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Characterizing Emotional Dysfunction in Borderline Personality, Major Depression, and their Co-occurrence

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Abstract

This research aimed to characterize patterns of emotional reactivity and dysregulation in borderline personality, depression, and their co-occurrence. In Study 1, 488 young adult women from the community were categorized into four groups based on self-reported major depressive disorder (MDD) and borderline personality disorder (BPD) symptoms (Low BPD/Low MDD; Low BPD/High MDD; High BPD/Low MDD; High BPD/High MDD). Immediate and prolonged subjective emotional reactivity to a laboratory stressor were assessed, and participants completed self-report and behavioral measures of emotion dysregulation. Study 2 extended these findings, examining emotional reactivity and dysregulation in a clinical population of 176 substance dependent patients with diagnoses of BPD and MDD and including a biological index of emotional reactivity. Results revealed greater prolonged fear reactivity in the High BPD/High MDD (vs. Low BPD/Low MDD) group in Study 1, and greater prolonged anxiety and negative affect reactivity in both High BPD groups (vs. Low BPD/Low MDD and Low BPD/High MDD groups) in Study 2 (but no differences in cortisol reactivity). Results also demonstrated greater subjective (but not behavioral) emotion dysregulation in the High BPD/High MDD (vs. Low BPD/Low MDD) group in Study 1 and both High BPD groups (vs. both Low BPD groups) in Study 2. Finally, the High BPD/High MDD group reported greater difficulties controlling impulsive behaviors compared with all other groups in Study 1 and the Low BPD groups in Study 2. Findings suggest that BPD pathology (but not MDD pathology alone) is characterized by

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greater prolonged emotional (especially anxiety/fear-related) reactivity and heightened emotion dysregulation.

Keywords

borderline personality disorder; major depression; emotional reactivity; negative affect; emotion dysregulation; experimental assessment

1. Introduction

Although emotional dysfunction has been implicated in the development and maintenance of numerous forms of psychopathology [1], it is considered particularly central to borderline personality disorder (BPD) [2–7]. However, given that the presence of co-occurring disorders is the norm, rather than the exception, in BPD [8], identifying patterns of emotional dysfunction specific to BPD is a crucial next step. Among the most common disorders to co-occur with BPD is major depressive disorder (MDD; e.g., 71–87%) [8,9], which is itself associated with heightened emotional dysfunction [10,11]. Thus, the present laboratory-based studies extend existing research by examining two specific aspects of emotional dysfunction (i.e., emotional reactivity and emotion dysregulation) in participants with and without BPD and MDD pathology.

One domain of emotional dysfunction theorized to be particularly relevant to BPD is emotional reactivity [2,3,7], defined as the amplitude of emotional response (across subjective, physiological, or expressive domains) to internal or external stimuli [12]. Empirical research supports the relevance of emotional reactivity to BPD. Research examining self-reported trait emotional reactivity indicates a positive association between BPD pathology and emotional reactivity [13–15], as well as greater self-reported emotional reactivity among individuals with versus without BPD [16–18]. Participants with BPD also report more frequent shifts in negative affect (NA) and greater emotional reactivity to daily negative interactions than healthy controls in studies using ecological momentary assessment [19–22]. Moreover, laboratory-based studies provide further support for heightened subjective emotional reactivity in BPD (for exceptions, see references [23,24]), finding greater self-reported emotional reactivity in response to laboratory stressors among patients with (vs. without) BPD [25] and individuals high (vs. low) in BPD pathology [26]. Finally, although studies of biological emotional reactivity in BPD (as indexed by cortisol reactivity in response to stressors) have produced mixed results [27–30], recent research provides support for heightened cortisol reactivity in BPD, although only at low levels of posttraumatic stress disorder (PTSD) symptoms [29] or high levels of dissociation [30].

Another important domain of emotional dysfunction in BPD is emotion dysregulation. As defined here, emotion dysregulation refers to maladaptive responses to emotions, including: (a) a lack of awareness, understanding, and acceptance of emotions; (b) the inability to control behaviors when experiencing emotional distress; (c) lack of access to adaptive strategies for modulating the duration and/or intensity of aversive emotional experiences; and (d) an unwillingness to experience emotional distress as part of pursuing meaningful activities in life [31,32]. Extant research provides strong support for an association between

BPD and all of the dimensions of emotion dysregulation noted above (as assessed with the Difficulties in Emotion Regulation Scale [31]; e.g., [24,33–35]). Studies using other self-report measures have also found evidence for a relation between BPD and many dimensions of emotion dysregulation, including lower emotional clarity [36], greater nonacceptance and avoidance of emotions [37,38], and greater use of avoidant regulation strategies [33]. Finally, studies using behavioral and laboratory-based measures of emotion dysregulation indicate multiple forms of emotion dysregulation among individuals with BPD, including lower emotional awareness and clarity [39], greater unwillingness to experience distress in order to pursue goal-directed behavior [32,34], and greater difficulties controlling behaviors in the context of distress [40].

Emotional dysfunction is also a prominent feature of MDD pathology, although the precise nature of this dysfunction is unclear. For instance, whereas individuals with both current [10] and lifetime [41] MDD exhibit greater NA in their daily lives than those without MDD, research on emotional reactivity in MDD has produced mixed results, with some studies finding evidence of heightened emotional reactivity in the laboratory among individuals with current and remitted MDD pathology [42–44] and others suggesting that both remitted and currently depressed individuals display less emotional reactivity than control [45,46] (at least with regard to sadness [45]). Despite these inconsistent findings, however, results of a recent meta-analysis suggest that MDD pathology is associated with blunted subjective emotional reactivity to negative stimuli in the laboratory, $d = -.36$ [47]. Moreover, research on biological (i.e., cortisol) emotional reactivity in MDD has produced similar results, with studies suggesting that both MDD [48] and remitted MDD [46] are associated with blunted cortisol reactivity to stressors, compared with non-depressed controls (for exceptions, see references [49,50]). Notably, investigations of emotion dysregulation in MDD pathology have produced more consistent findings, with both current and remitted MDD pathology evidencing positive associations with emotion dysregulation [41,51–53].

Despite evidence to suggest that BPD and MDD frequently co-occur [8,9], little research has directly compared emotional dysfunction in BPD and MDD. Nonetheless, preliminary theoretical and empirical literature suggest that BPD may be characterized by greater emotional reactivity to acute stressors and heightened emotion dysregulation, relative to MDD [54,55]. For example, Goodman and colleagues [54] have theorized that, despite some phenotypic overlap between BPD and MDD, the nature of the emotional dysfunction in these disorders differs, with BPD characterized by episodic emotional reactivity and emotion dysregulation in response to this reactivity and MDD characterized by sustained mood problems. These researchers also suggest that BPD symptoms dominate the clinical presentation in BPD-MDD co-occurrence [54] – consistent with findings that the presence of co-occurring BPD in MDD is associated with greater depressive symptoms [56,57].

Consistent with this theoretical literature, results of a recent laboratory study revealed heightened subjective emotional reactivity in the form of anger reactivity (but not other emotions) following a shame-specific emotion induction among participants with BPD, compared to depressed and healthy controls [55]. Moreover, although no studies have examined differences in biological emotional reactivity in BPD versus MDD, research examining other aspects of biologically-indexed emotional dysfunction in BPD and MDD

suggests higher resting cortisol levels in BPD (but not MDD) versus controls [58], as well as hypo-suppression of cortisol in response to the dexamethasone suppression test in MDD but not BPD [59]. Finally, extant research provides initial support for distinct patterns of self-reported emotion dysregulation in BPD and MDD pathology. For instance, when controlling for MDD pathology, BPD pathology is uniquely associated with greater overall emotion dysregulation [60], as well as the specific emotion regulation difficulties of emotional avoidance [61], lack of access to effective emotion regulation strategies [60], and difficulties controlling impulsive behaviors when distressed [60]. Further, relative to patients with MDD, individuals with BPD report heightened levels of overall emotion dysregulation, as well as a greater reliance on maladaptive and avoidant emotion regulation strategies [62].

Taken together, results of the aforementioned studies provide preliminary support for heightened levels of certain aspects of emotional dysfunction among individuals with BPD versus MDD pathology. Nonetheless, important limitations exist. First, research has not examined the impact of co-occurring BPD and MDD pathology on emotional dysfunction; thus, it is unclear if this co-occurrence is associated with a different pattern of emotional dysfunction than BPD or MDD alone. Second, past research exploring distinct patterns of emotional dysfunction in BPD and MDD has relied on subjective assessments of emotional dysfunction; thus, differences in behaviorally- or biologically-assessed emotional dysfunction as a function of BPD and MDD pathology are unknown. Third, the one study to date that compared emotional reactivity in the laboratory among individuals with BPD versus MDD used a shame-specific emotion induction [55]. Given evidence that emotional reactivity in the laboratory is influenced by the type of stressor utilized [63], the generalizability of the results of that study to other stressors remains unclear. Moreover, although results of that study provide preliminary evidence of prolonged anger among individuals with BPD (but not MDD), the focus was on emotional responses before and after the shame induction and then subsequent recovery [55]. However, no research to date has examined the time course of emotional dysfunction in response to ongoing emotional stimuli among individuals with high and low levels of BPD and MDD pathology. This is a critical limitation of past research given theoretical and empirical literature highlighting the relevance of prolonged emotional responses (including reactivity) to BPD [25].

Extant literature highlights the need for more comprehensive and nuanced investigations of emotional reactivity and emotion dysregulation when characterizing patterns of emotional dysfunction in BPD, MDD, and their co-occurrence. Thus, the present investigation sought to examine unique patterns of emotional reactivity and emotion dysregulation in BPD pathology, MDD pathology, and their co-occurrence across both community and clinical samples. To this end, we utilized a multi-method, laboratory-based design to examine both immediate and prolonged subjective and biological emotional reactivity to a laboratory stressor, as well as both subjective and behaviorally-indexed emotion dysregulation. We hypothesized that, relative to MDD pathology, BPD pathology would be associated with heightened negative emotional reactivity to the laboratory stressor, both in general and across the specific emotions of fear/anxiety, anger, and shame. In addition, we hypothesized that BPD pathology (compared to MDD pathology) would be associated with greater levels of overall emotion dysregulation, as well as the specific dimension of emotion dysregulation involving difficulties controlling impulsive behaviors when distressed. Finally, given past

evidence of greater clinical severity in BPD-MDD co-occurrence, relative to either disorder alone [64], we expected that individuals with both BPD and MDD pathology would demonstrate greater emotional reactivity and emotion dysregulation than all other groups.

2. Study 1

2.1. Aims

The goal of this study was to examine patterns of emotional dysfunction associated with heightened levels of BPD and MDD pathology (and their co-occurrence) in a large sample of young adult women from the community. Levels of subjective emotional reactivity to a laboratory stressor, as well as subjective and behavioral emotion dysregulation, were examined among community women low in both BPD and MDD symptoms (Low BPD/Low MDD), low in BPD and high in MDD symptoms (Low BPD/High MDD), high in BPD and low in MDD symptoms (High BPD/Low MDD), and high in both BPD and MDD symptoms (High BPD/High MDD).

2.2. Material and Methods

2.2.1. Participants—The current data were drawn from a large, multi-site, prospective study of emotion dysregulation and sexual revictimization among young adult women in the community. This study includes a community sample of young adult women from four sites in the Southern and Midwestern United States (including Mississippi, Nebraska, and Ohio). Recruitment methods included random sampling from the community, in addition to community advertisements. The current study uses data from only the Wave 1 assessment.

Participants (N = 488) ranged in age from 18 to 25 years (M = 21.7, SD = 2.2) and were ethnically diverse (55.7% White; 33.8% African American/Black; 4.3% Asian/Pacific Islander; 0.6% Latina). With regard to highest level of educational attainment, 20.5% of participants had received their high school diploma or GED, and 75% had completed at least some higher education. Most participants (68.2%) reported a household income less than $30,000 and were single and never married (83.2%).

