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Is borderline personality disorder really an emotion dysregulation disorder and, if so, how?: A comprehensive experimental paradigm

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Abstract

Background: Leading theories suggest that borderline personality disorder (BPD) is an emotion dysregulation disorder involving lower basal vagal tone, higher baseline emotion, heightened emotional reactivity, delayed emotional recovery, and emotion regulation deficits. However, the literature to date lacks a unifying paradigm that tests all of the main emotion dysregulation components and comprehensively examines whether BPD is an emotion dysregulation disorder and, if so, in what ways. The present study addressed empirical gaps with a unified paradigm that assessed whether BPD is characterized by five leading emotion dysregulation components compared to generalized anxiety disorder (GAD) and healthy control (HC) groups.

Methods: Emotion was assessed across self-report, sympathetic, and parasympathetic indices. Participants with BPD, GAD, and HCs (N=120) first underwent baseline periods assessing *basal vagal tone* and *baseline emotional intensity*, followed by rejection-themed stressors assessing *emotional reactivity*. Participants then either reacted normally to assess *emotional recovery* or attempted to decrease emotion using mindfulness or distraction to assess *emotion regulation implementation deficits*.

Results: Individuals with BPD and GAD exhibited higher self-reported and sympathetic baseline emotion compared to HCs. The BPD group also exhibited self-reported emotion regulation deficits using distraction only compared to the GAD group.

Conclusions: There is minimal support for several emotion dysregulation components in BPD, and some components that are present appear to be pervasive across high emotion dysregulation groups rather than specific to BPD. However, BPD may be characterized by problems disengaging from emotion using distraction.

Keywords: Borderline personality disorder; emotion dysregulation; emotion regulation; vagal tone

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Is borderline personality disorder really an emotion dysregulation disorder and, if so, how?: A comprehensive experimental paradigm

Linehan's (1993) seminal Biosocial theory purports that emotion dysregulation (ED), involving biological vulnerabilities to disrupted emotion and emotion regulation deficits (i.e., difficulties altering emotion; Gross & Thompson, 2007), is central to BPD. Linehan (1993) postulated three components of the former: heightened sensitivity (i.e., lower threshold for emotion), heightened reactivity (i.e., larger increase in emotion post-provocation), and delayed recovery (i.e., slower decreases in emotion post-provocation; Linehan, 1993). Research testing this theory is mixed, but methodological limitations obfuscate clear conclusions about whether BPD is characterized by ED.

Antecedent-Based Emotion Dysregulation

Basal vagal tone. Emotion processes occur before (antecedent-based) or after (response-based) provocation (Gross, 1998). Although Linehan's (1993) ED components are response-based, subsequent studies suggest that BPD may be characterized by antecedent-based ED such as low basal vagal tone. Basal vagal tone is indexed by baseline respiratory sinus arrhythmia (RSA), which reflects variations in heart rate with respiration and represents the interaction of two efferent pathways from the parasympathetic nerve to the heart on cardiac activity (Porges, Doussard-Roosevelt, & Maita, 1994). Basal vagal tone theoretically indicates greater vulnerability to intense negative emotion whereas *increases* in vagal tone reflect increases in parasympathetic activation and consequential *decreases* in reactivity (Beauchaine, 2001; Porges et al., 1994). Although there are exceptions (Austin, Rinolio, & Porges, 2007; Gratz, Richmond, Dixon-Gordon, Chapman, & Tull, 2019), several studies and a meta-analysis evince lower baseline RSA in BPD compared to healthy control (HC) and clinical groups (e.g., Koenig,

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Kemp, Feeling, Thayer, & Kaess, 2016; Kuo & Linehan, 2009; Kuo, Fitzpatrick, Metcalfe, & McMain, 2016; Weinberg, Klonsky, & Hajcak, 2009). However, research examining these processes in BPD is limited by inconsistent physiological recording procedures (Carr, Maarten de Vos, & Saunders, 2017) and is in need of standardization.

Baseline emotion. Literature also demonstrates heightened baseline emotion in BPD across self-reported and sympathetic indices compared to clinical and healthy groups (e.g., Ebner-Priemer et al., 2007; Elices et al., 2012; Feliu-Soler et al., 2013; Gratz, Rosenthal, Tull, Lejuez, & Gunderson, 2010; Köhling et al., 2016; Kuo & Linehan, 2009; Kuo et al., 2016; Scott, Levy, & Granger, 2013). However, some findings suggest that physiological indices do not distinguish BPD from other clinical groups (e.g., Kuo & Linehan, 2009), suggesting that BPD may be uniquely characterized by self-reported, but not physiological, antecedent-based ED.

Response-Based Emotion

Of the response-based emotion processes specified in Linehan's (1993) model, emotional reactivity, recovery, and regulation have received the most empirical attention.

