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Negative Affect Instability among Individuals with Comorbid Borderline Personality Disorder and Posttraumatic Stress Disorder

Emily M. Scheiderer, Ting Wang, Rachel L. Tomko, Phillip K. Wood, and Timothy J. Trull

Department of Psychological Sciences, University of Missouri – Columbia

Abstract

Ecological momentary assessment (EMA; Stone & Shiffman, 1994) was utilized to examine affective instability (AI) in the daily lives of outpatients with borderline personality disorder (BPD; $n=78$) with and without posttraumatic stress disorder (PTSD). A psychiatric control group ($n=50$) composed of outpatients with major depressive disorder/dysthymia (MDD/DYS) was employed to compare across subgroups: BPD-only, BPD+PTSD, MDD/DYS-only, and MDD/DYS+PTSD. Compared to the BPD-only group, the BPD+PTSD group had significantly greater instability of fear and sadness, but did not significantly differ in instability of hostility or aggregate negative affect. This pattern of elevated instability of fear and sadness was not present—and, in fact, was reversed—in the MDD/DYS group. Results emphasize the importance of examining AI within the context of specific comorbidities and affect types. Treatment and research addressing AI in the context of BPD-PTSD comorbidity may benefit from a focus on fear and sadness as separate from hostility or general negative affect.

Keywords

borderline personality disorder; posttraumatic stress disorder; comorbidity; ecological momentary assessment; affective instability

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Borderline personality disorder (BPD) describes a pervasive, long-term pattern of instability, or dysregulation, of interpersonal relationships, identity, emotion, behavior, and cognition that causes significant distress and/or life impairments for the affected individual (5th edition of the *Diagnostic and Statistical Manual of Mental Disorders [DSM-5]*; American Psychological Association [APA], 2013). Emotion dysregulation is theorized to be a core

Correspondence concerning this article should be addressed to Emily M. Scheiderer, Department of Psychological Sciences, University of Missouri, 104 McAlester Hall, Columbia, MO 65211. emily.scheiderer@gmail.com.

Authorship

All authors contributed to the study design. E.M.S., T.W., R.L.T., and T.J.T. collaborated on manuscript preparation. Prior data collection was overseen by T.J.T. with consultation from P.K.W. Data analyses were performed by E.M.S., T.W., and R.L.T. under the supervision of P.K.W. and T.J.T. Revisions were made by E.M.S. and T.J.T. with consultation from T.W. All authors approved the final version of the paper for submission.

feature of BPD, from which other central features of this disorder (e.g., repeated self-injury) may follow, functioning as maladaptive attempts to modulate and cope with the rapid and intense fluctuations of affect that characterize this dysregulation (e.g., Crowell, Beauchaine, & Linehan, 2009; Glenn & Klonsky, 2009; Linehan, 1993). Emotion dysregulation in BPD is a complex, multifaceted process that includes emotional sensitivity/reactivity, heightened intensity and lability of negative affect, deficits in emotion regulation strategies, and maladaptive regulation strategies (Carpenter & Trull, 2013; Nica & Links, 2009). The present study focuses on one of these components, affective instability (AI), and particularly the impact of posttraumatic stress disorder comorbidity on AI in an outpatient BPD sample.

Posttraumatic stress disorder (PTSD) has been characterized as a severe anxiety disorder that develops in response to an event that involves actual or threatened death or serious injury, or threat to the physical integrity of oneself or others, and to which the individual's response involves intense fear, helplessness, or horror (*DSM-IV*; APA, 2000). The problematic response to this event is characterized by: intrusive recollection, or re-experiencing, of the traumatic event; avoidance of stimuli associated with the trauma; and general numbing of responsiveness, which may manifest as dissociative symptoms; and persistent hyper-arousal (APA, 2000).¹

High rates of BPD-PTSD comorbidity have been found in both clinical and community samples. Studies relying on treatment-seeking clinical samples have presented rates of PTSD among individuals with BPD ranging from 25% to 58%, and rates of BPD among individuals with PTSD ranging from 10% to 76% (see Pagura et al., 2010). Recent epidemiological studies have reported prevalence rates of comorbid PTSD among those meeting diagnosis of BPD in the range from 30% to 50% (e.g., Grant et al., 2008; Pagura et al., 2010; McGlashan et al., 2000); though some have reported lower numbers, for example 17% (Lenzenweger, Lane, Loranger, & Kessler, 2007). Odds ratios examining the association between BPD and PTSD also reflect this high comorbidity, with BPD having 7–10 times the odds of co-occurring PTSD (Grant et al., 2008; Tomko, Trull, Wood, & Sher, 2014).

Despite its substantial prevalence, the comorbidity of BPD and PTSD is not yet well understood. Existing clinical (e.g., Harned, Rizvi, & Linehan, 2010; Heffernan & Cloitre, 2000; Zlotnick et al., 2003) and community studies (e.g., Pagura et al., 2010; Pietrzak, Goldstein, Southwick, & Grant, 2011) emphasize that BPD-PTSD comorbidity presents a variety of difficulties (e.g., greater general impairment, lower levels of health-related quality of life, and increased usage of intensive healthcare services)—above and beyond that for either disorder alone. Prior findings, however, are mixed with regard to whether BPD-PTSD comorbidity affects presentation of the core features of either of the individual disorders (see, e.g., Harned et al., 2010; Pagura et al., 2010) or, rather, affects only general, non-specific impairments (e.g., facets of general functioning and health-related quality of life; e.g., Heffernan & Cloitre, 2000; Zlotnick et al., 2003).

¹Recent revisions to the *DSM* (*DSM-5*; APA, 2013), published subsequent to completion of the present study, place PTSD in a trauma- and stressor-related disorders category, separate from anxiety disorders, and alter the organization and specification of some of the criteria and developmentally sensitive diagnostic thresholds for this disorder.

Though the specific construct of affective instability (AI) has garnered less attention within the PTSD literature (as compared to the BPD literature), the broader ‘umbrella’ construct of emotion dysregulation appears to play an important role in PTSD (Marshall-Berenz, Morrison, Schumacher, & Coffey, 2011; New et al., 2009; Tull, Barrett, McMillan, & Roemer, 2007), suggesting that AI may be an important core feature of BPD to examine in the context of PTSD comorbidity. For instance, Tull et al. reported an association of posttraumatic stress symptom severity with lack of emotional acceptance, difficulty engaging in goal-directed behavior when upset, limited access to effective emotion regulation strategies, and lack of emotion clarity. The association between overall emotion regulation difficulties and posttraumatic stress symptom severity in this study remained significant when controlling for negative affect (Tull et al., 2007). Findings of an association between PTSD and heightened responding (subjective and psychophysiological) to emotionally evocative stimuli (e.g., Litz, Orsillo, Kaloupek, & Weathers, 2000; McDonagh-Coyle et al., 2001; Orsillo, Batten, Plumb, Luterek, & Roessner, 2004; Veazey, Blanchard, Hickling, & Buckley, 2004), further, suggest a potential parallel with the emotional reactivity characteristic of AI in BPD. However, findings of an association between PTSD and *attenuated* physiological response to emotionally evocative stimuli suggest that PTSD has a more complex impact on physiological responding than originally conceptualized (Limberg, Barnow, Freyburger, & Hamm, 2011; McTeague, Lang, Laplante, Cuthbert, Shumen, & Bradley, 2010).