2.2.2. Self-report Measures—The *Borderline Evaluation of Severity Over Time* (BEST) [65] was used to assess BPD pathology. The BEST is a 15-item measure of BPD symptom severity and dysfunction over the past month (in the current sample, $\alpha = 0.85$). The BEST has good test-retest reliability and convergent and discriminant validity [66,67]. For the purposes of this study, and consistent with past research [68–72], participants were classified according to the severity of their BPD symptoms. Specifically, a score of ≥30 was used as the cutoff to indicate the presence of clinically-relevant levels of BPD pathology (with individuals above this cutoff classified as High BPD, and those below this cutoff classified as Low BPD; see references [37,73]). In support of the validity of this cutoff, a score of ≥30 on the BEST falls within a one-half SD of the mean BPD symptom severity of BPD outpatient samples (37.5 ± 12.0), and more than 1 SD above the mean for outpatients without a personality disorder (21.5 ± 7.8) [73]. Further, this cutoff is associated with a positive predictive power value of .93 and a negative predictive power value of .77 with respect to BPD diagnoses on the Diagnostic Interview for DSM-IV Personality Disorders

[74], suggesting that this is an appropriate method of delineating individuals for the High BPD group. Moreover, research using this cutoff within other community adult samples has provided support for its construct validity, finding higher rates of self-injury (one of the most common behaviors in BPD) among individuals classified as High versus Low BPD on this measure [68,69].

MDD pathology was assessed using the *Depression Anxiety Stress Scales-21* (DASS-21) [75,76], a 21-item self-report questionnaire designed to differentiate between core symptoms of depression, anxiety, and stress. Participants rate the extent to which each item applies to them on a 4-point Likert-type scale (0 = *never*, 3 = *almost always*). The DASS-21 has been found to demonstrate adequate test-retest reliability [77] and good construct and discriminant validity [75–77]. Internal consistency for the 7-item depression scale in this sample was good ($\alpha = 0.89$). Consistent with scoring guidelines [75,76], participants scoring ≥14 (indicating "moderate" symptoms of depression) on the depression scale were designated as High MDD whereas those scoring < 14 on the depression scale were designated as Low MDD.

Self-reported emotion dysregulation was assessed using the *Difficulties in Emotion Regulation Scale* (DERS [31], a 36-item self-report measure that assesses individuals' typical levels of emotion dysregulation across six domains: nonacceptance of negative emotions, difficulties engaging in goal-directed behaviors when distressed, difficulties controlling impulsive behaviors when distressed, limited access to emotion regulation strategies perceived as effective, lack of emotional awareness, and lack of emotional clarity. Participants rate the extent to which each item applies to them on a 5-point Likert-type scale (1=*almost never,* 5=*almost always)*. The DERS has demonstrated good test-retest reliability and adequate construct and predictive validity [31,78]. Further, the DERS and its subscales have been found to predict performance on objective measures of emotion regulation [32,79,80]. Items were recoded so that higher scores indicate greater emotion dysregulation. Internal consistency in the current sample was acceptable for the overall scale ($\alpha = 0.95$) and all subscales ($\alpha = 0.66–0.93$).

Finally, exposure to potentially traumatic events (PTE) was assessed using the *Life Events Checklist* (LEC; [81]). The LEC lists 16 PTE and instructs participants to indicate any event they have experienced personally. For the purposes of this study, the number of PTE participants reported experiencing was summed to provide an overall index of traumatic exposure and included in follow up analyses to control for the influence of traumatic exposure on the relations of interest.

2.2.3. Laboratory Measures—To assess both subjective emotional reactivity and the unwillingness to experience emotional distress (one component of emotion dysregulation as defined here; [31,32,35]), this study used a modified version of the Paced Auditory Serial Addition Task – Computerized (PASAT-C), an empirically-supported laboratory stressor shown to induce emotional distress in the form of anxiety, frustration, and irritability [82–84]. During this task, numbers are flashed sequentially on a computer screen, and participants are instructed to sum the most recent number with the previous number (using the computer mouse to click on the correct answer). After providing each sum, the

participant must ignore the sum and add the following number to the most recently presented number. When a correct answer is provided, a point is obtained. If an incorrect answer is provided, or if the participant fails to provide an answer before the next number is presented, an "explosion" sound is played and the score does not change.

The version of the PASAT-C used here consisted of four levels, the first three of which had increasingly shorter latencies between number presentations. Because the correct answer must be provided prior to the presentation of the next number in order to obtain a point, difficulty increases as latencies decrease. The first level (low difficulty) lasted 1 min and had a 3-s latency between number presentations; the second level (moderate difficulty) lasted 2 min and had a 2-s latency; and the third level (high difficulty) lasted 1 min and had a 1-s latency. As such, the third level is designed to make it virtually impossible for participants to provide a correct answer prior to the presentation of the next number (thereby inducing distress). Following a brief 1 min rest period to complete the negative emotion ratings (see below), the final level began. The final level had the same latency between number presentations as the third level (i.e., 1-s), but lasted seven minutes and included an option to terminate the task. Latency in seconds to task termination is used as a measure of the emotion regulation dimension of the willingness to experience emotional distress in order to pursue goal-directed behavior [32,35,79].

In support of its construct validity, this task has been shown to induce emotional distress in the form of anxiety, anger, frustration, and irritability among clinical and nonclinical samples [25,34,84]. Further, latency to termination scores on this task are significantly correlated with other behavioral measures of the willingness to experience distress [34,79], as well as self-report measures of emotion dysregulation, emotional avoidance, and emotional nonacceptance [32,79,85]. Finally, providing evidence that latency to termination scores on this task are not simply a measure of skill level or distress in response to the task, neither NA in response to the task nor task performance has been found to be significantly associated with latency to task termination [34,79].

To assess emotional reactivity to the PASAT-C, participants completed the NA scale of the Positive and Negative Affect Schedule (PANAS-NA; [86]) before the PASAT-C (post-stressor), following completion of the third (most difficult) level of the task (peak-stressor), and after the task (post-stressor). Participants rated the extent to which they were currently experiencing 10 forms of NA on a scale from 1 (*very slightly or not at all*) to 5 (*extremely*). In addition to summing responses to all 10 items to create a total NA scale, composites for specific emotions were calculated (as identified by Watson and colleagues [86]). Specifically, we examined *general distress* (derived from "distressed," "upset"), *shame/guilt* (derived from "ashamed," "guilty"), *anger* (derived from "hostile," "irritable"), *anxiety* (derived from "jittery," "nervous"), and *fear* (derived from "scared," "afraid"). Internal consistency was adequate for the overall NA scale (αs = 0.81–0.87), as well as each composite (*r*s = .38–.72, *p*s < .001).

2.2.4. Procedure—All methods received approval by the Institutional Review Boards of all participating institutions. After providing written informed consent, participants completed an interview and a series of self-report questionnaires. All questionnaires were

administered online and completed on a computer in the laboratory of one of the study sites. Next, participants completed the laboratory portion of the study. After completing tasks for the larger study, participants were instructed to sit quietly for another 5 min baseline period, and then received standardized instructions for completing the PASAT-C. Once participants confirmed that they understood the instructions, the PASAT-C began. Participants were reimbursed $75 for this session.

2.3. Results

2.3.1. Preliminary Analyses—Several variables (i.e., emotion composites of fear and shame/guilt) exhibited non-normal distributions (skew > 3.0, kurtosis > 10.0) [87,88] and were log (base 10) transformed, resulting in normal distributions (with the exception of pre-stressor shame/guilt ratings, which remained leptokurtic; kurtosis = 24.66, SE = 0.22).[1] Based on self-report measures examining symptoms of MDD (i.e., DASS-21) and BPD (i.e., BEST), participants were classified into one of four groups: (1) Low BPD/Low MDD (n = 322), (2) Low BPD/High MDD (n = 38), (3) High BPD/Low MDD (n = 62), and (4) High BPD/High MDD (n = 66).

Chi-square analyses and analyses of variance (ANOVA) were conducted to examine demographic equivalence across the four groups. Given the small number of participants in several of the race/ethnicity categories, this variable was collapsed into a dichotomous variable of White (61.5%) versus non-White (38.5%). The groups were comparable in terms of racial/ethnic status (White vs. non-White; χ^2 [3] = 1.78, p = .62) and age (F [3, 484] = 0.09, p = .96). Zero-order correlations and t tests were conducted to explore the impact of demographic factors on the dependent variables (i.e., emotional reactivity on the PANAS-NA, DERS scores, and PASAT-C quit time) to identify potential covariates for primary analyses [89]. Demographic variables generally did not exhibit significant relations with the dependent variables (ps = .07–.99), with a few exceptions: age (r = −.12, p = .01) and non-White race/ethnicity (t = 2.29, p = .02) were associated with latency to terminate the PASAT-C, and White race/ethnicity was associated with greater emotion dysregulation overall and across five of the DERS subscales (i.e., nonacceptance, goals, strategies, awareness, and clarity; ts = 2.17 – 4.42, ps ≤ .03). As such, age and racial/ethnic background were included as covariates in relevant analyses. In addition, to ensure that differences in emotional reactivity post-stressor were not due simply to any group differences in latency to terminate the PASAT-C, latency to terminate scores were included as a covariate in all analyses of emotional reactivity.

2.3.2. Manipulation Check—Providing support for the use of the PASAT-C as a measure of the unwillingness to experience emotional distress, results of a 2 (quit vs. no quit) ×3 (pre-stressor vs. peak-stressor vs. post-stressor) repeated measures ANOVA for NA revealed a significant main effect of time, F (2, 952) = 286.22, p < .001, η_p^2 = 0.38,

[1]Non-parametric (Kruskal-Wallis) analyses of shame and guilt ratings revealed some significant differences across groups. Although the residualized change score of shame/guilt ratings from pre- to peak-stressor did not differ across groups (H [3] = 1.27, p = .74), there were significant group differences in terms of the residualized shame/guilt ratings from peak- to post-stressor (H [3] = 10.24, p = .02), with larger increases in shame/guilt found within the High BPD/High MDD group, relative to the Low BPD/Low MDD group, p = .03.

demonstrating that the PASAT-C resulted in a significant increase in NA across all participants. Further, the quit × time interaction was not significant, F (2, 952) = 0.81, p = .42, η_p^2 = 0.002, indicating that the decision to quit the task was not due simply to the experience of greater NA.

2.3.3. Primary Analyses

Subjective emotional reactivity: To examine patterns of emotional reactivity to the laboratory stressor, we conducted a series of 4 (Group: Low BPD/Low MDD vs. Low BPD/High MDD vs. High BPD/Low MDD vs. High BPD/High MDD) x 3 (Time: Pre-PASAT-C, Peak-PASAT-C, Post-PASAT-C) analyses of covariance (ANCOVAs) with overall NA and the NA composites as dependent variables, and PASAT-C latency to termination scores as a covariate (with the Huynh-Feldt correction applied in cases of sphericity). Analyses were followed-up with Bonferroni-corrected pairwise comparisons.

As shown in Table 1, results revealed significant effects of Time for overall NA, distress, shame/guilt, anxiety, and anger, with heightened levels of negative emotions reported at peak- and post-stressor, relative to pre-stressor. Furthermore, there were significant effects of Group for all negative emotions except shame/guilt, with the Low BPD/Low MDD group reporting lower levels of anxiety than the Low BPD/High MDD and High BPD/High MDD groups, and lower levels of overall NA, distress, and anger relative to all other groups. Finally, there was a significant Group x Time interaction for fear, such that only the Low BPD/Low MDD group had significant increases in fear from pre- to peak-stressor, $p < .01$ (ps > .53 for the other three groups). Conversely, only the High BPD/High MDD group had a significant increase in reported fear from peak- to post-stressor, p = .001 (resulting in higher levels of fear post-stressor, relative to the Low BPD/Low MDD group, p = .002).

Behaviorally-indexed emotion dysregulation: To examine group differences in the unwillingness to experience emotional distress, we conducted an ANCOVA with Group (Low BPD/Low MDD vs. Low BPD/High MDD vs. High BPD/Low MDD vs. High BPD/High MDD) as the independent variable, latency to terminate the PASAT-C as the dependent variable, and age and race/ethnicity as covariates. As shown in Table 1, there was no significant effect of Group.