Emotional reactivity. Unlike with antecedent-based emotion, reactivity research that examines the extent to which emotion increases in response to a stressor from baseline in BPD is mixed. Some studies show heightened self-reported reactivity in BPD (e.g., Chapman, Dixon-Gordon, Butler, & Walters, 2015; Dixon-Gordon, Gratz, & Tull, 2013; Dixon-Gordon, Yiu, & Chapman, 2013; Gratz, Richmond, Dixon-Gordon, Chapman, & Tull, 2019; Rosenthal et al., 2016), while others do not (e.g., Elices et al., 2012; Evans, Howard, Dudas, Denman, & Dunn, 2013; Feliu-Soler et al., 2013; Sansone, Wiederman, Hatic, & Flath, 2010; Scott et al., 2013). Some suggest heightened sympathetic and parasympathetic reactivity in BPD (e.g., Austin et al., 2007; Dixon-Gordon et al., 2013a, 2013b; Ebner-Priemer et al., 2004; Rosenthal et al., 2016)

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while others either do not (e.g., Baschnagel, Coffey, Hawk, Schumacher, & Holloman, 2013; Chapman et al., 2015; Dixon-Gordon et al., 2015; Ebner-Priemer et al., 2005; Gratz et al., 2019; Kuo & Linehan, 2009; Kuo et al., 2016), or *lower* sympathetic reactivity (e.g., Herpertz, Kunert, Schwenger, &. Sass, 1999; Herpertz et al., 2000; Pfaltz et al., 2015). Studies examining neural activity and other emotion indices (e.g., eyeblink, electromyography) are similarly mixed (e.g., Baschnagel et al., 2013; Baskin-Sommers et al., 2012; Elices et al., 2012; Hazlett et al., 2012; Pfaltz et al., 2015; Smoski et al., 2011; Rosenthal et al., 2016; Scott et al., 2013; Hepertz et al., 2019, 2000). Taken together, emotional reactivity is highly discordant across self-report and physiological indices, leading some to suggest that physiological emotional reactivity may not characterize BPD to the same extent as self-report (Rosenthal et al., 2008).

Emotional recovery. The few studies that have investigated emotional recovery in BPD produce similarly mixed findings across indices. Although studies suggest that BPD or high BPD feature groups exhibit delayed self-reported emotional recovery (Chapman et al., 2015; Ebner-Priemer et al., 2015), several other studies do not (Chapman et al., 2015; Ebner-Priemer et al., 2015; Fitzpatrick & Kuo, 2015; Gratz et al., 2010; Jacob et al., 2009; Scheel et al., 2013; Weinberg et al., 2009). Similarly, two studies show delayed recovery in BPD heart rate or parasympathetic functioning (Chapman et al., 2015; Fitzpatrick & Kuo, 2015), but several suggest comparable sympathetic and parasympathetic recovery in BPD and control groups (Chapman et al., 2015; Fitzpatrick & Kuo, 2015; Scott et al., 2013; Weinberg et al., 2009).

Emotion regulation implementation. Finally, although individuals with BPD *report* greater difficulties with emotion regulation compared to HCs (Daros et al., 2018), studies suggest that they can suppress emotions and reappraise emotional content following training in these strategies to a similar or better extant than healthy groups across neural, self-report, and

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parasympathetic indices (e.g., Baczkowski et al., 2017; Chapman, Rosenthal, & Leung, 2009; Krause-Utz, Walther, Lis, Schmahl, & Bohus, 2018; Lang et al., 2012; Marissen et al., 2010; Ruocco, Meaglia, Ayaz, & Chute, 2010; Schulze et al., 2011). Individuals with BPD can also implement BPD-relevant treatment strategies such as mindfulness and distraction when trained to the same extent as healthy groups across self-report and physiological indices (Chapman et al., 2009; Kuo, et al., 2016). Notably, mindfulness and distraction decrease emotion through opposite mechanisms, either by directing attention *towards* or *away from* it, respectively (Sheppes & Meiran, 2008). Therefore, individuals with BPD do not exhibit deficits in implementing emotion regulation strategies that they are trained in (i.e., emotion regulation implementation) across self-reported and psychophysiological domains, regardless of mechanism of action, although other emotion regulation forms may be deficient in BPD (e.g., *perceived* effectiveness of strategies and downregulation of elevated baseline emotion; Daros et al., 2018; Kuo et al., 2016; Southward & Cheavens, 2018).

Is BPD an Emotion Dysregulation Disorder?

Taken together, studies suggest that BPD may be an ED disorder with respect to antecedent-, but not response-, based emotion. However, such conclusions are obfuscated by methodological limitations. First, although ED is arguably most commonly associated with BPD, it also underpins other affective disorders such as generalized anxiety disorder (GAD; Mennin, Heimberg, Turk, & Fresco, 2005). GAD is also considered to be an ED disorder given that it is associated with heightened baseline emotion, emotional reactivity, and emotion regulation deficits across student and clinical samples (Mennin et al., 2005). However, few studies have compared ED in BPD to other high-ED clinical groups, resulting in minimal clarity regarding whether BPD is *uniquely* characterized by ED. Second, although individuals with BPD exhibit

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heightened reactivity to BPD-relevant themes (e.g., rejection or abandonment) moreso than generic stressors (Limberg, Barnow, Freyberger, & Hamm, 2011), much of the extant research used generalized inductions and may fail to capture ED in this group. Third, most studies have not examined whether dissociation is related to outcomes and, if so, controlled for it, even though it is common in BPD (Ross, 2007) and dampens ED (Ebner-Premier et al., 2005). Fourth, existing studies measure self-reported emotion through discrete measurements periodically administered, losing variability in real-time emotion changes. Such limitations have been overcome with continuous rating dials (Ruef & Levenson, 2007), but this innovation has not been applied to BPD ED research.