The growing body of research on emotion dysregulation in PTSD includes various findings suggestive of an important impact of comorbid PTSD on affective responding (e.g., Harned, Rizvi, & Linehan, 2010; Marshall-Berenz et al., 2011; Rüscher et al., 2007) and psychophysiological and neural correlates of affective responding (e.g., Kraus et al., 2009; Limberg et al., 2011; McTeague et al., 2010; Rodrigues et al., 2011; Schmahl et al., 2004; Schmahl et al., 2009) in individuals with BPD. In Harned et al.’s examination of the impact of co-occurring PTSD on suicidal female outpatients with BPD, those women with the additional diagnosis of PTSD scored significantly higher (than those with BPD and no PTSD) on measures of emotion dysregulation and anger suppression, and scored lower on expression of positive emotions. Harned et al. reasoned that the pattern of emotion dysregulation theorized to be central to PTSD—i.e., a pattern involving trauma-relevant triggers associated with vacillation between overwhelming emotional experiencing and emotional numbing (e.g., Horowitz, 1986)—may exacerbate the emotion dysregulation seen in BPD. Broadly speaking, the emotion dysregulation pattern characteristic of BPD—which involves emotional reactivity to the environment (particularly to interpersonal triggers) associated with intense and labile emotional experiencing that can incur dissociation (see, e.g., Ebner-Priemer et al., 2005)—is arguably analogous to that described in PTSD. That these two patterns of dysregulation would, in combination, exacerbate each other seems quite plausible. Moreover, Harned et al. found that the additional diagnosis of PTSD in their BPD sample was associated with a trend towards greater frequency of non-suicidal self-injury (NSSI) as well as significantly greater endorsement of the use of NSSI for interpersonal influence. In line with the conceptualization of emotion dysregulation as a core facet of BPD from which self-damaging behaviors may arise as maladaptive compensatory responses (e.g., Crowell, Beauchaine, & Linehan, 2009; Glenn & Klonsky, 2009; Linehan,

1993), Harned et al. attributed this increase in NSSI to the aforementioned exacerbation of emotion dysregulation. Such a possibility highlights the clinical and public health importance of further investigating and clarifying the impact of PTSD comorbidity on emotion dysregulation in BPD.

Defining AI across disorders

Definitions of affective lability/instability reference the frequency, speed, and range of changes in affective states (e.g., Oliver & Simons, 2004). However, affective instability (AI) may exhibit disorder-specific patterns. For instance, the marked fluctuations in affect in BPD appear to occur primarily among negative affective states and in response to environmental cues, differing from, for example, the pattern of AI characteristic of bipolar disorder (APA, 2013). In contrast to the literatures on BPD and bipolar disorder, the literature on emotion dysregulation in PTSD mentions relatively little with regard to affective lability/instability in this disorder. Rather, alexithymia, maladaptive and avoidant coping styles, and limited access to and inflexible use of effective emotion regulation strategies, have received more attention and are thought to be characteristic of the pattern of emotion dysregulation in PTSD (Tull, Barrett, McMillan, & Roemer, 2007).

Though findings specifically attempting to characterize the pattern of AI in PTSD are sparse, they do suggest a pattern similar to that seen in BPD. Kashdan, Uswatte, Steger, and Julian (2006) reported higher variability in negative affect in veterans with PTSD as compared to those without PTSD. Marshall-Berenz and colleagues (2011) found that severity of the PTSD symptom of re-experiencing uniquely predicted affective lability, and severity of the PTSD symptom of hyperarousal uniquely predicted negative affect intensity in individuals with BPD. In a recent study, Santangelo and colleagues (2014) utilized ecological momentary assessment to examine the patterns of AI seen in patients with BPD as compared to those with PTSD, bulimia nervosa (BN), and healthy controls. Replicating prior findings (e.g., Ebner-Priemer et al., 2007; Jahng et al., 2008), Santangelo et al. found a pattern of AI among BPD patients that, when compared with healthy controls, was characterized by significantly heightened instability of affect valence and intensity. When compared with the PTSD and BN control groups, however, the pattern of AI found in BPD was not specific to this disorder: the patients with PTSD and BN showed patterns of heightened AI that were similar to those seen in the BPD group (Santangelo et al., 2014). In supplementary analyses to examine the impact of the highly prevalent PTSD comorbidity in their BPD group, Santangelo et al. did not find any significant difference in AI between the “pure” BPD patients ($n = 21$) and the BPD patients with comorbid PTSD ($n = 22$). These findings of a lack of specificity of AI in BPD and lack of impact of the PTSD comorbidity were somewhat surprising. Particularly, the former finding is at odds with the conventional conceptualization of AI as fundamental to BPD and patterned somewhat differently across disorders.

Santangelo et al. acknowledged the unexpected nature of their findings and worked to rule out alternative explanations (e.g., heterogeneity with regard to presence or lack of AI within the BPD group) for this ‘missing specificity’ of AI in BPD. However, it remains possible that the short duration (24 hours) and high frequency (once every 15 minutes) of data

collection, as well as the inclusion of inpatient participants, may have limited both the ecological validity of this study (i.e., its generalizability to daily life) and its ability to capture differences in AI across disorders. In the present study, we utilized similar EMA methodology to address the possibility of an impact of PTSD comorbidity on AI in BPD, but did so with EMA data collection over the course of 28 days, with longer between-collection time intervals, and from an all-outpatient sample. As discussed below, our findings present an interesting counterpoint to those presented by Santangelo and colleagues.

Currently, the presence and nature of a potential impact of PTSD comorbidity on AI in BPD remain unclear. Beyond the supplementary examination by Santangelo et al. (2014), few studies have examined this issue; and to our knowledge, no other studies focusing on this topic have used ecological momentary assessment (EMA).