Subjective emotion dysregulation: To examine group differences in self-reported emotion dysregulation, we conducted an ANCOVA with Group (Low BPD/Low MDD vs. Low BPD/High MDD vs. High BPD/Low MDD vs. High BPD/High MDD) as the independent variable and total DERS as the dependent variable, as well as a multivariate analysis of covariance (MANCOVA) with the DERS subscales as the dependent variables. Race/ethnicity status was included as a covariate in these analyses. Results of the ANCOVA revealed a significant effect of Group for the total DERS, F (3, 483) = 98.74, $p < .001$, η_p^2 = .38. Likewise, the multivariate test revealed a significant effect of Group for the DERS subscales as a whole, F (21, 1352.47) = 16.53, $p < .001$, η_p^2 = .17, which was also found for all subscales of the DERS, Fs = 14.37–94.47, ps < .001, η_p^2s = .08–.37 (see Table 1). As shown in Table 1, the Low BPD/Low MDD group reported lower levels of emotion dysregulation across all DERS scales, compared with all other groups, ps < .05. In addition,

the High BPD/High MDD group reported greater levels of both overall emotion dysregulation and the specific dimensions involving emotional nonacceptance and difficulties controlling impulsive behaviors when distressed than both Low BPD groups, *p*s < .05, as well as greater difficulties accessing effective emotion regulation strategies than all other groups, *p*s < .05. Finally, the High BPD/Low MDD group reported significantly greater difficulties controlling impulsive behaviors when distressed than the Low BPD/High MDD group, $p = .01$.

2.3.4. Group Differences in Emotional Reactivity and Dysregulation Controlling for Traumatic Exposure—

To ensure that the observed group differences in emotional reactivity and emotion dysregulation are not due solely to the shared associations of BPD, MDD, and emotional dysfunction with traumatic exposure [90–96], we reran the primary analyses including the number of PTE participants experienced as a covariate. Findings did not change when number of PTE was included in the models.

2.4. Discussion

Findings from this study suggest that individuals with high levels of both BPD and MDD pathology evidence heightened emotional dysfunction, relative to individuals with low levels of these symptoms. Specifically, participants high in both BPD and MDD pathology exhibited greater subjective emotional reactivity than participants low in both BPD and MDD pathology, as well as greater emotion dysregulation (both overall and across the specific dimensions involving emotional nonacceptance, limited access to effective regulation strategies, and difficulties controlling impulsive behaviors when distressed) than participants low in BPD pathology. Likewise, with regard to the time course of emotional reactivity to the laboratory stressor, individuals high in both BPD and MDD pathology reported heightened prolonged (but not immediate) fear reactivity to the stressor, compared with participants low in both BPD and MDD pathology. Finally, individuals high in BPD (but not MDD) pathology reported greater difficulties controlling impulsive behaviors when distressed than those high in MDD (but not BPD) pathology.

3. Study 2

3.1. Aims

The goal of this study was to extend the findings of Study 1 by examining emotional reactivity and dysregulation in a clinical sample of substance use disorder (SUD) inpatients, obtaining interview-based diagnoses of BPD and MDD, and including a biological index of emotional reactivity (i.e., salivary cortisol in response to the laboratory stressor; [97,98]). Given the high rates of both BPD and MDD among SUD patients [99], this was considered an optimal sample for examining patterns of emotional dysfunction in BPD, MDD, and their co-occurrence. Likewise, given that the presence of a co-occurring SUD has been found to exacerbate BPD and MDD symptoms relative to either of these disorders alone [100,101], the use of a SUD sample increases the likelihood of capturing clinically-relevant levels of BPD and MDD pathology (especially in contrast to the community sample used in Study 1). Finally, given that SUD patients in general exhibit heightened levels of emotional reactivity and emotion dysregulation [78,102,103], examination of our hypotheses within this sample

was expected to provide a more conservative test of the relevance of emotional reactivity and emotion dysregulation to BPD, MDD, and BPD-MDD co-occurrence.

3.2. Material and Methods

3.2.1. Participants—Data were collected as part of a larger study examining predictors of residential SUD treatment dropout. The larger study recruited consecutively admitted patients in a residential SUD treatment facility in central Mississippi. Participants in the current study (N = 176; 60.2% male) ranged in age from 18 to 61 years (M = 35.7, SD = 10.2) and were ethnically diverse (55.7% White; 35.2% African American/Black; 5.1% Native American; 1.7% Latino/a). With regard to their highest level of educational attainment, 33.0% of participants had received their high school diploma or GED, and 39.2% had completed at least some higher education. Most participants (81.0%) reported a household income less than $30,000 and were single (68.8%). To be eligible for inclusion in this study, participants were required to have a Mini-Mental Status Exam [104] score of ≥ 24 and no psychotic symptoms. Eligible participants were recruited for this study no sooner than 72 hrs after entry in the facility (to limit the possible interference of withdrawal symptoms on study engagement).

3.2.2. Clinical Interviews—The Structured Clinical Interview for DSM-IV Axis I Disorders (SCID) [105] was used to assess for the presence of lifetime and current MDD and the Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV) [74] was used to assess for the presence of BPD and its symptoms. Given the low base rate of current (i.e., past month) MDD in this sample (n = 34), as well as evidence of persistent emotional dysfunction among individuals with remitted MDD [41,43,44], we used lifetime MDD diagnoses to categorize participants into High and Low MDD groups. Likewise, given the documented associations of subthreshold BPD (i.e., 3 or 4 BPD criteria) with emotional dysfunction [61] and disability [106], participants endorsing 3 or 4 BPD criteria (n = 58) were excluded from the present study to reduce overlap between High and Low BPD groups.

Both the SCID and DIPD-IV have demonstrated adequate interrater and test-retest reliability [105,107]. Interviews were conducted by bachelors- or masters-level clinical assessors trained to reliability with one of the investigators (KLG or MTT). All interviews were reviewed by a PhD-level psychologist (MTT), with diagnoses confirmed in consensus meetings. For the SCID, diagnostic discrepancies were found in fewer than 5% of cases. For the DIPD-IV, diagnostic discrepancies were found in fewer than 10% of cases. In these instances, areas of disagreement were discussed as a group and a consensus was reached.

3.2.3. Self-report Measures—As in Study 1, the depression scale of the DASS-21 [75,76] was used to assess current depression symptoms and the DERS [31] was used to assess overall emotion dysregulation and its six specific dimensions. Internal consistency in the current sample was acceptable for the depression scale of the DASS-21 (α = 0.88), the overall DERS score (α = .92), and the DERS subscale scores (αs = .72–.88). Additionally, the number of PTE experienced by participants was assessed using the LEC [81] and

included in follow up analyses to control for the influence of traumatic exposure on the relations of interest.

3.2.4. Laboratory Measures—As in Study 1, the empirically-supported PASAT-C [82,84] was used to assess both emotional reactivity and emotion dysregulation in the form of the unwillingness to experience emotional distress [32,35,79]. The version of the PASAT-C used in this study was identical to that used in Study 1, with the exception of the duration of the first and second levels (i.e., the first level lasted 1 min, and the second level lasted 5 min).

Subjective emotional reactivity: In line with other studies assessing reactivity to the PASAT-C [29,84], participants were asked to report on their levels of anxiety, irritation, and frustration before the task (pre-stressor), following completion of the third (most difficult) level of the task (peak-stressor), and after the task (post-stressor). Each item was rated from 0 to 100, with the average rating across all three forms of NA combined to create a composite NA variable. In addition, an *anger* composite was computed (derived from "irritable," "frustrated"), and anxiety was indexed by the single-item assessment. Internal consistency of the NA (αs = 0.78–0.82) and the two-item anger composite (*r*s = .73–.75, *p*s < .001) variables was adequate in this sample.

Biological index of emotional reactivity: Saliva samples were obtained at three time points during the study: (a) immediately before the PASAT-C (pre-stressor), (b) 20 min following the PASAT-C (peak-stressor; given evidence that cortisol levels do not peak for approximately 20 min following presentation of an emotionally-evocative cue [98,108]), and (c) 40 min following the PASAT-C (post-stressor). Participants were asked to place a swab under their tongue for at least 1 min, and to place the saturated swab into a plastic vial that was then sealed and stored in a freezer. All samples were assayed in duplicate for salivary cortisol off-site by Salimetrics, LLC using a highly sensitive enzyme immunoassay (Salimetrics, State College, PA). The test used 25 μL of saliva per determination, with a lower limit of sensitivity of 0.003 μg/dL, standard curve range from 0.012 μg/dL to 3.0 μg/dL, an average intra-assay coefficient of variation (CV) of 3.8%, and an average inter-assay CV of 5.1%. Given the influence of the time of day, caffeine, and food intake on cortisol levels [108], these variables were examined as potential covariates in the cortisol analyses.

3.2.5. Procedures—All procedures were reviewed and approved by the institution's Institutional Review Board. Patients who met inclusion criteria were provided with information about study procedures and associated risks, following which written informed consent was obtained. The study took part in two sessions conducted on separate days (to limit participant burden). In the first session, participants were interviewed with the SCID and the DIPD-IV. In the second session (occurring approximately 4 days after the first session), participants completed a battery of questionnaires, followed by the laboratory procedures. Specifically, after they were provided with instructions for completing the PASAT-C, saliva samples were collected and participants were asked to rate their current levels of anxiety, irritation, and frustration (providing a baseline assessment of subjective

emotional responses). After this baseline assessment period, participants completed the PASAT-C. A second saliva sample was obtained approximately 20 min after the PASAT-C, and a final saliva sample was obtained after an additional 20 min. Participants received $15 for completing each session.

3.3. Results

3.3.1. Preliminary Analyses—Based on clinical interviews examining symptoms of lifetime MDD (i.e., SCID) and BPD (i.e., DIPV-IV), participants were assigned to one of four groups: (1) Low BPD/Low MDD group (n = 71), defined as fewer than 3 BPD criteria and the absence of a lifetime history of MDD; (2) Low BPD/High MDD group (n = 30), defined as fewer than 3 BPD criteria and the presence of a lifetime history of MDD; (3) High BPD/Low MDD group (n = 24), defined as 5 or more BPD criteria and the absence of a lifetime history of MDD; and (4) High BPD/High MDD group (n = 51), defined as the presence of 5 or more BPD criteria and the presence of a lifetime history of MDD.[2]

Chi-square analyses and ANOVAs were conducted to examine demographic equivalence across the groups. Both High BPD groups had disproportionately more females than the other two groups (χ^2 [3] = 23.68, p < .001). Given the small number of participants in several of the race/ethnicity categories, this variable was collapsed into a dichotomous variable of White (55.7%) versus non-White (44.3%). The groups were not significantly different with regard to race/ethnicity (χ^2 [3] = 7.07, p = .07) or age (F [3, 172] = 1.89, p = .13).

The cortisol variables exhibited non-normal distributions (skew > 3.0, kurtosis > 10.0; [87,88]) and were square root transformed (consistent with recommended guidelines; [109]), resulting in normal distributions. Zero-order correlations and t tests were conducted to explore the impact of demographic factors on the dependent variables (i.e., subjective emotional responses to the PASAT-C, salivary cortisol, DERS scores, and latency to terminate the PASAT-C) to identify potential covariates for primary analyses [89]. Given the aforementioned group differences in gender, this was not included as a covariate [110]. Demographic variables did not exhibit significant relationships with the dependent variables (ps = .12–.94), with a few exceptions: i.e., age was associated with latency to terminate the PASAT-C (r = −.20, p = .01) and baseline and peak salivary cortisol (rs = −.17–.18, ps = .02–.03), and White racial/ethnic background was associated with greater emotion dysregulation on two subscales (i.e., difficulties with emotional awareness and clarity; ts = 2.14 – 3.11, ps ≤ .03). We also examined the zero-order associations between other potential covariates (i.e., time of day, time since last meal, and time since last caffeine consumption) and salivary cortisol; findings revealed that time of day, time since last meal, and time since last caffeine consumption were associated with cortisol at some assessment point (rs = .17–.26, ps < .05). As such, age was included as a covariate in analyses of latency to terminate the PASAT-C, race/ethnicity was included as a covariate in analyses of DERS subscales,

[2]Of note, independent t tests revealed no significant differences in current depressive symptoms on the DASS-21 between participants with a current versus lifetime only MDD diagnosis in the High MDD groups (ts = −0.82 and −0.92, ps > .35). Moreover, participants in the High MDD groups reported significantly greater current depressive symptoms on the DASS-21 (M = 17.27, corresponding to moderately severe symptoms) than those in the low MDD groups (who reported mild symptoms of depression on average, M = 12.07), t = 3.32, p = .001.

and age, time of day, time since last meal, and time since last caffeine consumption were included as covariates in analyses of cortisol. In addition, to ensure that differences in emotional reactivity post-stressor were not simply due to any differences in latency to terminate the PASAT-C, latency to termination scores were included as a covariate in all analyses of emotional reactivity.