Clear assessment of ED in BPD thus requires (a) a high ED clinical control group, (b) BPD-relevant inductions, (c) controlling for dissociation when necessary, (d) comprehensive assessment across emotion domains, and (e) continuous measurement of self-reported emotion. No studies have met all of these criteria under one experimental umbrella, which prohibits probing ED with granularity and drawing firm conclusions about which components characterize BPD when study-based variability is constant.

Accordingly, the present study examined whether baseline RSA, baseline emotion, reactivity, recovery, and emotion regulation implementation vary in BPD compared to HCs and a GAD control group across continuous self-report and physiological indices, using BPD-relevant emotion inductions, and controlling for dissociation where necessary. We examined two emotion regulation strategies with distinct mechanisms, mindfulness and distraction (e.g., Linehan, 1993; 2015), to clarify if emotion regulation implementation deficits in BPD are specific to, or pervasive across, strategy types. We hypothesized that individuals with BPD would exhibit lower baseline RSA and higher baseline emotion relative to HCs. Consistent with Linehan (1993), we

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also hypothesized that individuals with BPD would exhibit heightened reactivity, delayed recovery, and greater emotion regulation implementation deficits in both strategies, relative to HCs. We considered comparisons with the GAD group exploratory.

Methods

Participants

Forty age and sex-matched individuals with BPD, GAD, and HCs (N=120),18-60 years-old were recruited online and via fliers. Consistent with other BPD studies (e.g., Elices et al., 2012; Kuo & Linehan, 2009), exclusion criteria were: medication that influences psychophysiology (e.g., beta-blockers, psychiatric medications other than selective serotonin reuptake inhibitors (Licht et al., 2010)); and current comorbid medical (e.g., epilepsy, heart/respiratory conditions) or psychiatric (i.e., alcohol or substance use dependence; bipolar I disorder, severe psychotic disorder) conditions that interfere with psychophysiology or task completion. To promote some distinction between BPD and control groups, prospective participants in the GAD and HC groups were excluded if they endorsed 4+ BPD criteria, or self-harm/suicidality or impulsivity BPD criteria. HCs were also excluded if they had any current psychiatric diagnoses or were taking any current psychiatric medications. Demographic and diagnostic information across groups is in Table 1 and 2.

Measures

The Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders-IV-TR (SCID-IV-TR; First, Spitzer, Gibbon, & Williams, 1995) was administered to assess for GAD and diagnostic comorbidities. The International Personality Disorders Examination-BPD Module (IPDE-BPD; Loranger et al., 1994) was used to assess BPD. Undergraduate and masters-level assessors trained to reliability against a gold-standard assessor

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and supervised by a licensed clinical psychologist administered all interviews, with prevalence-adjusted bias-adjusted kappas (PABAKs; Byrt, Bishop, & Carlin, 1993) with the gold-standard assessor ranging from .67 to 1.00 across modules (average PABAK for SCID-IV-TR=.97 for IPDE-BPE=.95).

The Dissociative State Scale (DSS; Stiglmayr, Shapiro, Stieglitz, Limberger, & Bohus, 2001) is a 21-item scale that was used to measure state dissociation, with higher scores reflecting higher dissociation (α ranged from .92 to .93 across baseline and trials).

To characterize the sample, the Borderline Symptom List-23 (BSL-23; Bohus et al., 2007) was used to measure BPD severity (α=.97), and the Depression, Anxiety, and Stress Scales (DASS; Lovibond & Lovibond, 1995) was used to measure depression, anxiety, and stress severity (subscale α=.92 to .97).

Emotion indices. Participants were asked to keep one hand on a rating dial throughout the experiment and adjust it to reflect shifts in *self-reported emotional intensity*. The dial ranges from 0 (very negative) to 9 (very positive) and allowed for a continuous, moment-by-moment, assessment of negative emotion, which was decomposed into 30-second epochs.[1]

Participants also rated the following experiences using visual analogue scale from 0 (not at all) to 100 (very) before and after each emotion induction: afraid, alone/lonely, angry, anxious, ashamed, disgusted, empty, guilty, hopeless, rejected, sad, and tense. Ratings for these emotions were summed together to yield a negativity composite used in manipulation check analyses. Following the implementation of mindfulness and distraction, participants were asked to rate how effective the strategy was in affecting their emotions from 0 (made much worse or much

[1] The emotion induction stories ranged from two minutes and four seconds to two minutes and nine seconds. Each story involved four epochs, with the first three lasting 30-seconds and the fourth ranging from 34 to 39 seconds.

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more intense) to 100 (made much better or much less intense).

Skin conductance responses (SCRs) were examined as a *sympathetic* index of emotion. Electrodes were placed on the medial phalanges of the middle and index finger of participant's non-dominant hand (Fowles et al., 1981). SCRs were digitized using low (35 Hz) and high (.05 Hz) pass filters at 1000 samples per second and a gain of 1000. Mindware Technologies EDA 2.40 program was used to process the data. A rolling filter detected and edited artifacts. SCR was indexed as the number of responses exceeding 0.05 μS per 30-second epoch.