Present Study

The present study examined affective instability (AI) in the daily lives of outpatients with BPD, with and without PTSD comorbidity. By utilizing EMA methodology, over the course of a 28-day data collection period, in conjunction with an analytic approach based on the mean squared successive difference (MSSD), and employing a depressive disorder control group, this study expands upon the prior empirical contributions. Specifically, this study used a major depressive disorder/dysthymia (MDD/DYS) psychiatric control group to make comparisons of affective instability across the following diagnostic groups and comorbidity subgroups: BPD-only, BPD+PTSD, MDD/DYS-only, and MDD/DYS+PTSD.

The use of a psychiatric—and particularly depressive disorder—comparison group, in the present study served multiple purposes. Reliance on healthy control groups in a majority of prior studies of AI in BPD has potentially limited progress on this topic, for example, allowing for possible third-variable explanations for group differences related to overall level of psychiatric or mood impairment (Trull et al., 2008; Solhan et al., 2009; Santangelo et al., 2014). Moreover, it is of more clinical import to distinguish emotion dysregulation patterns in BPD from those in other psychiatric disorders that are also characterized by emotion dysregulation than to distinguish patterns seen in individuals with BPD from those seen in healthy controls. Depressive disorder (including major depressive disorder [MDD] and dysthymia [DYS]) was chosen as the diagnostic category for the psychiatric control group in the present study. Like BPD, depression is associated with emotion dysregulation and elevated levels of negative affect (e.g., Falkenberg, Kohn, Schoepker, & Habel, 2012). In contrast to the increased emotional reactivity often evident in BPD, individuals with depression are thought to have a tendency toward blunted initial reactions to emotional events or stimuli (e.g., Bylsma, Morris, & Rottenberg, 2008; Peeters, Berkhof, Rottenberg, & Nicolson, 2010). Thus, a primary difference between BPD and depression is the temporal pattern of negative affect, with BPD demonstrating greater emotional reactivity and instability of affect. Previous findings reported by Trull and colleagues (2008), on a subset of the data employed in the present study, provided evidence of these distinct patterns of affective instability in BPD versus MDD/DYS, despite relatively high levels of mean negative affect in both diagnostic groups: the BPD group showed more instability of

hostility, sadness, and fear; there were no significant differences in the instability of positive affect or general negative affect across the BPD and MDD/DYS groups (Trull et al., 2008).

High rates of PTSD comorbidity among individuals with depression (e.g., Bleich Koslowsky, Dolev, & Lerer, 1997; Breslau, Davis, Andreski, & Peterson, 1991; Keane & Wolfe, 1990; Kessler et al., 1995; Kulka et al., 1990; Shalev et al., 1998) present another point of similarity between the ‘near-neighbor’ diagnoses of BPD and depression. So far, little is known regarding the specific patterns of AI characteristic of BPD-PTSD comorbidity or MDD/DYS-PTSD comorbidity; and even less is known regarding the comparison of AI across these two comorbid groups. Given the similarities and differences of emotion dysregulation in BPD and in depression, comparison of AI in the context of BPD-PTSD comorbidity versus in the context MDD/DYS-PTSD comorbidity—a comparison of primary interest in the present study—has the potential to improve our understanding of the impact of PTSD comorbidity on the presentation of AI in BPD.

Based on the literature reviewed above, we hypothesized that PTSD comorbidity generally would elevate levels of negative affect instability in the BPD group, but that this effect might not emerge consistently across the different subtypes of negative affect. In an examination of the symptom correlates of PTSD in individuals with BPD, Bolton, Mueser, and Rosenberg (2006) found significantly higher levels of depression and anxiety in the comorbid group, but no significant difference in hostility. Although this study examined mean levels, or intensity, of affect types, rather than instability, and featured a small sample size, it provided reason to consider that PTSD comorbidity with BPD may impact negative affect subtypes related to depression (e.g., sadness) and anxiety (e.g., fear), more so and/or differently than hostility.

The potential impact of PTSD comorbidity on levels of AI in the MDD/DYS group is less clear. However, we reasoned that the blunted (versus elevated) emotional reactivity associated with depression might result in smaller increases in AI than PTSD comorbidity would otherwise impart. Specifically, we hypothesized that participants with comorbid BPD and PTSD would show greater negative affect instability (both in total and particularly in the negative affect subtypes of sadness and fear) than BPD-only participants; and in contrast, PTSD comorbidity would not impact, or would not as greatly impact, negative affect instability among MDD/DYS participants.

Method

Participants

Participants were recruited from local psychiatric outpatient clinics serving the general community of a moderate sized Midwestern city that included a major university. *DSM-IV-TR* Axis I and Axis II diagnostic interviews were used to establish the eligibility of participants in a larger study examining affective instability (Trull et al., 2008). To be included in the main diagnostic group of interest for this study, participants were required to meet *DSM-IV-TR* (APA, 2000) diagnostic criteria for BPD, including endorsement of the criterion of affective instability. Eighty-one participants met criteria for BPD with affective instability (AI). Of this 81, three did not provide sufficient information to diagnose PTSD.

Of the remaining 78 who met criteria for BPD with AI, 33 (42.31%) also met criteria for lifetime PTSD. The psychiatric control group ($n=50$) was composed of psychiatric outpatients who met diagnostic criteria for current major depressive disorder (MDD) or for current dysthymia (DYS) and who did not meet criteria for either BPD or the specific symptom of affective instability. Only two potential MDD/DYS participants were excluded because they met the BPD AI criterion. Within this MDD/DYS group, 12 met criteria for comorbid lifetime PTSD (24.00%). Thus, the base rates for endorsing lifetime PTSD in this sample were different for the BPD and MDD/DYS groups. The highest psychiatric comorbidity rates within the BPD group were those of MDD (60.26%), panic disorder (46.15%), and PTSD (42.31%); and the highest within the MDD/DYS group were those of social phobia (40.00%), generalized anxiety disorder (32.00%), and avoidant personality disorder (30.00%).² Most participants (92.31% of the BPD group and 78.00% of the MDD/DYS group) were females. The mean age was 32.36 years ($SD=11.81$) for the BPD +PTSD group and 32.09 years ($SD=11.97$) for the BPD-only group. The mean age was 37.92 years ($SD=9.15$) for the MDD/DYS+PTSD group and 33.76 years ($SD=12.65$) for the MDD/DYS-only group. Most participants were Caucasian (86.26%) and had family incomes of less than \$25,000 (70.23%). The majority of participants had comorbid lifetime anxiety disorders (80.31%) and mood disorders (96.12%).³ Demographics by group are shown in Table 1. Institutional review board (IRB) approval was obtained from the University of Missouri. Participants provided written informed consent and were reimbursed US\$200.00.