3.3.2. Manipulation Check—Providing support for the use of the PASAT-C as a measure of the unwillingness to experience emotional distress, results of a 2 (quit vs. no quit) × 3 (pre-stressor vs. peak-stressor vs. post-stressor) repeated measures ANOVA for NA revealed a significant main effect of time, F (2, 432) = 86.21, $p < .001$, $\eta_p^2 = 0.29$, demonstrating that the PASAT-C resulted in a significant increase in NA across all participants. Further, the quit × time interaction was not significant, F (2, 432) = 1.98, $p = .14$, $\eta_p^2 = 0.01$, indicating that the decision to quit the task was not simply due to the greater experience of NA.

3.3.3. Primary Analyses

Subjective emotional reactivity: To examine patterns of subjective emotional reactivity to the laboratory stressor, we conducted a series of 4 (Group: Low BPD/Low MDD vs. Low BPD/High MDD vs. High BPD/Low MDD vs. High BPD/High MDD) x 3 (Time: Pre-PASAT-C, Peak-PASAT-C, Post-PASAT-C) ANCOVAs with overall NA and the anger and anxiety scores as dependent variables, and latency to terminate the PASAT-C as a covariate (with the Huynh-Feldt correction applied in cases of sphericity). Analyses were followed-up with Bonferroni-corrected pairwise comparisons.

As shown in Table 2, there were significant effects of both Time and Group for all negative emotions. With regard to the former, results revealed higher negative emotions at peak-stressor and post-stressor (relative to pre-stressor). As for the group differences in negative emotions, the Low BPD/Low MDD group reported lower levels of overall NA and anger relative to all other groups, and both High MDD groups reported greater anxiety than the Low BPD/Low MDD group. Finally, results revealed significant Group x Time interactions for both overall NA and anxiety, with both High BPD groups (but not the Low BPD/High MDD group) reporting significantly higher levels of both NA and anxiety post-stressor, relative to the Low BPD/Low MDD group (for the High BPD groups: ps ≤ .01; for the Low BPD/High MDD group, ps > .10). With regard to the specific time course of emotional responding to the stressor, although all groups reported significant increases in NA from pre- to peak-stressor, ps < .05, only the Low BPD/Low MDD and the High BPD/High MDD groups reported a significant increase in NA from peak- to post-stressor, ps ≤ .05 (with the High BPD/Low MDD group reporting a non-significant increase in NA from peak- to post-stressor; $p = .11$); conversely, the Low BPD/High MDD group reported no increase in NA from peak- to post-stressor ($p > .99$). With regard to anxiety, only the High BPD/High MDD and Low BPD/High MDD groups reported an increase from pre- to peak-stressor, ps < .05 (ps > .18 for both Low MDD groups). However, all groups other than the Low BPD/High MDD group ($p > .99$) reported significant increases in anxiety from peak- to post-stressor, ps < .05.

Biological index of emotional reactivity: To examine cortisol reactivity to the laboratory stressor, we conducted a 4 (Group: Low BPD/Low MDD vs. Low BPD/High MDD vs. High BPD/Low MDD vs. High BPD/High MDD) x 3 (Time: Pre-stressor, Peak-stressor, Post-stressor) ANCOVA with salivary cortisol as the dependent variable, and age, time of day, time since last meal, time since last caffeine consumption, and PASAT-C latency to termination scores as covariates (with the Huynh-Feldt correction applied in cases of sphericity). Although results revealed a significant effect of Time on salivary cortisol (with lower cortisol levels observed at post-stressor, relative to pre- or peak-stressor, *p*s < .001), neither the effect of Group nor the Group x Time interaction was significant (Table 2).

Behavioral index of emotion dysregulation: To examine group differences in the unwillingness to experience emotional distress, we conducted an ANCOVA with Group (Low BPD/Low MDD vs. Low BPD/High MDD vs. High BPD/Low MDD vs. High BPD/High MDD) as the independent variable, latency to terminate the PASAT-C as the dependent variable, and age as a covariate. As shown in Table 2, results revealed no significant effect of Group.

Subjective emotion dysregulation: To examine group differences in self-reported emotion dysregulation, we conducted an ANCOVA with Group (Low BPD/Low MDD vs. Low BPD/High MDD vs. High BPD/Low MDD vs. High BPD/High MDD) as the independent variable and total DERS as the dependent variable, as well as a MANCOVA with the DERS subscales as the dependent variables. Race/ethnicity was included as a covariate in both of these analyses. Results revealed a significant effect of Group for the total DERS, F (3, 162) = 16.65, $p < .001$, $\eta_p^2 = .24$, as well as a significant effect of Group for the DERS subscales as a whole, F (18, 444.55) = 3.66, $p < .001$, $\eta_p^2 = .12$ (which was found for all subscales of the DERS, with the exception of the DERS awareness subscale; see Table 2). As shown in Table 2, both High BPD groups reported significantly greater levels of both overall emotion dysregulation and the specific dimension involving difficulties controlling impulsive behaviors when distressed, compared to both Low BPD groups. Moreover, the High BPD/High MDD group reported significantly greater difficulties with emotional acceptance, emotional clarity, access to effective emotion regulation strategies, and the pursuit of goal-directed behaviors when distressed, compared to both Low BPD groups, *ps* < .05. Notably, the Low BPD/High MDD group did not report significantly greater emotion dysregulation than the Low BPD/Low MDD group.[3]

3.3.4. Group Differences in Emotional Reactivity and Dysregulation Controlling for Traumatic Exposure—

To control for the influence of traumatic exposure on the observed group differences, we reran the primary analyses including the number of PTE participants experienced as a covariate. Findings did not change when number of PTE was included in the models, with one exception: the Group x Time interaction for NA reactivity did not remain significant, $p = .11$.

[3]The pattern of findings did not change when restricting the sample to only those participants with current MDD or no history of MDD, although the Group x Time interaction for NA reactivity did not reach significance, $p = .07$.

3.4. Discussion

Consistent with the results of Study 1, findings from Study 2 provide evidence for heightened prolonged (but not immediate) subjective emotional reactivity (with regard to both overall NA and anxiety) among patients with BPD (both with and without co-occurring MDD). Likewise, findings provide further support for heightened emotion dysregulation in BPD (relative to MDD), revealing greater levels of both overall emotion dysregulation and the specific dimension involving difficulties controlling impulsive behaviors when distressed among both High BPD groups (compared to both low BPD groups), and finding no significant differences in self-reported emotion dysregulation between the Low BPD groups with and without MDD. Notably, however, results revealed no significant group differences in biologically-indexed emotional reactivity.

4. General Discussion

The overarching aim of this research was to examine unique patterns of emotional reactivity and emotion dysregulation in BPD pathology, MDD pathology, and their co-occurrence in both community and clinical samples. Findings provide partial support for study hypotheses. With regard to emotional reactivity, although both BPD and MDD pathology were associated with heightened levels of negative emotions in general (relative to low levels of BPD and MDD pathology or an absence of these diagnoses), the time course of this reactivity differed. Whereas MDD (alone or in combination with BPD) was associated with greater immediate (but not prolonged) anxiety reactivity in Study 2, BPD pathology (alone or in combination with MDD pathology) was associated with heightened prolonged emotional reactivity in both studies. Specifically, individuals high in BPD pathology (compared to those low in both MDD and BPD pathology) reported heightened emotional reactivity with regard to fear and anxiety-related emotions following prolonged exposure to the laboratory stressor (although not immediately in response to the stressor). Findings of the relevance of fear and anxiety spectrum emotions to BPD pathology are not without support in the literature [4,111,112], as individuals with BPD have been found to report heightened fear-related affective instability [18,113], demonstrate greater fear instability in their daily lives compared with depressed individuals [113], and evidence greater reactivity to fear- and threat-related cues (assessed across psychophysiological and neurological domains) [114,115].

Notably, however, these findings are among the first to highlight a distinct time course in emotional responding in BPD versus MDD pathology. Specifically, our findings suggest that whereas individuals with MDD (alone or in combination with BPD) may exhibit heightened anxiety reactivity when initially presented with a stressor, the heightened reactivity in BPD (vs. MDD) pathology is evident only after prolonged exposure to a stressor. These findings are consistent with theoretical and empirical literature emphasizing the particular relevance of prolonged emotional responses (including reactivity) to BPD [3,23,25], and underscore the importance of examining how emotional responding unfolds over time when delineating specific domains of emotional dysfunction within BPD. In particular, our findings suggest that, at least relative to individuals low in both BPD and MDD pathology, individuals with heightened BPD pathology may not exhibit stronger immediate emotional reactions to

unpleasant stimuli. However, they may exhibit stronger and more persistent emotional reactions following prolonged exposure to unpleasant emotional stimuli, indicative of impaired habituation to emotional stimuli. Such findings are consistent with recent research on the biological underpinnings of emotional dysfunction in BPD [116]. Specifically, Hazlett and colleagues [116] found that patients with BPD exhibit a normal (i.e., similar to healthy controls) pattern of amygdala activation in response to *novel* unpleasant images relative to a clinical comparison group (i.e., patients with schizotypal personality disorder). However, with repeated exposure to these unpleasant images, BPD patients exhibited prolonged and stronger amygdala activation compared to both healthy and clinical comparison groups, suggestive of an abnormal amygdala habituation response.

Importantly, these findings also have potential clinical implications, as the prolonged nature of emotional responses in BPD may result in unique clinical challenges. For instance, given the delay between stressor onset and peak emotional reactivity, it may be especially difficult for patients with BPD to identify the cue for their distress. Such delayed reactivity may pose difficulties for obtaining sufficient emotional support. Whereas high levels of distress expressed immediately after a stressful event may elicit validation within the context of social and therapeutic relationships, lower immediate distress following a stressful event may lead social supports to underestimate the difficulties experienced by patients with BPD. Furthermore, prolonged experiences of distress that persist or even increase over time could lead to fatigue of emotion regulation capacities, setting the stage for heightened emotion-related impulsivity within this population.

These differences in the time course of subjective emotional reactivity notwithstanding, it is important to note that results revealed no significant group differences in biological emotional reactivity as indexed by salivary cortisol in response to the laboratory stressor. Given that the observed group differences in subjective emotional reactivity were specific to anxiety/fear-related emotions, it is possible that our use of a laboratory stressor that elicits anger-spectrum emotions in addition to anxiety may have interfered with our ability to detect between group differences in biologically-indexed emotional reactivity. Likewise, although cortisol levels were assessed at three time points corresponding to pre-, peak-, and post-stressor (facilitating the examination of biological reactivity during and after the stressor), this design precludes the examination of differences in immediate and prolonged biological emotional reactivity. This particular biological index of emotional reactivity may be more useful for examining differences in overall emotional reactivity and recovery than differences in the time course of emotional reactivity in response to ongoing emotional stimuli. Future research should include other biological indices of emotional reactivity that may be more sensitive to change in response to the duration of stressor exposure, such as affective startle eye-blink response [117] and amygdala blood oxygen level-dependent response [116]. Additionally, given evidence that certain co-occurring symptoms may influence cortisol reactivity in BPD (with heightened reactivity observed only at low levels of PTSD symptoms [29] or high levels of dissociation [30]), future research should assess the impact of these and other co-occurring symptoms on biological emotional reactivity in BPD and MDD.