Respiratory sinus arrhythmia (RSA) was examined as an index of basal vagal tone and *parasympathetic* emotion. A two-electrode electrocardiography configuration (i.e., one electrode placed 3 to 4 inches beneath the right clavicle, and the other placed beneath the left rib cage) with a bioimpedance module for grounding was used, with a respiratory band around the chest and a sampling frequency of 1000 samples/sec. Mindware Technologies HRV 2.33 software was used to calculate R-R intervals across 30-second epochs. Mindware software detected R-spikes using the Shannon Energy Envelope technique (Manikandan & Soman, 2012), and all data was further visually inspected and double-scored to scan for artifacts and correct R-spike identification. Following detection of R-spikes, interbeat intervals (IBIs) for subsequent beats were computed by subtracting the time at which they occurred from one another. Spectral analysis decomposed the electrocardiogram into three frequency ranges, applying validated algorithm to calculate spectral densities and retaining RSA signals within the frequency of .12 Hz to .4 Hz. All measurements were taken with participants sitting upright with their feet placed on the floor. Data collected during segments of the experiment wherein participants were clearly not complying with instructions, or the electrocardiogram was not detectable due to noise, were removed from analyses.

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Emotion induction. We developed three, 2-minute rejection-themed scripts with standardized numbers of thoughts and physiological sensations described in them across scripts, and standardized intensity of emotional words, across scripts (see open science framework for more information on script development).[2] Pilot data with 55 undergraduates demonstrated scripts elicit equivalent amounts of pre- to post-self-reported general negative emotion, $F(2,53)=1.62$, $p=.21$.

Procedures

Procedures were approved by institutional research boards. Interested participants were pre-screened and, if eligible, invited for an in-person eligibility assessment (SCID-IV-TR, IPDE). Eligible participants were invited to return for the experiment and instructed to avoid stimulants (e.g., caffeine, tobacco) on the experiment day. The time of experiments varied throughout the day and evening. Physiological equipment was attached and participants were instructed on rating dial use. Participants completed an initial a 10-minute resting baseline with no stimuli presented. To ensure stability, only the last 5 minutes of this baseline was analyzed (Jennings, Kamarck, Stewart, Eddy, & Johnson, 1992).

See Figure 1 for an illustration of the experiment. Following baseline, the laboratory task was a within-subjects design involving three trials (REACT, MINDFULNESS, DISTRACTION), each of which involved four phases. In Phase 1 *(instruction phase)* of the first trial (REACT condition), participants were instructed to "act as they normally would" (Kuo et al., 2016) when prompted. This condition allowed for assessment of post-induction recovery

[2] Additional information regarding: 1) the development and piloting of the mood induction scripts; 2) the means, standard deviations, and effect sizes for the mood inductions; and 3) the scripts themselves, are available on Open Science Framework (OSF) at the following Project Page: https://osf.io/cqaj2/

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without instructed emotion regulation implementation. In Phase 1 of the second and third trials, participants were trained in the MINDFULNESS or DISTRACTION conditions. For MINDFULNESS, participants were instructed to nonjudgmentally notice emotional experiences without rejection or amplification (Kuo et al., 2016; Erisman & Roemer, 2010). For DISTRACT, participants were instructed to distract themselves from induction content by thinking of something neutral (Kuo et al., 2016; Sheppes & Meiran, 2008) to avoid confounding distraction with positive emotion inductions by attending to positively-valenced thoughts. Participants verbally repeated instructions to ensure understanding, with clarification provided as needed.

Across all trials, during Phase 2 (*trial-baseline phase*), participants sat quietly for five minutes while baseline measurements were taken again. Next, in Phase 3 (*induction phase*), participants listened to an induction script and were asked to imagine themselves in the scenario depicted for two minutes (i.e., four 30-second epochs). Finally, in Phase 4 (*regulation phase*), an on-screen prompt cued participants to engage in the emotion regulation strategy. Participants implemented react, mindfulness, or distraction for 2.5 minutes (i.e., five 30-second epochs), after which the DSS was administered. Rating dial and physiological recordings were continuous. Order of strategy presentation (mindfulness and distract), and pairings of strategy and emotion induction scripts were counterbalanced. Participants rated the extent to which they attempted to use mindfulness and distraction when instructed, ranging from 0% to 100% of the time.

Data Analytic Strategy

Generalized Estimating Equations (GEE; Burton, Gurrin, & Sly, 1998; Hubbard et al., 2010) was used to analyze data with SPSS version 26. GEE is a semi-parametric extension of generalized linear modeling and maximizes power by examining outcome variables over continuous time courses and retaining information from participants with missing data.

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Autoregressive, exchangeable, and unstructured covariance structures were examined and the one with the lowest Quasilikelihood under the Independence Model Criterion (QIC) value was selected. A negative binomial distribution was specified for SCR data which is typically an overdispersed count variable (Atkins & Gallop, 2007). Continuous predictors were median-centered.