PTSD diagnostic status—A large proportion of the individuals who met diagnostic criteria for lifetime PTSD in this sample also met for current PTSD (84.4%; 38 out of 45). Broken down by group, 28 of the 33 BPD+PTSD individuals met for current PTSD status, as did 10 of the 12 MDD/DYS+PTSD individuals. Given the small size of our groups and the evidence suggesting that emotional dysregulation and other functional impairments associated with the PTSD diagnosis appear to persist to a notable degree after PTSD symptom improvement (Schnurr, Hayes, Lunney, McFall, & Uddo, 2006) and even after individuals no longer meet for the current PTSD diagnostic status (Westphal et al., 2011), we included all individuals meeting for *lifetime* PTSD in the comorbid PTSD subgroups in the analyses described below. Additionally, we conducted supplementary versions of those analyses central to our research question in which we included only those individuals who met for *current* PTSD in the comorbid subgroups.

Measures

Psychiatric diagnoses—To establish eligibility and determine psychiatric comorbidities, all participants completed the Structured Clinical Interview for *DSM-IV* Axis I Disorders and the Structured Interview for *DSM-IV* Personality (First et al., 1995; Pfohl, Blum, & Zimmerman, 1994). Interviews were completed by master's-level clinical psychology graduate students who received extensive training in these diagnostic assessments (see Solhan et al., 2009). Audio recordings of 20 randomly selected participant interviews were

²Each psychiatric comorbidity rate represents lifetime diagnostic prevalence, with the exception of the rate for generalized anxiety disorder, for which only current status was assessed.

³These percentages include PTSD and MDD, respectively.

rated independently by an alternative trained interviewer to provide the reliability. Agreement was excellent for presence or absence of affective instability ($\kappa=1.0$), diagnosis of MDD/DYS ($\kappa=1.0$), diagnosis of lifetime PTSD ($\kappa=1.0$), diagnosis of BPD ($\kappa=.90$), and number of BPD symptoms (intraclass correlation coefficient=.95).

Ecological momentary assessment of affect—*Ecological momentary assessment* (EMA; Stone & Shiffman, 1994), alternatively referred to as *experience sampling methodology* (ESM; Larson & Csikszentmihalyi, 1983), utilizes repeated sampling of participants' moods, behaviors, and experiences in 'real time' and in their natural environments, thus bypassing many of the problems of retrospective recall and lack of ecological validity. With data collected repeatedly over time for each participant, EMA can account for the temporal aspect that distinguishes instability from mere variability; and it is well suited to examine instability of affect (Ebner-Priemer, Eid, Kleindienst, Stabenow, & Trull, 2009). In the present study, affect was assessed using items from the Positive and Negative Affect Schedule-Extended version (PANAS-X; Watson & Clark, 1999; see also Watson, Clark, & Tellegen, 1988). Items were presented to each participant on the electronic diary (ED) during each of six daily momentary assessments. For each mood item, respondents were asked to rate, on a five point Likert scale (1='very slightly or not at all' to 5='extremely'), the extent to which they felt the particular mood state since the last prompt. Several additional mood items were administered from the PANAS-X (Watson & Clark, 1999) in order to separately calculate the following negative affect subscales: Hostility (six items), Fear (six items), and Sadness (five items). These latter three mood subscales were used to characterize specific negative affects relevant to BPD and to the *DSM-IV-TR* definition of affective instability.

Procedure

Participants were issued an electronic diary (Palm Zire 31[®] handheld computer) for a period of 28 days. The electronic diary (ED) alarmed six times per day, prompting the individual to answer questions about current affects, experiences, and behaviors. The alarm times were determined by a software program that stratified the participants' usual waking hours (as reported by the participants prior to the study start) into six equal intervals, and then randomly selected a time within each interval. Collected data were also time-stamped to determine whether participants responded to the prompts in a timely manner and to scale affective instability more accurately as a function of time (see Trull et al., 2008 for more details regarding the electronic diary software and protocol).

Data Analysis

Mean squared successive difference (MSSD)—Temporal dependency, amplitude, and frequency of shifts in affect comprise the main quantitative components of AI (Ebner-Priemer et al., 2009), all of which must be incorporated into a statistical index in order to properly quantify AI. One such index, the *mean squared successive difference* (MSSD; von Neumann, Kent, Bellinson, & Hart, 1941) has been favored among recent EMA studies of AI (e.g., Ebner-Priemer et al., 2007; Trull et al., 2008; see Ebner-Priemer et al., 2009, and Jahng, Wood, & Trull, 2008, for further explanation and review of uses of MSSD in this literature) and has performed well as an index for comparison of AI among clinical groups

(Ebner-Priemer et al., 2009). MSSD is calculated by averaging squared successive differences (SSDs) between consecutive assessments of mood scores across the study period for each participant. By squaring the differences, SSD indices give more weight to the larger differences.

Detrended MSSD: Common definitions of AI refer to mood variation that is not systematic or predictable from previous mood states. However, mood also varies in systematic ways (e.g., by time of day) and these variations can distort analysis of true AI (Trull et al., 2008). Because the purpose of the current study is to investigate non-systematic AI, the current analyses are adjusted for four potentially influential time factors: (a) trends due to time of the day of assessment; (b) assessments obtained in weekdays versus weekends; (c) general trends over all days of assessment; (d) and trends reflecting the interaction of weekend versus weekday with time of day. In order to account for mood change not related to time-bounded ordinary cyclical change, the total variance of mood score was decomposed into variance explained by these four time factors and residual variance. These four time factors were implemented as multiple predictors to account for the variances demonstrated in mood score within each participant. The residual variance of mood score for each participant was used to calculate the MSSD, which was then referred to as the detrended MSSD. Additionally, due to the random, non-uniform scheduling of observations, the MSSDs analyzed in the current paper were adjusted for irregular temporal spacing of successive observations over time (see Jahng, Trull, & Wood, 2008).

Statistical modeling—Given an unbalanced number of observations across participants, it is less precise to calculate the individual MSSD and then compare group MSSD (Jahng et al., 2008). Thus, we instead used generalized multilevel modeling with gamma distribution and log link to test the mean difference in detrended MSSD directly and then tested whether the slope parameter for the effect of interest was significantly different from zero (Jahng et al., 2008). The multiple equation form was:

$$E(\text{Detrended SSD}|\alpha_j, \beta_j) = \alpha_j \beta_j = \mu_j$$

$$\text{Var}(\text{Detrended SSD}|\alpha_j, \beta_j) = \alpha_j \beta_j^2$$

$$\text{Level 1 link function: } \eta_j = \log(\mu_j)$$

$$\text{Level 1 structural model: } \eta_j = b_{0j}$$

$$\text{Level 2 model: } b_{0j} = \gamma_{00} + \gamma_{01}\text{Group} + \gamma_{02}\text{PTSD} + \gamma_{03}\text{PTSD*Group} + u_{0j}, u_{0j} \sim N(0, \tau^2)$$

Where the SSD is the square of successive differences, γ_{01} is the difference of the log of MSSD between the BPD and MDD/DYS group; γ_{02} is the difference of the log of MSSD between PTSD and non-PTSD, γ_{03} is the parameter indicating the interaction effect. Thus, the parameter of most interest is γ_{03} .