As for group differences in emotion dysregulation, consistent with both theory and past research [54,60,118], results revealed that BPD pathology was associated with greater emotion dysregulation across a broad range of domains compared to MDD pathology alone. Furthermore, the heightened emotion dysregulation found in BPD was particularly pronounced for the dimension of emotion dysregulation involving the control of impulsive behaviors in the context of emotional distress. These findings are in line with recent research underscoring the relevance of emotion-related impulsivity in particular in BPD pathology [54,118], and highlight the strong links between emotional and behavioral regulation within this population. Surprisingly, however, findings revealed no significant group differences in the behavioral measure of emotion dysregulation. These findings are inconsistent with past research indicating greater unwillingness to experience emotional distress on this measure among individuals with BPD [32,34]. The absence of group differences in Study 1 could be due to the use of a community sample wherein BPD and MDD pathology was likely less severe than would be found within a clinical sample. Conversely, the sample for Study 2 was composed entirely of SUD patients in residential treatment. Studies have found that the unwillingness to experience distress may be a dimension of emotion dysregulation that is particularly relevant to SUD patients regardless of their co-occurring psychopathology [119,120]. Future research would benefit from comparing patients with BPD and MDD diagnoses to healthy comparison groups or comparison groups characterized by lower levels of psychopathology, similar to past studies which have demonstrated greater unwillingness among individuals with BPD [32].

Of note, several contradictory findings regarding the specific facets of emotional dysfunction unique to BPD, MDD, and/or their co-occurrence emerged between the two studies. In particular, in Study 1, it was the co-occurrence of BPD and MDD pathology (vs. heightened levels of either BPD or MDD pathology alone) that related to heightened levels of prolonged fear reactivity and emotion dysregulation. Conversely, in Study 2, the presence of BPD pathology in and of itself (with or without co-occurring MDD) was particularly salient with regard to emotional dysfunction, as individuals with BPD (with and without MDD) evidenced greater prolonged subjective emotional reactivity and reported greater emotion dysregulation than individuals without BPD (including those with MDD). This discrepancy may be due, in part, to the different samples and methods of assessing BPD and MDD pathology in these studies, with Study 1 examining heightened levels of BPD and MDD symptoms on self-report measures among community women and Study 2 examining threshold BPD and MDD diagnoses *per se* among SUD patients. Thus, individuals in the High BPD groups in Study 2 may have had more severe BPD pathology than those in the High BPD groups in Study 1, leading to a stronger relation between BPD and emotional dysfunction in Study 2 (as the presence of more severe BPD in these High BPD groups may have trumped the influence of other forms of pathology and dominated the clinical presentation; see reference [54]). In Study 1, on the other hand, the extent of the BPD pathology in the High BPD groups, albeit elevated, may not have been so severe as to completely dominate the clinical presentation, allowing for MDD pathology to exert an additive influence on emotional dysfunction.

The possible impact of co-occurring SUD pathology on the patterns of emotional dysfunction in BPD and MDD observed in Study 2 also warrants discussion. Specifically, findings that the presence of a co-occurring SUD among individuals with BPD or MDD may increase the severity of these disorders [100,101] suggest that the levels of emotional dysfunction observed in this study may have been elevated relative to a non-SUD sample. Conversely, the heightened emotional dysfunction associated with SUD in general [78,102,103] may interfere with the ability to detect emotional dysfunction specific to BPD and/or MDD, lowering the magnitude of the observed relations relative to a non-SUD sample. In the absence of a non-SUD control group, the precise impact of this sample on the pattern of findings in this study cannot be determined. Future research examining patterns of emotional reactivity and emotion dysregulation in BPD, MDD, and their occurrence among individuals with and without a SUD is needed to disentangle the impact of a co-occurring SUD on emotional dysfunction in BPD and MDD.

Several limitations warrant consideration. First, the number of participants with BPD pathology in the absence of depressive symptoms was quite small in both studies, which may limit the generalizability of findings relative to the BPD-only group. This is reflective of the prominence of depressive symptoms in BPD [121], and the difficulty of obtaining “pure” samples of either BPD or MDD pathology [122]. Second, although analyses of cortisol reactivity controlled for several factors found to influence cortisol levels (e.g., time of day, time since last meal/caffeine), we did not assess for all relevant factors that may influence biological responding in general or cortisol reactivity in particular, including menstrual cycle phase, physical health, medication use, and dissociation [30, 123–125]. Failure to control for these factors may have impeded our ability to detect group differences in cortisol reactivity.

Third, the present research focused on brief assessments of a range of negative emotions. Future studies could expand this research by using more comprehensive measures of emotional responding across other emotional states (e.g., positive emotional states). Finally, although the present research aimed to disentangle the unique and additive effects of BPD and MDD pathology on emotional reactivity and emotion dysregulation, future studies should examine the influence of other relevant co-occurring disorders on emotional dysfunction in BPD and MDD, including SUD [102], avoidant personality disorder [35], and PTSD [126–129]. With regard to the latter, although our findings suggest that traumatic exposure does not influence patterns of emotional reactivity and emotion dysregulation in individuals with BPD and/or MDD, it is possible that the presence of a PTSD diagnosis might. Specifically, not only is a PTSD diagnosis associated with increased emotional reactivity and emotion dysregulation relative to traumatic exposure alone [130,131], the presence of PTSD pathology has been found to influence emotional reactivity and emotion dysregulation within BPD [29,132]. Future studies examining the influence of PTSD and other co-occurring disorders on patterns of emotional dysfunction in BPD, MDD, and BPD-MDD co-occurrence are needed to further explicate the precise nature of emotional dysfunction in BPD and MDD pathology.

5. Conclusions

The present research constitutes an important step forward in research on emotional dysfunction in BPD. Whereas burgeoning research has identified patterns of emotional dysfunction in BPD pathology relative to controls, there is a dearth of research differentiating emotional responding in BPD from other clinical conditions. Results of the present research suggest that individuals with heightened BPD pathology may exhibit greater prolonged negative emotional reactivity, particularly with regard to fear- and anxiety-related emotions, compared to individuals without clinically significant BPD or MDD features. Further, our findings suggest that the emotion dysregulation in BPD pathology may be larger in magnitude and/or scope than found in MDD pathology, and that BPD in particular may be associated with difficulties controlling behaviors when experiencing distress. Future research is needed to disentangle patterns of emotional reactivity in BPD and other frequently co-occurring psychiatric diagnoses, such as PTSD. Furthermore, investigation of the generalizability of our laboratory findings to real-world emotional responding is warranted and may elucidate clinically-relevant targets for intervention.

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References

1. Kring AM, Bachorowski J. Emotions and psychopathology. Cogn Emot. 1999; 13:575–99.
2. Gunderson, JG.; Zanarini, MC.; Kisiel, C. Borderline personality disorder. In: Widiger, T.; Frances, A.; Pincus, H.; Ross, R.; First, M.; Davis, W., editors. DSM-IV sourcebook. 1. Vol. 2. Washington, DC: American Psychiatric Publishing; 1995. p. 717-32.
3. Linehan, MM. Cognitive-behavioral treatment of borderline personality disorder. Guilford Press; 1993.
4. Livesley WJ, Jang KL, Vernon PA. Phenotypic and genetic structure of traits delineating personality disorder. Arch Gen Psychiatry. 1998; 55:941–8. [PubMed: 9783566]
5. Siever LJ, Davis KL. A psychobiological perspective on the personality disorders. Am J Psychiatry. 1991; 148:1647–58. [PubMed: 1957926]
6. Skodol AE, Gunderson JG, Shea MT, McGlashan TH, Morey LC, Sanislow CA, et al. The Collaborative Longitudinal Personality Disorders Study (CLPS): Overview and implications. J Pers Disord. 2005; 19:487–504. [PubMed: 16274278]
7. Skodol A, Siever L, Livesley W, Gunderson JG, Pfohl B, Widiger TA. The borderline diagnosis II: Biology, genetics, and clinical course. Biol Psychiatry. 2002; 51:951–63. [PubMed: 12062878]
8. Zanarini MC, Frankenburg FR, Hennen J, Reich DB, Silk KR. Axis I comorbidity in patients with borderline personality disorder: 6-year follow-up and prediction of time to remission. Am J Psychiatry. 2004; 161:2108–14. [PubMed: 15514413]
9. McGlashan TH, Grilo CM, Skodol AE, Gunderson JG, Shea MT, Morey LC, et al. The Collaborative Longitudinal Personality Disorders Study: Baseline Axis I/II and II/II diagnostic co-occurrence. Acta Psychiatr Scand Scand. 2000; 102:256–64.
10. Bylsma LM, Taylor-Clift A, Rottenberg J. Emotional reactivity to daily events in major and minor depression. J Abnorm Psychol. 2011; 120:155–67. [PubMed: 21319928]

11. Garnefski N, Kraaij V. Relationships between cognitive emotion regulation strategies and depressive symptoms: A comparative study of five specific samples. Pers Individ Dif. 2006; 40:1659–69.
12. Rothbart, M.; Derryberry, D. Advances in developmental psychology. In: Lamb, M.; Brown, A., editors. Advances in developmental psychology. Vol. 1. Hillsdale, NJ: Erlbaum; 1981. p. 37-86.
13. Cheavens JS, Rosenthal MZ, Daughters SB, Nowak J, Kosson D, Lynch TR, et al. An analogue investigation of the relationships among perceived parental criticism, negative affect, and borderline personality disorder features: The role of thought suppression. Behav Res Ther. 2005; 43:257–68. [PubMed: 15629754]
14. Henry C, Mitropoulou V, New AS, Koenigsberg HW, Silverman J, Siever LJ. Affective instability and impulsivity in borderline personality and bipolar II disorders: Similarities and differences. J Psychiatr Res. 2001; 35:307–12. [PubMed: 11684137]
15. Tragesser SL, Robinson RJ. The role of affective instability and UPPS impulsivity in borderline personality disorder features. J Pers Disord. 2009; 23:370–83. [PubMed: 19663657]
16. Herpertz S, Schwenger U, Kunert HJ, Lukas G, Gretzer U, Nutzmann J, et al. Emotional responses in patients with borderline as compared with avoidant personality disorder. J Pers Disord. 2000; 14:339–51. [PubMed: 11204341]
17. Herpertz S, Gretzer A, Mühlbauer V, Steinmeyer EM, Sass H. Experimental detection of inadequate affect regulation in patients with self-mutilating behavior. Nervenarzt. 1998; 69:410–8. [PubMed: 9629557]
18. Harvey PD, Ph D, Mitropoulou V, Schmeidler J, New AS, Goodman M, et al. Characterizing affective instability in borderline personality disorder. Am J Psychiatry. 2002; 159:784–8. [PubMed: 11986132]
19. Stein K. Affect instability in adults with a borderline personality disorder. Arch Psychiatr Nurs. 1996; 10:32–40. [PubMed: 8904033]
20. Stiglmayr CE, Grathwol T, Linehan MM, Ihorst G, Fahrenberg J, Bohus M. Aversive tension in patients with borderline personality disorder: A computer-based controlled field study. Acta Psychiatr Scand. 2005; 111:372–9. [PubMed: 15819731]
21. Sadikaj G, Russell JJ, Moskowitz DS, Paris J. Affect dysregulation in individuals with borderline personality disorder: Persistence and interpersonal triggers. J Pers Assess. 2010; 92:490–500. [PubMed: 20954051]
22. Stepp SD, Hallquist MN, Morse JQ, Pilkonis PA. Multimethod investigation of interpersonal functioning in borderline personality disorder. Personal Disord Theory, Res Treat. 2011; 2:490–500.
23. Jacob GA, Hellstern K, Ower N, Pillmann M, Scheel CN, Rüsch N, et al. Emotional reactions to standardized stimuli in women with borderline personality disorder: Stronger negative affect, but no differences in reactivity. J Nerv Ment Dis. 2009; 197:808–15. [PubMed: 19996718]
24. Kuo JR, Linehan MM. Disentangling emotion processes in borderline personality disorder: Physiological and self-reported assessment of biological vulnerability, baseline intensity, and reactivity to emotionally evocative stimuli. J Abnorm Psychol. 2009; 118:531–44. [PubMed: 19685950]
25. Gratz KL, Rosenthal MZ, Tull MT, Lejuez CW, Gunderson JG. An experimental investigation of emotional reactivity and delayed emotional recovery in borderline personality disorder: The role of shame. Compr Psychiatry. 2010; 51:275–85. [PubMed: 20399337]
26. Dixon-Gordon KL, Chapman AL, Lovasz N, Walters KN. Too upset to think: The interplay of borderline personality features, negative emotions, and social problem solving in the laboratory. Personal Disord Theory, Res Treat. 2011; 2:243–60.
27. Walter M, Bureau J-F, Holmes BM, Bertha EA, Hollander M, Wheelis J, et al. Cortisol response to interpersonal stress in young adults with borderline personality disorder: A pilot study. Eur Psychiatry. 2008; 23:201–4. [PubMed: 18325742]
28. Nater UM, Bohus M, Abbruzzese E, Ditzen B, Gaab J, Kleindienst N, et al. Increased psychological and attenuated cortisol and alpha-amylase responses to acute psychosocial stress in female patients with borderline personality disorder. Psychoneuroendocrinology. 2010; 35:1565–72. [PubMed: 20630661]