Analyses were run separately for each outcome (i.e., baseline emotional intensity/biological vulnerability, reactivity, recovery, mindfulness, distraction) and index nested within them (i.e., self-report, SCR, RSA). *For baseline emotional intensity/biological vulnerability*, emotion across 30-second epochs for the last 5-minutes of the initial baseline was the outcome, with group (BPD, GAD, HC) as a between-subjects factor. *For reactivity*, emotional intensity for each 30-second epoch across the three induction phases (four epochs per phase=12 repeated measures) was the outcome. For *recovery,* emotion across 30-second epochs during the regulation phase of the REACT condition was the outcome. In both analyses, group was a between-subjects factor, with either emotion from the trial-baselines preceding the inductions (reactivity analyses) or emotion from the induction preceding the REACT phase (recovery analyses) entered as a within-subjects covariate. In both, epoch was entered as a within-subjects predictor and group×epoch interactions were entered to test whether groups varied in change in emotion over the induction or regulation phase of the REACT condition, respectively. For *mindfulness/distraction analyses,* emotion across the 30-second epochs during either the regulation phase of the MINDFULNESS and DISTRACT conditions were the outcomes. Group was entered as between-subjects factor, emotion from the induction phase that preceded it was entered as within-subjects covariate, and epoch was entered as a within-subjects predictor. Group×epoch interactions were entered to test whether groups varied in change in

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emotion over the emotion regulation implementation phase. Univariate analyses of variance were run to examine group differences in perceived effectiveness of distraction and mindfulness. In line with concerns regarding potential dilution of results due to the application of multiple tests corrections, they were not employed (e.g., O'Keefe, 2003; Rothman, 1990).

Identification of Potential Covariates. Higher dissociation was significantly or trending towards significantly correlated with self-reported negative emotion across several experiment components (significant or trending *r*s range from -.22 to -.17, *p*s from <.02 to .06), and negative emotion from the visual analogue scales (all $ps \leq .001$). Dissociation was not significantly correlated with physiological indices during any phases across conditions (*p*s range from .10 to .97), or the perceived effectiveness of emotion regulation strategies (*p*s range from .14 to .15). We therefore entered dissociation from each trial into the model as a within-subjects covariate for analyses examining self-reported emotion processes only.

Comparisons of the number of current comorbid psychological disorders within each group suggested that the BPD group had higher rates of alcohol abuse and dysthymic disorder, and lower rates of GAD (See Table 2). Groups did not significantly differ in age (p=.73), sex, gender identity, or race/ethnicity (*p*s range from .17 to .92). However, there were significant differences across Race/Ethnicity groups collapsed into three categories: White/Caucasian/European Origin, Asian, and Other, $\chi^2(1)=11.97$, p=.02. We therefore included current comorbid alcohol abuse and dysthymic disorder, and Race/Ethnicity (with three categories), into all primary study analyses as between-subjects covariates.

Results[3]

[3] Primary study analyses were re-run with the removal of categorical comorbidity covariates (i.e., alcohol abuse, dysthymic disorder) and the inclusion of continuous depression and anxiety subscale scores from the DASS. Significant main effects and interactions relevant to study

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Mean BPD severity in the BPD group was within a standard deviation of that of treatment-seeking inpatient samples with BPD (Kröger et al., 2013), but mean BPD severity within the GAD and HC groups were not. See Table 1 for information regarding specific group differences in severity measures.

Manipulation Check

Participant means and standard deviations for outcomes and dissociation are in Table 3. GEE analyses examining change in negative emotion across epochs from trial-baseline to induction within each group revealed main effects of phase (trial-baseline to induction) for self-reported negative emotion, which increased in BPD (B=-1.26, *SE*=.16), GAD (B=-1.33, *SE*=.18, and HC (B=-.73, *SE*=.16) groups (all *p*s > .001). However, although there was a main effect of phase for SCRs for HCs (B=.23, *SE*=.09), χ^2 (1)=6.42, *p*=.01, this effect was not significant in BPD or GAD groups *(p*s=.22). As well, although there was a main effect of phase for RSA for the GAD group (B=-.20, *SE*=.03), χ^2 (1)=32.45, *p*<.001, this effect was not significant in BPD or HC groups (*ps*=.64 and .35, respectively). These findings indicate that the induction induced negative emotion across self-reported domains, but was variable in its impact on physiological ones. Groups did not differ with respect to the extent to which they attempted to implement mindfulness, *F*(2, 116)=.78, *p*=.46, or distraction, *F*(2, 114)=1.81, *p*=.17.

Antecedent-Based Emotion

Primary study results are in Table 4. Group predicted self-reported baseline emotional intensity, wherein BPD (B=-.98, *SE*=.33) and GAD (marginally significant; B=-.57, *SE* =.29)

hypotheses remained significant or marginally significant (*p* = .05) with two exceptions: the main effect of group predicting self-reported emotion during the baseline and induction periods became non-significant (*p* = .46 and .13, respectively). Non-significant main effects and interactions relevant to study hypotheses remained non-significant.

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groups exhibited more self-reported negative emotion at baseline than HCs, $\chi^2(1)=8.87$, $p=.003$, and $\chi^2(1)=4.00$, $p=.05$, respectively, but did not differ from each other, $\chi^2(1)=2.24$, $p=.13$. Group also predicted baseline SCR, wherein BPD (B=.56, *SE*=.23) and GAD (B=.64, *SE* =.24) groups exhibited more self-reported negative emotion at baseline than HCs, $\chi^2(1)=5.79$, $p=.02$, and $\chi^2(1)= 6.89$, $p=.01$, respectively, but did not differ from each other, $\chi^2(1)=.09$, $p=.76$. There was not a main effect of group predicting basal RSA.