Results

In total, the 78 BPD patients produced 11,699 complete sets of mood ratings, and the 50 MDD/DYS patients contributed 7,339 complete sets of mood ratings. Compliance rates were calculated by dividing the number of completed and usable sets of mood ratings by the total

number of prompts while the participant was in the study (28 days*6 prompts*n participants). Average compliance rates were 0.853 (SD = 0.08) for the BPD group and 0.871 (SD = 0.07) for the MDD/DYS group; Wald's $\chi^2(1) = 1.710$, ns.

Mean affect scores did not differ significantly across the BPD and MDD/DYS groups. For the BPD group, mean scores for overall negative affect (NA; 21 items), fear (6 items), hostility (6 items), sadness (5 items), and positive affect (PA; 10 items) were the following: 1.62 (SD = 0.63), 1.61 (SD = 0.65), 1.53 (SD = 0.60), 1.74 (SD = 0.78), and 2.07 (SD = 0.56). For the MDD/DYS group, these mean affect scores were 1.58 (SD = 0.43), 1.59 (SD = 0.57), 1.38 (SD = 0.39), 1.85 (SD = 0.70), and 2.16 (0.61).⁴ Descriptive statistics for detrended MSSD of NA, fear, hostility, sadness, and PA are presented, by group and subgroup, in Table 2. The BPD group had higher detrended MSSD values than did the MDD/DYS group for each affect category. Within the BPD group, the PTSD subgroup exhibited higher MSSD values than the no-PTSD subgroup for NA and its subtypes (fear, hostility, and sadness); whereas the PTSD subgroup of the MDD/DYS group tended to show the reverse, exhibiting *lower* MSSD values than the no-PTSD subgroup for NA, fear, and sadness.

Results of our statistical modeling (Table 3) indicated that a subset of the mean differences seen in Table 2 reflected significant effects and that some important interactions between Group (BPD versus MDD/DYS) and PTSD were present in these data.⁵ Due to the focus of our central research question on whether (and how) PTSD comorbidity would affect AI differently amongst those with BPD as compared to those with MDD/DYS, we compared models with and without inclusion of the interaction of Group (BPD versus MDD/DYS) and PTSD. For the models predicting MSSD of fear and MSSD of sadness, respectively, both the Akaike information criterion (AIC) and Bayesian information criterion (BIC) indicated superior fit for those models including both the main effects and interaction of Group and PTSD. This was not the case for the models predicting MSSD of hostility or positive affect, for which the main effects-only models exhibited superior fit. For prediction of MSSD of negative affect, whereas the AIC was just slightly lower (by a difference of approximately 0.23) for the model including the interaction, the BIC was lower (by a difference of approximately 2.62) for the main effects-only model. This disagreement between the AIC and BIC, at least in part, reflects the BIC's heavier penalty for model complexity. Given the greater magnitude of difference across the BICs than across the AICs in this case, and the statistical value of parsimony, we interpreted the fit statistic results as slightly in favor of the main effects-only model for the prediction of MSSD of aggregate negative affect (NA). Moreover, examining the results across all of the full (including the interaction term) models, the maximum likelihood parameter estimates for the interaction of Group and PTSD were significant only for the models predicting MSSD of fear and MSSD of sadness. Inspection of the standard deviations of the means for each cell in the 2x2 design (see Table 2) yielded no indication that the lack of significant interactions for the MSSDs of NA,

⁴Subgroup mean affect scores are available by request.

⁵All analyses presented were repeated controlling for use of the following medication types: mood stabilizers, antipsychotics, anxiolytics, and antidepressants. Results were essentially the same, suggesting that the original pattern of results was not substantially influenced by medication usage.

hostility, and positive affect resulted from statistical artifact, as these standard deviations were all within a reasonable range.

Looking more closely at the results for instability of fear (Table 3), the interaction effect of Group (BPD versus MDD/DYS) and PTSD was significant, $\gamma_{03} = 1.079$, $p = .004$, 95% CI [0.348, 1.811]; the main effect of Group was not significant; and the main effect of PTSD was significant, $\gamma_{02} = -0.661$, $p = .031$, 95% CI [-1.262, -0.060]. The means of log detrended SSD of fear, by Group and by PTSD comorbidity status, are plotted in Figure 1. The BPD+PTSD subgroup demonstrated the highest fear instability scores among the four subgroups (BPD-only, BPD+PTSD, MDD/DYS-only, MDD/DYS+PTSD). For BPD participants, comorbidity with PTSD significantly *increased* the instability of fear (absolute difference estimate = 0.418, $p = .049$, 95% CI [0.002, 0.835]); however, for MDD/DYS participants, comorbidity with PTSD was associated with significantly *lower* instability of fear (absolute difference estimate = -0.661, $p = .031$, 95% CI [-1.262, -0.060]). The comparison of the BPD+PTSD subgroup with the MDD/DYS+PTSD subgroup, thus, was also significant (absolute difference estimate = 1.001, $p = .0014$, 95% CI [0.389, 1.613]).

For instability of sadness, the interaction effect between Group (BPD versus MDD/DYS) and PTSD was significant, $\gamma_{03} = 1.031$, $p = .004$, 95% CI [.331, 1.732]; the main effect of Group was not significant; and the main effect of PTSD was significant, $\gamma_{02} = -0.647$, $p = .028$, 95% CI [-1.223, -0.071]. The means of log detrended SSD of sadness, by group and by PTSD comorbidity status, are plotted in Figure 1. The BPD+PTSD subgroup demonstrated the highest sadness instability scores among the four subgroups (BPD-only, BPD+PTSD, MDD/DYS-only, MDD/DYS+PTSD). For BPD participants, comorbidity with PTSD *increased* the instability of sadness, but the absolute difference estimate was only marginally significant (estimate = 0.384, $p = .059$, 95% CI [-0.014, 0.783]). For MDD/DYS participants, comorbidity with PTSD was associated with significantly *lower* instability of sadness (absolute difference estimate = -0.647, $p = .028$, 95% CI [-1.223, -0.071]). As with instability of fear, comparison of the BPD+PTSD subgroup with the MDD/DYS+PTSD subgroup, yielded the largest contrast in instability of sadness (absolute difference estimate = 0.800, $p = .008$, 95% CI [0.214, 1.386]).