29. Dixon-Gordon KL, Gratz KL, Tull MT. Multimodal assessment of emotional reactivity in borderline personality pathology: The moderating role of posttraumatic stress disorder symptoms. Compr Psychiatry. 2013; 54:639–48. [PubMed: 23375184]
30. Simeon D, Knutelska M, Smith L, Baker BR, Hollander E. A preliminary study of cortisol and norepinephrine reactivity to psychosocial stress in borderline personality disorder with high and low dissociation. Psychiatry Res. 2007; 149:177–84. [PubMed: 17169436]
31. Gratz KL, Roemer L. Multidimensional assessment of emotion regulation and dysregulation: Development, factor structure, and initial validation of the Difficulties in Emotion Regulation Scale. J Psychopathol Behav Assess. 2004; 26:41–54.
32. Gratz KL, Rosenthal MZ, Tull MT, Lejuez CW, Gunderson JG. An experimental investigation of emotion dysregulation in borderline personality disorder. J Abnorm Psychol. 2006; 115:850–5. [PubMed: 17100543]
33. Beblo T, Fernando S, Kamper P. Increased attempts to suppress negative and positive emotions in borderline personality disorder. Psychiatry Res. 2013; 210:505–9. [PubMed: 23871409]
34. Bornovalova MA, Gratz KL, Daughters SB, Nick B, Delany-Brumsey A, Lynch TR, et al. A multimodal assessment of the relationship between emotion dysregulation and borderline personality disorder among inner-city substance users in residential treatment. J Psychiatr Res. 2008; 42:717–26. [PubMed: 17868698]
35. Gratz KL, Tull MT, Matusiewicz AM, Breetz AA, Lejuez CW. Multimodal examination of emotion regulation difficulties as a function of co-occurring avoidant personality disorder among women with borderline personality disorder. Personal Disord Theory, Res Treat. 2013; 4:304–14.
36. Leible TL, Snell WE. Borderline personality disorder and multiple aspects of emotional intelligence. Pers Individ Dif. 2004; 37:393–404.
37. Gratz KL, Tull MT, Gunderson JG. Preliminary data on the relationship between anxiety sensitivity and borderline personality disorder: The role of experiential avoidance. J Psychiatr Res. 2008; 42:550–9. [PubMed: 17637464]
38. Yen S, Zlotnick C, Costello E. Affect regulation in women with borderline personality disorder traits. J Nerv Ment Dis. 2002; 190:693–6. [PubMed: 12409863]
39. Levine D, Marziali E, Hood J. Emotion processing in borderline personality disorders. J Nerv Ment Dis. 1997; 185:240–6. [PubMed: 9114809]
40. Chapman AL, Dixon-Gordon KL, Layden BK, Walters KN. Borderline personality features moderate the effect of a fear induction on impulsivity. Personal Disord Theory, Res Treat. 2010; 1:139–52.
41. Huffziger S, Ebner-Priemer U, Zamoscik V, Reinhard I, Kirsch P, Kuehner C. Effects of mood and rumination on cortisol levels in daily life: An ambulatory assessment study in remitted depressed patients and healthy controls. Psychoneuroendocrinology. 2013; 38:2258–67. [PubMed: 23684479]
42. Ellis AJ, Vanderlind WM, Beevers CG. Enhanced anger reactivity and reduced distress tolerance in major depressive disorder. Cognit Ther Res. 2012; 37:498–509.
43. Hankin BL, Wetter EK, Flory K. Appetitive motivation and negative emotion reactivity among remitted depressed youth. J Clin Child Adolesc Psychol. 2013; 41:611–20. [PubMed: 22901275]
44. Bagley SL, Weaver TL, Buchanan TW. Sex differences in physiological and affective responses to stress in remitted depression. Physiol Behav. 2011; 104:180–6. [PubMed: 21396947]
45. Rottenberg J, Kasch KL, Gross JJ, Gotlib IH. Sadness and amusement reactivity differentially predict concurrent and prospective functioning in major depressive disorder. Emotion. 2002; 2:135–46. [PubMed: 12899187]
46. Morris MC, Rao U, Wang L, Garber J. Cortisol reactivity to experimentally manipulated psychosocial stress in young adults at varied risk for depression. Depress Anxiety. 2014; 31:44–52. [PubMed: 23606237]
47. Bylsma LM, Morris BH, Rottenberg J. A meta-analysis of emotional reactivity in major depressive disorder. Clin Psychol Rev. 2008; 28:676–91. [PubMed: 18006196]
48. Burke HM, Davis MC, Otte C, Mohr DC. Depression and cortisol responses to psychological stress: A meta-analysis. Psychoneuroendocrinology. 2005; 30:846–56. [PubMed: 15961250]

49. Young EA, Abelson JL, Cameron OG. Effect of comorbid anxiety disorders on the hypothalamic-pituitary-adrenal axis response to a social stressor in major depression. Biol Psychiatry. 2004; 56:113–20. [PubMed: 15231443]
50. Lange C, Zschucke E, Ising M, Uhr M, Bermpohl F, Adli M. Evidence for a normal HPA axis response to psychosocial stress in patients remitted from depression. Psychoneuroendocrinology. 2013; 38:2729–36. [PubMed: 23931983]
51. Roemer L, Lee JK, Salters-Pedneault K, Erisman SM, Orsillo SM, Mennin DS. Mindfulness and emotion regulation difficulties in generalized anxiety disorder: Preliminary evidence for independent and overlapping contributions. Behav Ther. 2009; 40:142–54. [PubMed: 19433145]
52. Tull MT, Gratz KL. Further examination of the relationship between anxiety sensitivity and depression: The mediating role of experiential avoidance and difficulties engaging in goal-directed behavior when distressed. J Anxiety Disord. 2008; 22:199–210. [PubMed: 17419002]
53. Vujanovic AA, Zvolensky MJ, Bernstein A. The interactive effects of anxiety sensitivity and emotion dysregulation in predicting anxiety-related cognitive and affective symptoms. Cognit Ther Res. 2008; 32:803–17.
54. Goodman M, New AS, Triebwasser J, Collins KA, Siever L. Phenotype, endophenotype, and genotype comparisons between borderline personality disorder and major depressive disorder. J Pers Disord. 2010; 24:38–59. [PubMed: 20205498]
55. Scheel CN, Schneid E-M, Tuescher O, Lieb K, Tuschen-Caffier B, Jacob GA. Effects of shame induction in borderline personality disorder. Cognit Ther Res. 2013; 37:1160–8.
56. Comtois KA, Cowley DS, Dunner DL, Roy-Byrne PP. Relationship between borderline personality disorder and Axis I diagnosis in severity of depression and anxiety. J Clin Psychiatry. 1999; 60:752–8. [PubMed: 10584763]
57. Levy KN, Edell WS, McGlashan TH. Depressive experiences in inpatients with borderline personality disorder. Psychiatr Q. 2007; 78:129–43. [PubMed: 17347875]
58. Wingenfeld K, Driessen M, Adam B, Hill A. Overnight urinary cortisol release in women with borderline personality disorder depends on comorbid PTSD and depressive psychopathology. Eur Psychiatry. 2007; 22:309–12. [PubMed: 17142011]
59. De La Fuente JM, Bobes J, Vizuete C, Mendlewicz J. Biological nature of depressive symptoms in borderline personality disorder: Endocrine comparison to recurrent brief and major depression. J Psychiatr Res. 2002; 36:137–45. [PubMed: 11886691]
60. Glenn CR, Klonsky ED. Emotion dysregulation as a core feature of borderline personality disorder. J Pers Disord. 2009; 23:20–8. [PubMed: 19267659]
61. Iverson KM, Follette VM, Pistorello J, Fruzzetti AE. An investigation of experiential avoidance, emotion dysregulation, and distress tolerance in young adult outpatients with borderline personality disorder symptoms. Personal Disord Theory, Res Treat. 2012; 3:415–22.
62. Conklin CZ, Bradley R, Westen D. Affect regulation in borderline personality disorder. J Nerv Ment Dis. 2006; 194:69–77. [PubMed: 16477183]
63. Obradović J, Bush NR, Boyce WT. The interactive effect of marital conflict and stress reactivity on externalizing and internalizing symptoms: The role of laboratory stressors. Dev Psychopathol. 2011; 23:101–14. [PubMed: 21262042]
64. Soloff P, Lynch K, Kelly TM, Malone KM, Mann JJ. Characteristics of suicide attempts of patients with major depressive episode and borderline personality disorder: A comparative study. Am J Psychiatry. 2000; 157:601–8. [PubMed: 10739420]
65. Pfohl, B.; Blum, N. Unpublished measure. Iowa City: University of Iowa; 1997. Borderline Evaluation of Severity over Time.
66. Blum N, Pfohl B, John DS, Monahan P, Black DW. STEPPS: A cognitive-behavioral systems-based group treatment for outpatients with borderline personality disorder—a preliminary report. Compr Psychiatry. 2002; 43:301–10. [PubMed: 12107867]
67. Pfohl B, Blum N, John DS, Mccormick B, Allen J, Black DW. Reliability and validity of the borderline evaluation of severity over time (BEST): A self-rated scale to measure severity and change in persons with borderline personality disorder. J Pers Disord. 2009; 23:281–93. [PubMed: 19538082]