Emotional Reactivity/Recovery

There was a significant main effect of group for self-reported reactivity such that the HCs exhibited less self-reported negative emotion across the induction phase than the BPD group (B=-.64, *SE*=.24), $\chi^2(1)=7.08$, $p=.01$, but not the GAD group, $\chi^2(1)=3.14$, $p=.08$. The BPD and GAD group did not differ from each other, $\chi^2(1)=1.73$, $p=.19$. Group did not predict SCR or RSA reactivity, or any recovery indices, and group×epoch interactions did not predict any reactivity or recovery index.

Emotion Regulation Implementation. A group×epoch interaction predicted self-reported emotion in the distraction condition. There was less decrease in negative emotion in BPD relative to the GAD group (B=-.22, *SE*=.07), $\chi^2(1)=8.44$, $p=.004$. However, there were no differences between change in emotion in the HC and BPD, $\chi^2(1)=.83$, $p=.36$, or GAD, $\chi^2(1)=2.92$, $p=.09$, groups. There was also a group×epoch interaction predicting SCR in the mindfulness condition. There was less decrease in SCR in the GAD group (B=.21, *SE*=.08), $\chi^2(1)=6.96$, $p=.01$, but not in the BPD group, $\chi^2(1)=2.55$, $p=.11$, compared to HCs. However, there were no differences in change in SCR in the BPD group compared to GAD group, $\chi^2(1)=1.21$ $p=.27$. There were no other group×epoch interactions across indices and conditions.

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Perceived effectiveness. Univariate analyses of variance indicated that there were not significant main effects of group predicting perceived effectiveness of mindfulness, $F(2,110)=.42$, $p=.66$, or distraction, $F(2,108)=.97$,$p=.38$.

Discussion

This study aimed to clarify whether BPD is uniquely or generally characterized by the prominent ED components by studying distinct and continuous emotion, using BPD-relevant inductions, compared to healthy *and* ED clinical control groups, after accounting for dissociation where necessary.

Antecedent-Based Emotion

In contrast to our hypotheses, no significant differences in basal vagal tone emerged between groups. Most people with BPD take psychiatric medications (Horz, Zanarini, Frankenburg, Reich & Fitzmaurice, 2010), many of which were exclusion criteria for this study, which may have eliminated higher-severity participants, obfuscating differences in low basal vagal tone across groups. However, as studies with similar inclusion criteria evince lower basal vagal tone in BPD (e.g., Kuo et al., 2016), severity may not wholly account for these null findings. As well, we found that high self-reported negative emotion and sympathetic baseline activity characterize both high ED groups relative to HCs, but these phenomena are not specific to BPD. Furthermore, post-hoc analyses that involved including continuous measures of depression and anxiety as covariates resulted in the main effect of group on self-reported baseline emotion becoming non-significant. These findings indicate that comorbid depression or anxiety severity may account for heightened self-reported baseline emotion.

Response-Based Emotion

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Emotional reactivity and recovery. Our findings further indicate discordance among reactivity indices. BPD and GAD groups exhibited higher self-reported negative emotion than the HC group during the induction phase after controlling for baseline emotion, suggesting that both of these groups experienced the inductions more intensely than HCs. However, post-hoc analyses with continuous measures of depression and anxiety, wherein the main effect of group became non-significant, suggest that these covariates may account for these differences. Further, group×epoch interactions predicting self-reported negative emotion were non-significant, indicating that the *rate of increase* during the emotion induction was comparable across groups. These findings suggest that, while individuals with BPD and GAD experience provocations with greater intensity, these findings may be due to the severity of comorbid mood and anxiety disturbances they experience, and their rate of increase following those provocations is not atypical, further suggesting that generally elevated resting emotion may be characteristic of ED disorders but not BPD specifically. Researchers have examined both the main effect of group and group×epoch interactions as indices of reactivity, and little attention has been paid to the ways in which it has been, or should be, quantified. These findings importantly indicate that greater attention to the quantification of emotional reactivity is needed as it may lead to notably distinct results.

Our self-reported and physiological recovery results join many studies in suggesting a lack differences between BPD and control groups on these outcomes (e.g., Chapman et al., 2015; Fitzpatrick & Kuo, 2015; Dixon-Gordon et al., 2015; Kuo et al., 2009). It is possible that the emotion induction scripts were not potent or generalizable enough to induce heightened reactivity or prolonged recovery in BPD, especially in the physiological domain wherein they resulted in mixed effects across groups. Indeed, heightened reactivity in BPD has been more

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typically observed in everyday interactions (Glaser et al., 2008; 2010). However, it is also possible that examining the effects of the induction within groups masked potentially significant impacts on the physiological domain due to low statistical power. Indeed, significant main effects of epoch in our primary study analyses suggest that both self-reported negative emotion and SCR increased across groups during the emotion induction, supporting its efficacy in these domains. Finally, delayed recovery may be specific to some emotions (e.g., anger) but not others, which was not examined in this study. However, past work also suggests that emotion-specific delayed recovery is evident in BPD compared to HCs but not clinical groups (Fitzpatrick & Kuo, 2015). BPD may thus not be characterized by heightened physiological reactivity, or delayed recovery, compared to other clinical groups.