Supplementary results for current PTSD comorbidity

Repeating the above analyses using current rather than lifetime PTSD comorbidity (i.e., those with lifetime but not current PTSD were shifted to the BPD- and MDD/DYS-only subgroups, and the comorbid subgroups were defined as BPD+PTSD_{current} and MDD/DYS+PTSD_{current}) yielded a very similar pattern of results. Subgroup scores for mean detrended MSSD, or instability, of total negative affect, fear, sadness, hostility, and positive affect shifted minutely if at all, preserving the pattern of relative differences presented in Table 2. Results of statistical modeling indicated that PTSD comorbidity significantly affected the instability of fear and sadness, but not total negative affect, hostility, or positive affect: For the models predicting MSSD of fear and MSSD of sadness, respectively, the AIC and BIC indicated superior fit for those models including both the main effects and interaction of Group and PTSD. The main effects-only models exhibited superior fit for the prediction of MSSD of total negative affect, hostility, and positive affect. In this supplementary set of

analyses, as seen above, the only significant ($p < .05$) maximum likelihood parameter estimates for the interaction of Group and PTSD resulted from the models predicting MSSD of fear and MSSD of sadness. The only significant absolute differences comparisons of instability of sadness and fear among the subgroups resulted from comparison of the BPD+PTSD_{current} and MDD/DYS+PTSD_{current} subgroups. However, all of the within-group comparisons (e.g., BPD-only versus BPD+PTSD_{current}) were in the same direction as described above, and most of these approached significance. Thus the PTSD_{current} version of the graphs of the means of log detrended SSD of *fear* and *sadness* for BPD and MDD/DYS participants with and without PTSD are nearly identical to those presented in Figure 1.⁶

Discussion

In this study, we compared the level of affective instability (AI) in individuals with BPD with and without comorbid PTSD, additionally implementing comparisons with a clinical control group composed of MDD/DYS individuals (also with and without comorbid PTSD). Different effects of PTSD comorbidity were found in the BPD and MDD/DYS groups. As hypothesized, for the negative affect subtypes of fear and sadness, the BPD+PTSD participants exhibited greater instability than the BPD-only participants. Further, the BPD+PTSD participants exhibited the greatest instability of all subgroups. Our results, however, suggest that PTSD comorbidity does not tend to be associated with significantly greater (or lesser) instability of aggregate negative affect (NA), hostility, or aggregate positive affect (PA). In contrast to our finding for the BPD group, the MDD/DYS+PTSD participants in the present study exhibited significantly *less* instability of fear and sadness than the MDD/DYS-only participants. As in the BPD group, PTSD comorbidity did not show a significant effect on instability of NA, hostility, or PA in our MDD/DYS clinical control group. Thus, our findings join those prior findings (e.g., Harned et al., 2010; Pagura et al., 2010) that have suggested an important impact of BPD-PTSD comorbidity on a core feature or features of one or both of these disorders, as opposed to finding only an increase in general impairments. Additionally, our findings present evidence of disorder-specific patterns of emotion dysregulation—namely, a disparate effect of lifetime PTSD comorbidity on instability of fear and sadness across BPD and MDD/DYS outpatient samples.

Looking specifically at which types of affect instability varied across which diagnostic groups and subgroups in the present study revealed several interesting patterns. The negative affects of sadness and fear, but not hostility, showed greater instability for BPD participants with comorbid PTSD than those with BPD only. Thus, instability of negative affect, a core feature of BPD, was in fact affected by PTSD comorbidity, but in a circumscribed, affect-specific manner. This affect-specific finding makes sense from the perspective that fear and sadness are core affective features of PTSD, whereas hostility, more frequently associated with BPD, is not. Moreover, this finding echoed the findings reported by Bolton, Mueser, and Rosenberg (2006) and supported our tentative hypothesis that PTSD comorbidity in the BPD group might not affect instability of hostility as much or in the same way as it would affect instability of sadness and fear. Still, the alternative finding (i.e., *increased* instability of hostility in the BPD+PTSD group as compared to the BPD-only group) arguably would

⁶Supplementary results, including figures, are available in full upon request (i.e., by contacting the corresponding author).

have been plausible, given that outbursts of hostility in response to idiopathic trauma cues are considered to be among the hyperarousal symptoms characteristic of PTSD (e.g., see Vrana, Hughes, Dennis, Calhoun, & Beckham, 2009). Future investigation of this issue may benefit from the use of larger and more diverse samples to allow for greater generalizability and for the examination of potential moderators.

Although our null findings for both hostility and NA overall warrant replication in larger and more diverse samples before firm conclusions should be made, the fact that our findings followed the above-described circumscribed pattern points us cautiously in the direction of a more clear understanding of BPD-PTSD comorbidity in daily life. For example, from a treatment perspective, when discussing instability of negative affect with comorbid BPD +PTSD patients, it may be more efficient and effective to focus on examples involving fear and sadness rather than grouping negative affect types together or focusing on hostility.

Among the MDD/DYS participants in the present study, PTSD comorbidity was not associated with significantly greater instability in any affect type; however, this comorbidity was associated with *lower* levels of instability for sadness and fear. Although the small size of the MDD/DYS-PTSD subgroup (n=12) necessitates caution in interpretation, the contrast between these findings for the MDD/DYS group and those described above for the BPD group suggests a fundamental difference in the impact of PTSD comorbidity on the presentation of BPD versus MDD/DYS. It is not that PTSD comorbidity elevates the instability of sadness and fear in any disorder that already features dysregulation of negative affect. Rather, this impact seems to characterize the interaction, or combination, of BPD and PTSD. And it is not that PTSD comorbidity elevates all varieties of negative affect instability in BPD. Rather, this elevation appears to be specific to the affects of sadness and fear.

Whereas Santangelo et al. (2014), found a lack of specificity of AI in BPD and no impact of PTSD comorbidity on AI in BPD, the present study found significant differences in negative affect instability across the BPD and psychiatric comparison group (in this case MDD/DYS) and did find an impact of comorbid PTSD on AI in BPD, which in turn, reflected another difference between the BPD and psychiatric comparison group. Differences in study design and sample composition may have contributed to these differences across the two sets of findings. Specifically, Santangelo et al. administered assessments every 15 minutes over a 24-hour period. In contrast, by implementing much more sparse data sampling over a longer period of time (28 days), the present study aimed to achieve (a) saturation of sampling across typical daily life contexts and events and (b) minimal reactance to assessment. In addition, whereas Santangelo et al. included both inpatient and outpatient participants, the present study included only outpatients living in their own typical, daily environments.