68. Gratz KL, Breetz A, Tull MT. The moderating role of borderline personality in the relationships between deliberate self-harm and emotion-related factors. 2010; 107:96–107.
69. Gratz KL, Tull MT. Extending research on the utility of an adjunctive emotion regulation group therapy for deliberate self-harm among women with borderline personality pathology. Personal Disord Theory, Res Treat. 2011; 2:316–26.
70. Gratz KL, Levy R, Tull M. Emotion regulation as a mechanism of change in an acceptance-based emotion regulation group therapy for deliberate self-harm among women with borderline personality pathology. J Cogn Psychother. 2012; 26:365–80.
71. Gratz KL, Dixon-Gordon KL, Tull MT. Predictors of treatment response to an adjunctive emotion regulation group therapy for deliberate self-harm among women with borderline personality disorder. Personal Disord Theory, Res Treat. 2014; 5:97–107.
72. Trull TJ. Borderline personality disorder features in nonclinical young adults: 1. Identification and validation. Psychol Assess. 1995; 7:33–41.
73. Gratz KL, Gunderson JG. Preliminary data on an acceptance-based emotion regulation group intervention for deliberate self-harm among women with borderline personality disorder. Behav Ther. 2006; 37:25–35. [PubMed: 16942958]
74. Zanarini, MC.; Frankenburg, FR.; Sickel, AE.; Yong, L. The Diagnostic Interview for DSM-IV Personality Disorders (DIPD-IV). Boston, MA: McLean Hospital; 1996.
75. Lovibond, SH.; Lovibond, PF. Manual for the Depression Anxiety Stress Scales. 2. Sydney, AU: Psychology Foundation; 1995.
76. Lovibond PF, Lovibond SH. The structure of negative emotional states: Comparison of the Depression Anxiety Stress Scales (DASS) with the Beck Depression and Anxiety Inventories. Behav Res Ther. 1995; 33:335–43. [PubMed: 7726811]
77. Brown TA, Chorpita BF, Korotitsch W, Barlow DH. Psychometric properties of the Depression Anxiety Stress Scales (DASS) in clinical samples. Behav Res Ther. 1997; 35:79–89. [PubMed: 9009048]
78. Gratz KL, Tull MT. The relationship between emotion dysregulation and deliberate self-harm among inpatients with substance use disorders. Cognit Ther Res. 2010; 34:544–53.
79. Gratz KL, Bornovalova MA, Delany-Brumsey A, Nick B, Lejuez CW. A laboratory-based study of the relationship between childhood abuse and experiential avoidance among inner-city substance users: The role of emotional nonacceptance. Behav Ther. 2007; 38:256–68. [PubMed: 17697851]
80. Vasilev CA, Crowell SE, Beauchaine TP, Mead HK, Gatzke-Kopp LM. Correspondence between physiological and self-report measures of emotion dysregulation: A longitudinal investigation of youth with and without psychopathology. J Child Psychol Psychiatry. 2009; 50:1357–64. [PubMed: 19811585]
81. Gray MJ, Litz BT, Hsu JL, Lombardo TW. Psychometric properties of the life events checklist. Assessment. 2004; 11:330–41. [PubMed: 15486169]
82. Brown RA, Lejuez CW, Kahler CW, Strong DR. Distress tolerance and duration of past smoking cessation attempts. J Abnorm Psychol. 2002; 111:180–5. [PubMed: 11866171]
83. Daughters SB, Lejuez CW, Kahler CW, Strong DR, Brown RA. Psychological distress tolerance and duration of most recent abstinence attempt among residential treatment-seeking substance abusers. Psychol Addict Behav. 2005; 19:208–11. [PubMed: 16011392]
84. Lejuez C, Kahler CW, Brown RA. A modified computer version of the paced auditory serial addition task (PASAT) as a laboratory-based stressor. Behav Ther. 2003; 26:290–3.
85. Tull MT, Jakupcak M, Roemer L. Emotion suppression: A preliminary experimental investigation of its immediate effects and role in subsequent reactivity to novel stimuli. Cogn Behav Ther. 2010; 39:114–25. [PubMed: 20127540]
86. Watson D, Clark LA, Tellegen A. Development and validation of brief measures of positive and negative affect: The PANAS scales. J Pers Soc Psychol. 1988; 54:1063–70. [PubMed: 3397865]
87. Curran P, West S, Finch J. The robustness of test statistics to nonnormality and specification error in confirmatory factor analysis. Psychol Methods. 1996; 1:16–29.
88. Kline, RB. Principles and practice of structural equation modeling. 2. New York, NY: Guilford Press; 2005.

89. Tabachnick, BG.; Fidell, LS. Using multivariate statistics. 5. Boston: Pearson Education, Inc./ Allyn and Bacon; 2001.

90. Shalev AY, Freedman S, Peri T, Brandes D, Sahar T, Orr SP, et al. Prospective study of posttraumatic stress disorder and depression following trauma. Am J Psychiatry. 1998; 155:630–7. [PubMed: 9585714]

91. Bleich A, Koslowsky M, Dolev A, Lerer B. Post-traumatic stress disorder and depression: An analysis of comorbidity. Br J Psychiatry. 1997; 170:479–82. [PubMed: 9307701]

92. Yen S, Shea M, Battle C. Traumatic exposure and posttraumatic stress disorder in borderline, schizotypal, avoidant, and obsessive-compulsive disorders: Findings from the Collaborative Longitudinal Personality Disorders Study. J Nerv Ment Dis. 2002; 190:510–8. [PubMed: 12193835]

93. Westphal M, Olfson M, Bravova M, Gameroff MJ, Gross R, Wickramaratne P, et al. Borderline personality disorder, exposure to interpersonal trauma, and psychiatric comorbidity in urban primary care patients. Psychiatry. 2013; 76:365–80. [PubMed: 24299094]

94. Miles SR, Teten A, Stanford M, Sharp C, Menefee D, Kent TA. Emotion dysregulation mediates the relationship between traumatic exposure and aggression in healthy young women. Pers Individ Dif. 2015; 76:222–7.

95. Gaher RM, Hofman NL, Simons JS, Hunsaker R. Emotion regulation deficits as mediators between trauma exposure and borderline symptoms. Cognit Ther Res. 2013; 37:466–75.

96. Glaser JP, van Os J, Portegijs PJM, Myin-Germeys I. Childhood trauma and emotional reactivity to daily life stress in adult frequent attenders of general practitioners. J Psychosom Res. 2006; 61:229–36. [PubMed: 16880026]

97. Denson TF, Spanovic M, Miller N. Cognitive appraisals and emotions predict cortisol and immune responses: A meta-analysis of acute laboratory social stressors and emotion inductions. Psychol Bull. 2009; 135:823–53. [PubMed: 19883137]

98. Dickerson SS, Kemeny ME. Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. Psychol Bull. 2004; 130:355–91. [PubMed: 15122924]

99. Chen KW, Banducci AN, Guller L, Macatee RJ, Lavelle A, Daughters SB, et al. An examination of psychiatric comorbidities as a function of gender and substance type within an inpatient substance use treatment program. Drug Alcohol Depend. 2011; 118:92–9. [PubMed: 21514751]

100. Links PS, Eynan R, Heisel MJ, Nisenbaum R. Elements of affective instability associated with suicidal behaviour in patients with borderline personality disorder. Can J Psychiatry. 2008; 53:112–6. [PubMed: 18357929]

101. Davis LL, Rush JA, Wisniewski SR, Rice K, Cassano P, Jewell ME, et al. Substance use disorder comorbidity in major depressive disorder: An exploratory analysis of the sequenced treatment alternatives to relieve depression cohort. Compr Psychiatry. 2005; 46:81–9. [PubMed: 15723023]

102. Fox HC, Axelrod SR, Paliwal P, Sleeper J, Sinha R. Difficulties in emotion regulation and impulse control during cocaine abstinence. Drug Alcohol Depend. 2007; 89:298–301. [PubMed: 17276626]

103. Sinha R, Fox HC, Hong KA, Bergquist K, Bhagwagar Z, Siedlarz KM. Enhanced negative emotion and alcohol craving, and altered physiological responses following stress and cue exposure in alcohol dependent indiviudals. Neuropsychopharmacology. 2009; 34:1198–208. [PubMed: 18563062]

104. Folstein MF, Folstein SE, McHugh PR. “Mini-mental state” A practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res. 1975; 12:189–98. [PubMed: 1202204]

105. First, MB.; Spitzer, RL.; Gibbon, M.; Williams, JBW. Structured clinical interview for DSM-IV Axis I disorders- Patient Edition (SCID-I/P, Version 2.0). 1996.

106. Wright ACG, Zalewski M, Hallquist MN, Hipwell AE, Stepp SD. Developmental trajectories of borderline personality disorder symptoms and psychosocial functioning in adolescence. J Pers Disord. in press.

107. Zanarini MC, Skodol AE, Bender D, Dolan R, Sanislow C, Schaefer E, et al. The Collaborative Longitudinal Personality Disorders Study: Reliability of Axis I and II diagnoses. J Pers Disord. 2000; 14:291–9. [PubMed: 11213787]

108. Nicolson, NA. Measurement of Cortisol. In: Luecken, LJ.; Gallo, C., editors. Handbook of physiological research methods in health psychology. Thousand Oaks, CA: Sage Publications; 2007. p. 37-74.
109. Hamilton, LC. Regression with graphics: A second course in applied statistics. Belmont, CA: Duxbury; 1992.
110. Miller GA, Chapman JP. Misunderstanding analysis of covariance. J Abnorm Psychol. 2001; 110:40–8. [PubMed: 11261398]
111. Gunderson JG, Singer MT. Defining borderline patients: An overview. Am J Psychiatry. 1975; 132:1–10. [PubMed: 802958]
112. Snyder S, Pitts WM. Characterizing anxiety in the DSM-III borderline personality disorder. J Pers Disord. 1988; 2:93–101.
113. Trull TJ, Solhan MB, Tragesser SL, Jahng S, Wood PK, Piasecki TM, et al. Affective instability: Measuring a core feature of borderline personality disorder with ecological momentary assessment. J Abnorm Psychol. 2008; 117:647–61. [PubMed: 18729616]
114. Silbersweig D, Clarkin JF, Goldstein M, Kernberg OF, Tuescher O, Levy KN, et al. Failure of frontolimbic inhibitory function in the context of negative emotion in borderline personality disorder. Am J Psychiatry. 2007; 164:1832–41. [PubMed: 18056238]
115. Baskin-Sommers A, Vitale J, MacCoon D, Newman J. Assessing emotion sensitivity in female offenders with borderline personality symptoms: Results from a fear-potentiated startle paradigm. J Abnorm Psychol. 2012; 121:477–83. [PubMed: 22250659]
116. Hazlett EA, Zhang J, New AS, Zelmanova Y, Goldstein KE, Haznedar MM, et al. Potentiated amygdala response to repeated emotional pictures in borderline personality disorder. Biol Psychiatry. 2012; 72:448–56. [PubMed: 22560044]
117. Hazlett EA, Speiser LJ, Goodman M, Roy M, Carrizal M, Wynn JK, et al. Exaggerated affect-modulated startle during unpleasant stimuli in borderline personality disorder. Biol Psychiatry. 2007; 62:250–5. [PubMed: 17258691]
118. Crowell SE, Beauchaine TP, Hsiao RC, Vasilev CA, Yaptangco M, Linehan MM, et al. Differentiating adolescent self-injury from adolescent depression: Possible implications for borderline personality development. J Abnorm Child Psychol. 2012; 40:45–57. [PubMed: 22016199]
119. Bornovalova MA, Gratz KL, Daughters SB, Hunt ED, Lejuez CW. Initial RCT of a distress tolerance treatment for individuals with substance use disorders. Drug Alcohol Depend. 2012; 122:70–6. [PubMed: 21983476]
120. Daughters SB, Lejuez CW, Bornovalova MA, Kahler CW, Strong DR, Brown RA. Distress tolerance as a predictor of early treatment dropout in a residential substance abuse treatment facility. J Abnorm Psychol. 2005; 114:729–34. [PubMed: 16351393]
121. Gunderson JG, Morey LC, Stout RL, Skodol AE, Shea MT, McGlashan TH, et al. Major depressive disorder and borderline personality disorder revisited: Longitudinal interactions. J Clin Psychiatry. 2004; 65:1049–56. [PubMed: 15323588]
122. McGlashan TH. Borderline personality disorder and unipolar affective disorder: Long-term effects of comorbidity. J Nerv Ment Dis. 1987; 175:467–73. [PubMed: 3625185]
123. Rimmele U, Seiler R, Marti B, Wirtz PH, Ehlert U, Heinrichs M. The level of physical activity affects adrenal and cardiovascular reactivity to psychosocial stress. Psychoneuroendocrinology. 2009; 34:190–8. [PubMed: 18922645]
124. Granger DA, Hibel LC, Fortunato CK, Kapelewski CH. Medication effects on salivary cortisol: Tactics and strategy to minimize impact in behavioral and developmental science. Psychoneuroendocrinology. 2009; 34:1437–48. [PubMed: 19632788]
125. Kirschbaum C, Kudielka BM, Gaab J, Schommer NC, Hellhammer DH. Impact of gender, menstrual cycle phase, and oral contraceptives on the activity of the hypothalamus-pituitary-adrenal axis. Psychosom Med. 1999; 61:154–62. [PubMed: 10204967]
126. Boden MT, Westermann S, McRae K, Kuo JR, Alvarez J, Kulkarni MR, et al. Emotion regulation and posttraumatic stress disorder: A prospective investigation. J Soc Clin Psychol. 2013; 32:296–314.