Emotion regulation implementation. Partially in line with our hypothesis, the BPD group exhibited lesser reduction in self-reported negative emotion in the distraction condition compared to the GAD, but not the HC, group. Distraction requires that an individual divert attention away from emotion, which is a core emotion deficit in BPD (Winter, 2016). Selby and Joiner (2009) purport that, following an emotional evocation, individuals with BPD ruminate, which exacerbates negative emotion. Such rumination may prevent individuals with BPD from shifting attention away from emotional content. Alternatively, individuals with BPD may be comparable to others in the use of distraction, and individuals with GAD may actually be particularly strong at it. Consistently, several theories of GAD emphasize the central role of avoidance of inner experiences (e.g., Behar, DiMarco, Hekler, Mohlman, & Staples 2009), which may facilitate distraction.

Results also suggested that the GAD group was particularly deficient in mindfulness relative to HCs in decreasing sympathetic activity. The high avoidance that characterizes GAD

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may have made mindfulness, which directs attention towards such experiences, particularly challenging for this group. Alternatively, perhaps both BPD and GAD groups exhibit difficulties decreasing sympathetic activity using a strategy that directs attention towards it (i.e., mindfulness), but the BPD group was more practiced and consequently effective in this skill, given its emphasis in leading BPD treatments (i.e., DBT; Linehan, 1993; 2015). Mindfulness is also a complex skill that may require more training than distraction to effectively implement, which may mask potential deficits in this strategy in the BPD group if even HCs struggle with its use. Future research should examine history with mindfulness as a moderator of strategy effectiveness.

It is notable that BPD-specific effects for emotion regulation implementation were limited to the self-reported domain. Perhaps individuals with BPD are particularly likely to report problems distracting even if they physiologically do not experience them. However, the BPD group was not more or less likely to perceive either mindfulness or distraction as less effective than other groups, indicating that their deficits in distraction may extend beyond their reports of its efficacy. Moreover, it is notable that post-hoc analyses that involved the inclusion of continuous measures of depression and anxiety did not alter the significance of these interactions. Thus, unlike with differences in general self-reported emotion across groups, emotion regulation problems or lack thereof may not be accounted for by common comorbidities in BPD and GAD groups.

Limitations, Implications, and Future Directions

Although this work reflects a rigorous and unifying assessment of ED in BPD, several limitations warrant consideration. The BPD-relevant stories may not have been sufficiently potent to replicate daily life stressors for those with BPD, and our manipulation check suggested

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that they failed to evoke distress in the parasympathetic domain. Further, this study may have been underpowered to detect group effects, particularly interaction effects, given its modest sample size. In addition, although inclusion of a clinical control group is a strength of this study, it is also limited by the high rates of overlap in BPD and GAD groups, wherein 40% of the BPD group also had GAD, which may have obfuscated otherwise apparent differences between these two groups. Future studies should extend this work by ensuring greater divergence between clinical groups and controlling for other comorbid diagnoses that commonly occur with BPD. This may be particularly important for antisocial personality disorder as well as psychopathy, given that both are highly associated with BPD (Zanarini et al., 1998; Sprague, Javdani, Sadeh, Newman, & Verona, 2012), and "Factor 1" psychopathy is characterized by processes that may blunt emotion dysregulation processes in BPD (e.g., lack of empathy, guilt, shallow affect; Harpur, Hare, & Hakstian, 1989). Moreover, we did not collect information on the treatment history of study participants, which may confound study findings given the overlap between the emotion regulation strategies studied and their emphasis in BPD treatments (e.g., Linehan, 1993; 2015). Indeed, it is possible that the BPD group did not show more signs of emotion regulation implementation deficits with mindfulness particularly because they were more likely to be already exposed to treatments that emphasize them.

The present study also only examined one form of emotion regulation (i.e., implementation of a skill following training), and other variants of emotion regulation, such as continued elevated emotion following successful emotion regulation implementation, and emotion regulation implementation without training, may be deficient in BPD and should be examined. Finally, despite evidence that some ED components may be emotion-specific (e.g., Fitzpatrick & Kuo, 2015; Jacob et al., 2009), we did not examine this possibility.

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Despite these limitations, the present work has clinical and research implications. BPD treatments such as DBT (Linehan, 1993; 2015) focus on skills to alter ED, even though several ED processes may not be problematic in, or unique to, BPD. Our results and extant literature call for an increased attention to additional processes beyond ED such as rumination (e.g., Selby & Joiner, 2009) and interpersonal dysfunction (Fitzpatrick, Wagner, & Monson, 2019), that may uniquely characterize BPD and require targeting. Regarding ED, clinicians and researchers are advised to focus on antecedent-based emotion and deficits in disengaging from emotion as clinical targets and areas for additional research.