We also examined the characteristics of traumatic experiences across subgroups in the present study to address the potential alternative explanation for differences in AI based on differences in specific types of traumatic experience (an alternative explanation set forth and ruled out by Santangelo et al. in their sample). Unfortunately, no trauma inventories were administered to participants in the original study, meaning that data regarding trauma type and timing for use in the present study had to be gleaned from interviewers' notes on the

PTSD section of the SCID-I interview. Despite this limitation, some notable aspects of the traumatic experience data were discernible: The traumatic experiences endorsed as index events for PTSD diagnoses, across groups, tended to be those considered “highly assaultive.” The most common type of index event in both the BPD+PTSD and MDD/DYS+PTSD subgroups was childhood sexual abuse/assault (CSA), making up approximately 53% and 42% of the index events in each subgroup, respectively. In the BPD+PTSD subgroup, close to 94% of reported index events were of a direct and assaultive nature. Those few events that did not fit that profile in the BPD+PTSD subgroup were, nonetheless, of a traumatic nature (e.g., loss of one’s baby, being badly injured in a car wreck, witnessing one’s own parent being murdered). Seventy-five percent of the index events reported in the MDD/DYS+PTSD subgroup were direct and assaultive; the three that were not involved witnessing and/or being involved in bad accidents. In many cases where childhood abuse was indicated as the index event, multiple subsequent traumatic events, often reaching into adulthood, were also recorded by the interviewer. Given the available information, the proportion of different types of index events and time elapsed since events appeared more similar than different across the BPD+PTSD and MDD/DYS+PTSD subgroups. Moreover, supplementary analyses predicting AI from a history of physical assault, sexual assault, or assault in general (with and without Group [BPD versus MDD/DYS] in the models) indicated that none of these classifications of assault significantly predicted instability of sadness, fear, overall NA, or PA in this sample. Therefore, difference in trauma profiles does not appear to be a viable alternative explanation for the differences that we found in AI across subgroups. However, collection of more thorough and detailed data regarding parameters and timing of traumatic experiences in future studies would improve our ability to rule out this alternative explanation.

Together with other related findings, our results can elucidate important aspects of the BPD-PTSD comorbidity. For example, research by Skodol and colleagues indicated that comorbidity on Axis I is associated with BPD stability (Skodol et al., 2002a), and presence of PD at intake predicts poor short- and long-term outcome of an Axis I disorder, even among patients matched on Axis I symptom severity at intake (Skodol et al., 2002b). Given that emotion dysregulation is seen as a potential driving force behind other characteristic symptoms of BPD (e.g., Crowell, Beauchaine, & Linehan, 2009; Glenn & Klonsky, 2009; Linehan, 1993), the exacerbation of instability of sadness and fear that accompanies PTSD comorbidity for those with BPD may be one of the mechanisms facilitating maintenance of full BPD diagnosis. And, given the finding from Marshall-Berenz et al. (2011) that the PTSD symptom of re-experiencing predicted greater instability of sadness and fear in BPD individuals, the finer-grained mechanism underlying this impact of PTSD comorbidity on BPD may reside primarily in the re-experiencing symptom. As researchers piece together the phenomenological picture of this comorbidity and increasingly take a longitudinal, lifespan-developmental approach to examining personality disorders, it may be important to examine not only the etiology of this comorbidity, but also the course of this comorbidity later in life—how PTSD comorbidity impacts the course of BPD and vice versa, and specifically whether the impact of PTSD symptoms on negative affect instability may be an important mechanism, or maintenance factor, to consider.

Increasing our understanding of the phenomenology of BPD-PTSD comorbidity is, and will continue to be, critical in our progress toward more specific intervention efforts for this relatively prevalent and burdensome comorbidity. Moreover, elucidating the dynamic patterns of AI, particularly patterns specific to different disorders and comorbidities, can provide clues to both treatment and etiology, extending our understanding beyond phenomenological description of symptoms (Ebner-Priemer et al., 2009). Clinical implications based on the findings of the present study may begin to provide such clues. Tailoring our therapeutic interventions to focus on the mindfulness of and emotion regulation of sadness and fear in cases of BPD with comorbid PTSD may be an important improvement on the typical treatment approach (i.e., typical stage I Dialectical Behavior Therapy [DBT] followed by a second stage in which the symptoms of comorbid PTSD may also be addressed), in which mindfulness and emotion regulation skills are implemented more generally and more uniformly for BPD individuals with and without PTSD. At present, despite long-standing recognition of the high rates of BPD-PTSD comorbidity in clinical populations, recent development and clinical trials of a dialectical behavior therapy (DBT) prolonged exposure (PE) protocol (DBT+DBT PE) by Harned and colleagues represents the first and only implementation of an integrated, concurrent, empirically-supported treatment directly targeting both BPD and PTSD (Harned, Korslund, Foa, & Linehan, 2012; Harned, 2013). A small group of interventions accompany DBT+DBT PE in the category of treatments that have been designed and/or evaluated with regard to addressing comorbid BPD and PTSD in some way (Bohus et al., 2013; Harned, 2014). These other interventions are less integrated, directly target one disorder while indirectly improving some symptoms from the other, and/or are tailored to specific patient sub-populations (for a review, see Harned, 2014). DBT for PTSD (DBT-PTSD; Steil, Dyer, Priebe, Kleindienst, & Bohus, 2011), for example, is a modular, or 'phase-based,' inpatient variant of DBT designed for individuals with PTSD subsequent to childhood sexual abuse (CSA) that has been found to be efficacious (that is, improving CSA-related PTSD and global functioning, though not BPD symptoms) in adult women with and without co-occurring BPD (Bohus et al., 2013). With intervention research and development still in its infancy in the area of comorbid BPD +PTSD treatment, findings such as those reported in the present study may be useful in fine tuning of the efficacy and efficiency of important new treatment developments.

Several other limitations of the present study should be acknowledged. First, we did not evaluate or control for the amount or type of psychological treatment participants received during the study period. In order to assess the potential effects of such treatment factors on affect with sufficient statistical power, a larger number of participants would be required as well as a more systematic sampling design to evaluate the effects of completed treatment. Our study design was naturalistic in the sense of not selecting participants based on types or stages of treatment. Second, additional studies employing samples with different demographic characteristics are needed to determine the generalizability of our results. Our participants were predominately women and predominately of White, non-Hispanic descent. Moreover, the two main diagnostic groups studied here, BPD participants and MDD/DYS participants, differed significantly ($p < .05$) in terms of gender and previous psychiatric hospitalization. Although we did not find that our results differed as a function of gender, we did not have a sufficient number of men to adequately test this hypothesis. Given that gender

is associated with negative affect and neuroticism, it will be especially important to replicate our findings in samples with greater representation of men. Also, although the repeated measures design of our study does confer higher levels of statistical power compared to traditional cross-sectional studies, the size of some of our subgroups (especially MDD/DYS +PTSD) was small. Therefore, it will be important to replicate these findings in larger samples. Finally, further studies are needed to examine the potential for different patterns of findings across PTSD resulting from different types of traumatic events (e.g., combat trauma versus natural disaster) and across groups characterized by onset of PTSD comorbidity at different points throughout the lifespan.

