, df, Cohen's d
[a]RSA (ms^2)	**BPD**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
Sad Film	.25(.11)	−.18(.11)	.10(.10)	t=−1.03 df=104.70 d=.33	**t=−2.89** **df=103.60** **d=−.75**	t=1.88 df=105.17 d=.61
Fear Film	−.07(.13)	.03(.13)	.14(.13)	t=1.15 df=83.51 d=.37	t=.52 df=85.14 d=.18	t=.61 df=86.35 d=.20
Anger Film	.14(.12)	.15(.12)	−.02(.12)	t=−1.02 df=78.66 d=.30	t=.04 df=78.14 d=.02	t=−1.06 df=78.46 d=.32
Neutral Film	−.04(.12)	.08(.12)	−.15(.12)	F=.89 df=2,134.39 d=.21	F=.89 df=2,134.39 d=.23	F=.89 df=2,134.39 d=.44
***[ab]*SCR**	**BPD**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
Sad Film	−.12(.13)	.27(.13)	.28(.13)	**t=2.16** **f=93.68** d=.71	**t=2.09** **df=92.77** d=−.70	t=.07 df=92.83 d=.02
Fear Film	.08(.17)	.51(.18)	.43(.17)	t=1.45 df=98.05 d=.48	t=1.76 df=98.86 d=.57	t=−.31 df=99.92 d=.11
Anger Film	−.31(.15)	.000003(.15)	−.07(.15)	t=1.82 df=111.13 d=.37	t=1.49 df=110.10 d=.48	t=−.31 df=110.68 d=.11
Neutral Film	−.14(.12)	.23(.12)	−.11(.13)	F=2.39 df=2,166.87 d=.06	F=2.39 df=2,166.87 d=.72	F=2.39 df=2,166.87 d=.64
[c]VAS	**BPD**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
Sad Film	13.35(4.93)	15.70(4.93)	27(4.93)	t=−1.96 df=57 d=.64	t=−.34 df=57 d=.11	t=−1.62 df=57 d=.53
Fear Film	19.60(5.39)	17.85(5.39)	26.65(5.39)	t=−.93 df=57 d=.30	t=.23 df=57 d=.08	t=−1.16 df=57 d=.38

	Phase Δ M(SE)	**Phase Δ M(SE)**	**Phase Δ M(SE)**	**t, df, Cohen's d**	**t, df, Cohen's d**	**t, df, Cohen's d**
[a]RSA (ms^2)	**BPD**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
Anger Film	12.55(4.88)	14.00(4.88)	19.95(4.88)	t=−1.07 df=57 d=.35	t=−.21 df=57 d=.07	t=−.86 df=57 d=.28
Neutral Film	1.60(1.36)	−1.79(1.36)	.94(1.36)	F=.10 df=2,57 d=.11	F=.10 df=2,57 d=.57	F=.10 df=2,57 d=.46

Note. All significant differences and trends are in bold. Denominator df were derived from Sattherwaite Approximation. BPD=borderline personality disorder, SAD=social anxiety disorder, NC=normal controls. RSA=respiratory sinus arrhythmia, SCR=skin conductance response, VAS=Visual Analogue Scale

[a]Controlled for dissociative state

[b]Values are square-root transformed and refer to number of non-specific fluctuations per minute

[c]Neutral Film analyses were conducted by omnibus F-test as a priori hypotheses were not specified

p<.05,

**p<.01,

***p<.001

J Abnorm Psychol. Author manuscript; available in PMC 2014 December 26.

Table 7

Mean Changes and Statistics from Baseline to Imagery: RSA, SCR, and Self-report

	Phase Δ M(SE)	**Phase Δ M(SE)**	**Phase Δ M(SE)**	**t, df**	**t, df**	**t, df**
[a]RSA (ms^2)	**BPD**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
Sad Imagery	−.08(.13)	−.11(.17)	.0018(.12)	t=.46 df=695.69 d=.15	t=−.147 df=695.83 d=.05	t=.55 df=695.68 d=.17
Fear Imagery	−.06(.10)	−.28(.10)	−.13(.10)	t=−.49 df=686.18 d=.16	t=−1.56 df=686.16 d=.51	t=1.07 df=686.24 d=.34
Anger Imagery	−.22(.18)	−.40(.16)	−.35(.16)	t=−.62 df=24.60 d=.18	t=−.76 df=24.61 d=.25	t=.15 df=36.79 d=.07
Neutral Imagery	−.11(.13)	−.32(.13)	−.25(.13)	F=.67 df=2,106.42 d=.25	F=.67 df=2,106.42 d=.38	F=.67 df=2,106.42 d=.12
[ab]SCR	**BPD**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
Sad Imagery	.52(.17)	.68(.18)	−.26(.16)	t=−1.10 df=27.36 d=1.11	t=.64 df=16.88 d=.22	t=−1.70 df=16.43 d=1.30
Fear Imagery	.41(.11)	.35(.11)	.30(.11)	t=−.74 df=686.91 d=.24	t=−.37 df=686.83 d=.13	t=−.37 df=687.14 d=.11
Anger Imagery	.11(.19)	.50(.16)	.17(.16)	t=.24 df=241.84 d=.08	t=1.56 df=241.89 d=.52	t=−1.46 df=200.51 d=.48
Neutral Imagery	−.21(.13)	.11(.12)	.16(.12)	F=2.64 df=2,102.01 d=.70	F=2.64 df=2,102.01 d=.60	F=2.64 df=2,102.01 d=.10
[c]VAS	**BPD**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
Sad Imagery	24.95(4.55)	19.25(4.43)	23.40(4.43)	t=.24 df=56 d=.08	t=.90 df=56 d=.30	t=−.66 df=56 d=.22
Fear Imagery	17.68(4.86)	14.70(4.74)	14.35(4.74)	t=.49 df=56 d=.16	t=.44 df=56 d=.14	t=.05 df=56 d=.02
Anger Imagery	21.42(4.82)	17.85(4.70)	21.65(4.70)	t=−.03 df=56 d=.01	t=.53 df=56 d=.17	t=−.57 df=56 d=.19

	Phase Δ M(SE)	**Phase Δ M(SE)**	**Phase Δ M(SE)**	**t, df**	**t, df**	**t, df**
[a]RSA (ms^2)	**BPD**	**SAD**	**NC**	**BPD vs. NC**	**BPD vs. SAD**	**SAD vs. NC**
Neutral Imagery	−3.32(1.72)	−2.71(1.67)	−1.25(1.67)	F=.40 df=2,56 d=.28	F=.40 df=2,56 d=.08	F=.40 df=2,56 d=.20

Note. All significant differences and trends are in bold. Denominator df were derived from Sattherwaite Approximation. BPD=borderline personality disorder, SAD=social anxiety disorder, NC=normal controls. RSA=respiratory sinus arrhythmia, SCR=skin conductance response, VAS=Visual Analogue Scale

[a]Controlled for dissociative state

[b]Values are square-root transformed and refer to number of non-specific fluctuations per minute

[c]Neutral Film analyses were conducted by omnibus F-test as a priori hypotheses were not specified

p<.05,

**p<.01,

***p<.001