127. Ehring T, Quack D. Emotion regulation difficulties in trauma survivors: The role of trauma type and PTSD symptom severity. Behav Ther. 2010; 41:587–98. [PubMed: 21035621]

128. Tull MT, Barrett HM, McMillan ES, Roemer L. A preliminary investigation of the relationship between emotion regulation difficulties and posttraumatic stress symptoms. Behav Ther. 2007; 38:303–13. [PubMed: 17697854]

129. McTeague LM, Lang PJ, Laplante M-C, Cuthbert BN, Shumen JR, Bradley MM. Aversive imagery in posttraumatic stress disorder: Trauma recurrence, comorbidity, and physiological reactivity. Biol Psychiatry. 2010; 67:346–56. [PubMed: 19875104]

130. Tull MT, Kiel EJ, McDermott MJ, Gratz KL. The effect of trauma cue exposure on cocaine cravings a mong cocaine dependent inpatients with and without posttraumatic stress disorder: Exploring the mediating role of negative affect and discrete negative emotional states. J Exp Psychopathol. 2013; 4:485–501.

131. Weiss NH, Tull MT, Anestis MD, Gratz KL. The relative and unique contributions of emotion dysregulation and impulsivity to posttraumatic stress disorder among substance dependent inpatients. Drug Alcohol Depend. 2013; 128:45–51. [PubMed: 22917752]

132. Harned MS, Rizvi SL, Linehan MM. Impact of co-occurring posttraumatic stress disorder on suicidal women with borderline personality disorder. Am J Psychiatry. 2010; 167:1210–7. [PubMed: 20810470]

Table 1

Study 1 means across participants low and high in BPD and MDD pathology.

					Test Statistic F (η_p^2)		
	Low BPD/Low MDD (N = 322)	**Low BPD/High MDD (N = 38)**	**High BPD/Low MDD (N = 62)**	**High BPD/High MDD (N = 66)**	**Group**	**Time**	**Group X Time**
Subjective Emotional Reactivity							
NA					9.85*** (.06)	24.31*** (.05)	0.74 (.01)
Pre-NA	12.75 (3.36)[a]	16.19 (6.00)[b]	15.20 (5.25)[b]	15.34 (4.28)[b]			
Peak-NA	19.30 (6.91)[a]	23.24 (8.59)[b]	21.75 (7.59)[b]	21.78 (7.94)[a,b]			
Post-NA	18.69 (7.14)[a]	23.14 (9.02)[b]	21.10 (8.60)[a,b]	22.48 (9.51)[b]			
Distress					7.28*** (.04)	35.62*** (.07)	0.26 (< .01)
Pre-Distress	1.28 (.56)[a]	1.72 (0.84)[a,b]	1.61 (0.83)[a,b]	1.60 (0.63)[b]			
Peak-Distress	2.66 (1.14)[a]	3.16 (1.29)[a]	3.01 (1.18)[a]	2.95 (1.22)[a]			
Post-Distress	2.56 (1.19)[a]	3.20 (1.33)[b]	2.89 (1.40)[a,b]	2.92 (1.32)[a,b]			
Shame/Guilt					2.18 (.01)	5.29** (.01)	1.05 (.01)
Pre-Shame	1.07 (0.32)[a]	1.20 (0.58)[a]	1.15 (0.51)[a]	1.14 (0.39)[a]			
Peak-Shame	1.44 (0.75)[a]	1.58 (0.92)[a]	1.58 (0. 82)[a]	1.47 (0.80)[a]			
Post-Shame	1.35 (0.73)[a]	1.55 (0.85)[a]	1.53 (0.84)[a]	1.57 (0.83)[a]			
Anxiety					9.48*** (.06)	3.80* (.01)	0.69 (< .01)
Pre-Anxiety	1.63 (0.69)[a]	2.12 (0.92)[a,b]	2.02 (0.94)[b]	2.10 (1.00)[b]			
Peak-Anxiety	2.07 (1.04)[a]	2.59 (1.20)[a,b]	2.34 (1.10)[a,b]	2.64 (1.28)[b]			
Post-Anxiety	1.98 (1.08)[a]	2.55 (1.33)[a,b]	2.22 (1.16)[a,b]	2.57 (1.41)[b]			
Anger					7.85*** (.05)	28.26*** (.06)	0.23 (< .01)
Pre-Anger	1.26 (0.43)[a]	1.73 (0.87)[b]	1.53 (0.70)[b]	1.58 (0.62)[b]			
Peak-Anger	2.21 (1.03)[a]	2.78 (1.24)[a,b]	2.58 (1.26)[b]	2.48 (1.07)[a,b]			
Post-Anger	2.23 (1.09)[a]	2.77 (1.31)[a]	2.56 (1.23)[a]	2.58 (1.29)[a]			
Fear					3.90** (.02)	1.01 (< .01)	2.21* (.01)
Pre-Fear	1.14 (0.37)[a]	1.32 (0.69)[a]	1.30 (0.62)[a]	1.25 (0.48)[a]			
Peak-Fear	1.27 (0.69)[a]	1.50 (1.00)[a]	1.37 (0.77)[a]	1.35 (0.81)[a]			

					Test Statistic F (η_p^2)		
	Low BPD/Low MDD (N = 322)	**Low BPD/High MDD (N = 38)**	**High BPD/Low MDD (N = 62)**	**High BPD/High MDD (N = 66)**	**Group**	**Time**	**Group X Time**
Post-Fear	1.22 (0.62)[a]	1.49 (0.92)[a,b]	1.34 (0.74)[a,b]	1.60 (1.08)[b]			
Behavioral Emotion Regulation							
PASAT-C quit	352.66 (129.76)[a]	370.18 (111.94)[a]	387.05 (101.74)[a]	355.82 (127.02)[a]	1.35 (.01)	--	--
Subjective Emotion Regulation							
Total	67.74 (17.64)[a]	89.00 (24.52)[b]	95.50 (22.31)[b,c]	104.91 (18.96)[c]	98.74*** (.38)	--	--
Awareness	12.16 (4.40)[a]	15.45 (5.78)[b]	14.13 (4.99)[b]	15.62 (5.51)[b]	14.37*** (.08)	--	--
Acceptance	10.68 (4.70)[a]	13.95 (7.08)[b]	15.92 (6.63)[b,c]	18.48 (6.36)[c]	48.79*** (.23)	--	--
Clarity	8.75 (2.95)[a]	11.55 (4.09)[b]	12.42 (4.24)[b]	12.97 (4.25)[b]	44.66*** (.22)	--	--
Goals	12.25 (4.60)[a]	15.61 (5.36)[b]	17.03 (4.91)[b]	17.67 (4.07)[b]	39.22*** (.20)	--	--
Strategies	13.08 (4.81)[a]	19.92 (6.50)[b]	20.55 (6.93)[b]	23.69 (6.39)[c]	94.47*** (.37)	--	--
Impulse	9.19 (3.49)[a]	11.84 (5.30)[b]	14.42 (5.10)[c]	15.68 (4.91)[c]	64.59*** (.29)	--	--

Note. Untransformed means presented. Different superscripts denote significant differences between groups based on Bonferonni-corrected pairwise comparisons. BPD = borderline personality disorder. DERS = difficulties in emotion regulation scale. MDD = major depressive disorder. NA = negative affect. PASAT-C = paced auditory serial addition task–computerized version.

* $p < .05$.

** $p < .01$.

*** $p < .001$.

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Table 2

Study 2 means across participants with and without BPD and MDD diagnoses.

					Test Statistic F (η_p^2)		
	Low BPD/Low MDD (N = 71)	**Low BPD/High MDD (N = 30)**	**High BPD/Low MDD (N = 24)**	**High BPD/High MDD (N = 51)**	**Group**	**Time**	**Group X Time**
Subjective Emotional Reactivity							
NA					8.95*** (.14)	39.10*** (.19)	2.15* (.04)
Pre-NA	18.27 (23.01)[a]	23.23 (17.25)[a]	28.54 (30.89)[a]	28.87 (20.95)[a]			
Peak-NA	29.07 (25.71)[a]	49.05 (25.26)[b]	45.03 (34.00)[a,b]	51.81 (28.06)[b]			
Post-NA	35.81 (25.04)[a]	51.89 (40.45)[a,b]	58.17 (35.00)[b]	60.52 (24.46)[b]			
Anxiety							
Pre-Anxiety	22.46 (27.09)[a]	28.04 (23.93)[a]	25.58 (28.27)[a]	32.91 (25.87)[a]			
Peak-Anxiety	26.70 (28.14)[a]	48.36 (28.79)[b]	35.67 (35.21)[a,b]	45.49 (29.54)[b]			
Post-Anxiety	35.58 (28.51)[a]	46.46 (30.25)[a,b]	57.79 (34.57)[b]	57.09 (28.22)[b]			
Anger							
Pre-Anger	16.17 (24.05)[a]	20.82 (17.97)[a]	30.02 (33.73)[a]	26.85 (22.36)[a]			
Peak-Anger	30.26 (27.50)[a]	49.39 (30.89)[b]	49.71 (37.15)[a,b]	54.97 (31.80)[b]			
Post-Anger	36.07 (27.67)[a]	54.61 (51.38)[a,b]	58.35 (39.22)[a,b]	62.24 (26.64)[b]			
Biological Emotional Reactivity					1.02 (.02)	3.62* (.03)	0.65 (.02)
Pre-Cortisol	0.22 (0.20)[a]	0.19 (0.12)[a]	0.25 (0.18)[a]	0.21 (0.10)[a]			
Peak-Cortisol	0.21 (0.20)[a]	0.20 (0.13)[a]	0.25 (0.19)[a]	0.18 (0.09)[a]			
Post-Cortisol	0.16 (0.10)[a]	0.16 (0.09)[a]	0.21 (0.14)[a]	0.16 (0.09)[a]			
Behavioral Emotion Regulation							
PASAT-C quit	187.00 (187.42)[a]	141.00 (169.20)[a]	94.46 (151.58)[a]	184.78 (184.64)[a]	1.58 (.03)	--	--
Subjective Emotion Regulation							
Total	72.77 (20.21)[a]	80.18 (24.78)[a]	97.09 (23.07)[b]	101.42 (23.94)[b]	16.65*** (.24)	--	--
Awareness	15.77 (5.39)[a]	16.69 (6.90)[a]	16.91 (6.16)[a]	18.40 (5.12)[a]	1.43 (.03)	--	--
Acceptance	11.28 (5.45)[a]	11.83 (5.00)[a,b]	15.87 (6.96)[b]	14.79 (6.52)[b]	5.33** (.09)	--	--

					Test Statistic F (η_p^2)		
	Low BPD/Low MDD (N = 71)	**Low BPD/High MDD (N = 30)**	**High BPD/Low MDD (N = 24)**	**High BPD/High MDD (N = 51)**	**Group**	**Time**	**Group X Time**
Clarity	9.94 (4.07)[a]	10.21 (3.90)[a]	12.52 (4.21)[a,b]	13.15 (4.24)[b]	6.33*** (.11)	--	--
Goals	11.47 (4.07)[a]	13.27 (5.09)[a]	14.39 (5.29)[a,b]	16.30 (4.55)[b]	10.13*** (.16)	--	--
Strategies	13.57 (5.46)[a]	16.59 (7.27)[a]	21.48 (7.25)[b]	21.39 (7.30)[b]	15.97*** (.23)	--	--
Impulse	10.76 (4.72)[a]	11.66 (5.59)[a]	15.91 (6.51)[b]	17.43 (5.43)[b]	17.72*** (.16)	--	--

Note. Untransformed means presented. Different superscripts denote significant differences between groups based on Bonferonni-corrected pairwise comparisons. BPD = borderline personality disorder. DERS = difficulties in emotion regulation scale. MDD = major depressive disorder. NA = negative affect. PASAT-C = paced auditory serial addition task – computerized version.

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