Future EMA research has the potential to improve our understanding of BPD and comorbid conditions in a number of ways, including the following: by clarifying the patterns of affective instability in daily life that differentiate among BPD, related diagnostic groups, and comorbidity-defined subgroups; by improving our understanding of AI as a dynamic process; by further characterizing associations among AI and other features (e.g., suicidality) of the disorders and/or combinations of disorders in which AI occurs; and by clarifying relationships among data garnered from various assessment methodologies (e.g., EMA, traditional retrospective self-report and/or trait-based questionnaires, physiological measurements collected in the laboratory). Ongoing improvements in EMA technology stand to increase the feasibility, agility, and scope of EMA protocols. The new and still developing possibilities of streamlined incorporation of physiological measurements into EMA, for instance, present exciting new directions for empirical investigation and treatment-related monitoring (Trull & Ebner-Priemer, 2013). Given that any emotional response is a complex, multifaceted phenomenon involving not only subjective experience, but also physiological arousal and motoric behavior, the value of accessing other, much more objective streams of physiological data to complement that of realtime self-report of emotional responding warrants attention (Rosenthal et al., 2008). Specific to the aims of the present study, the ambulatory assessment of physiological responding concurrent with subjective affective reports may prove particularly helpful in clarification of the phenomenology of BPD, PTSD, and BPD-PTSD comorbidity, especially given recent evidence of both similarity and divergence in pathophysiology across these diagnostic groups (e.g., Kraus et al., 2009; Limberg et al. (2011); Rodrigues et al., 2011; Schmahl et al., 2004; Schmahl et al., 2009), along with evidence associating subjective arousal with distress in individuals with BPD (Ebner-Priemer et al., 2008). From a treatment perspective, also, the utilization of EMA methods—including advanced physiological measurements—to longitudinally examine affect dysregulation as a marker of treatment outcome may be a critical direction to pursue in the further development and evaluation of treatment protocols designed to address BPD-PTSD comorbidity.

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Figure 1.

Means of log detrended SSD of *fear* and *sadness* for BPD and MDD/DYS participants with and without PTSD. The vertical lines are 95% confidence intervals around the mean. The horizontal line inside each box reflects the subgroup median. The length of the box represents the standard error range.

Table 1Demographic Features of Currently Depressed (MDD/DYS; $n = 50$) and Borderline Personality Disorder (BPD; $n = 78$) Participants

	MDD/DYS		BPD	
	<i>n</i>	%	<i>n</i>	%
Gender				
Female	39	78.00	72	92.31
Male	11	22.00	6	7.69
Ethnicity				
African-American	4	8.00	4	5.13
Caucasian	44	88.00	67	85.90
Hispanic	2	4.00	2	2.56
Others	0	0.00	5	6.41
Marital Status				
Single	22	44.00	39	50.00
Married	13	26.00	13	16.05
Divorced	6	12.00	13	16.05
Separated	3	6.00	4	4.94
Cohabitating	5	10.00	9	11.11
Other	1	2.00	0	0.00
Annual Income				
\$0 to \$25,000	34	68.00	55	70.51
\$25,001 to \$50,000	10	20.00	12	14.81
\$50,001 to \$75,000	3	6.00	5	6.17
\$75,001 to \$100,000	1	2.00	4	4.94
above \$100,000	2	4.00	2	2.47
Current Diagnosis				
Any substance disorder	3	6.00	14	17.95
Any anxiety disorder	33	66.00	64	82.05
Any mood disorder	50	100.00	51	65.38
Any eating disorder	2	4.00	19	24.36
Lifetime Diagnosis				
Any substance disorder	25	50.00	51	65.38

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	BPD		MDD/DYS	
	<i>n</i>	%	<i>n</i>	%
Any anxiety disorder	35	71.43	71	91.03
Any mood disorder	50	100.00	78	100.00
Any eating disorder	5	10.00	29	37.18

Table 2

Detrended Mean Squared Successive Difference (MSSD) of Affect Scores for Borderline Personality Disorder (BPD) and Currently Depressed (MDD/DYS) Participants with/without

Detrended MSSD	BPD (n = 78)		MDD/DYS (n = 50)	
	No PTSD (n = 45)		No PTSD (n = 38)	
	PTSD (n = 33)	All BPD	PTSD (n = 12)	All MDD
	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>	<i>M (SD)</i>
Negative Affect	0.27(0.26)	0.24(0.27)	0.14(0.14)	0.20(0.18)
Fear	0.37(0.41)	0.30(0.34)	0.14(0.12)	0.27(0.27)
Hostility	0.41(0.38)	0.40(0.42)	0.35(0.56)	0.27(0.28)
Sadness	0.54(0.41)	0.44(0.39)	0.25(0.21)	0.46(0.37)
Positive Affect	0.47(0.34)	0.47(0.36)	0.49(0.65)	0.41(0.32)
				0.43(0.42)

Table 3

Estimates of Effects on Instability Scores for Each Affect and Tests of Absolute Differences between Grouping Variables

<i>Effects</i>	Negative Affect		Sadness		Fear		Hostility		Positive Affect	
	Estimate		Estimate		Estimate		Estimate		Estimate	
Group (BPD vs. MDD/DYS), γ_{01}	0.289		-0.232		-0.079		0.296		0.078	
PTSD, γ_{02}	0.032		-0.647*		-0.661*		0.125		0.053	
Group*PTSD, γ_{03}	--		1.031**		1.079**		--		--	
<i>Absolute Difference Scores</i>										
Group Difference (BPD vs. MDD/DYS)	0.289		0.284		0.461*		0.296		0.078	
PTSD Diagnosis Difference	0.032		-0.131		-0.121		0.125		0.053	
PTSD Diagnosis Difference (MDD/DYS)	--		-0.647*		-0.661*		--		--	
PTSD Diagnosis Difference (BPD)	--		0.384 [†]		0.418*		--		--	

[†]
 $p < 0.10$.*
 $p < 0.05$.**
 $p < 0.01$.

Note. Interaction effects and contrasts are not reported for the models predicting instability of Negative Affect, Hostility, and Positive Affect; the models including only main effects showed better fit for these predictions.