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Table 1

Demographic and diagnostic information across groups

	Borderline Personality Disorder	Generalized Anxiety Disorder	Healthy Control
Age (M (SD))	25.35 (6.77)	25.95 (7.20)	24.70 (6.88)
Sex			
Male	17.5%	21.1%	20%
Female	82.5%	78.9%	80%
Race/ethnicity			
White/Caucasian/European Origin	40%	34.2%	15%
Aboriginal-Canadian/First Nations/Metis/Inuit	2.5%	0%	0%
Black-Canadian/Black/Caribbean Origin	5.0%	5.3%	10%
Chinese or Chinese-Canadian	22.5%	10.5%	30%
Korean or Korean-Canadian	2.5%	0%	2.5%
Other Asian or other Asian-Canadian	12.5%	13.2%	22.5%
Mexican or Mexican-Canada	0%	0%	5.0%
Other Hispanic/Latino	0%	5.3%	5.0%
East Indian	2.5%	5.3%	0%

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Middle Eastern	0%	5.3%	5.0%
Bi-racial or multi-racial	2.5%	7.9%	2.5%
Other	10%	13.2%	2.5%
BPD Severity[a,b,c] (M(SD))	1.86(.82)	.95(.67)	.13(.16)
Depression severity[a,b,c] (M(SD))	19.85(10.30)	11.48(9.19)	1.50(2.35)
Anxiety severity[a,b] (M(SD))	12.68(8.00)	10.15(7.90)	1.13(1.36)
Stress severity [a,b,] (M(SD))	23.63(9.07)	17.35(9.83)	3.23(3.70)

Note. There were significant group differences in BPD severity, $F(2,116)=79.12$, $p<.001$, depression, $F(2,117)=51.70$, $p<.001$, anxiety, $F(2,117)=34.15$, $p<.001$, and stress, $F(2,117)=68.08$, $p<.001$. For post-hoc contrasts examining group differences in BPD, depression, or anxiety severity only: [a] Significant difference at $p < .05$ between BPD and HC group; [b] Significant difference at $p < .05$ between GAD and HC group; [c] Significant difference at $p < .05$ between BPD and GAD group

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Table 2

Common Current Comorbidities across BPD and GAD groups

Diagnosis	Borderline Personality Disorder	Generalized Anxiety Disorder	Test of group differences
Major Depressive Disorder	30%	20%	$\chi^2(1)=1.07$, $p=.30$
Dysthymic Disorder	**22.5%**	**2.5%**	**$\chi^2(1)=7.31$, $p=.01$**
Bipolar II Disorder	5%	2.5%	$\chi^2(1)=.35$, $p=.56$
Substance induced mood disorder	2.5%	0%	$\chi^2(1)=1.01$, $p=.31$
Psychotic disorder not otherwise specified	2.5%	0%	$\chi^2(1)=1.01$, $p=.31$
Alcohol abuse	**12.5%**	**0%**	**$\chi^2(1)=5.33$, $p=.02$**
Substance abuse	5%	2.5%	$\chi^2(1)=.35$, $p=.56$
Panic disorder	15%	7.5%	$\chi^2(1)=1.13$, $p=.29$
Agoraphobia	10%	7.5%	$\chi^2(1)=.16$, $p=.69$
Agoraphobia without a history of panic disorder	5%	2.5%	$\chi^2(1)=.35$, $p=.56$
Social anxiety disorder	45%	42.5%	$\chi^2(1)=.05$, $p=.82$
Specific phobia	17.5%	25%	$\chi^2(1)=.67$, $p=.41$
Obsessive compulsive disorder	32.5%	15%	$\chi^2(1)=3.38$, $p=.07$
Posttraumatic stress disorder	15%	2.5%	$\chi^2(1)=3.91$, $p=.05$
Generalized anxiety disorder	**40%**	**100%**	**$\chi^2(1)=34.29$, $p<.001$**
Anorexia nervosa	5%	2.5%	$\chi^2(1)=.35$, $p=.56$

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Bulimia nervosa	5%	0%	$\chi^2(1)=.35$, $p=.56$
Binge eating disorder	2.5%	0%	$\chi^2(1)=1.01$, $p=.31$
Eating disorder not otherwise specified	5%	2.5%	$\chi^2(1)=.35$, $p=.56$

Note. Additional information about diagnostic comorbidities presented in Fitzpatrick et al. (2020). Significant group differences are bolded.

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Table 3

Means (standard deviation) across conditions, phases, and groups

			Borderline personality disorder	Healthy controls	Generalized anxiety disorder
Dissociation					
	Initial Baseline		1.58(.23)	.36(.08)	1.04(.21)
		React	1.80(.25)	.28(.09)	.98(.17)
		Mindfulness	1.46(.22)	.40(.11)	1.13(.18)
		Distraction	1.49(.23)	.30(.10)	1.05(.21)
Self-report					
	Initial Baseline		4.38(1.22)	5.44(1.37)	4.89(.96)
	React	Trial-Baseline	4.28(1.10)	5.30(1.57)	4.52(1.42)
		Induction	3.00(1.50)	4.00(1.51)	3.05(1.67)
		Regulation	3.11(1.70)	4.36(1.51)	3.36(1.60)
	Mindfulness	Trial-Baseline	4.14(1.60)	5.13(1.20)	4.45(1.46)
		Induction	2.83(1.50)	4.03(1.79)	3.27(1.62)
		Regulation	3.23(1.98)	3.55(1.69)	3.40(1.80)
	Distraction	Trial-Baseline	4.12(1.42)	4.97(1.68)	4.32(1.51)
		Induction	2.93(1.48)	3.71(1.38)	2.98(1.53)
		Regulation	3.35(1.87)	3.59(1.66)	3.54(1.95)
Skin conductance responses					
	Initial Baseline		1.62(2.07)	.77(.82)	1.36(1.53)

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React	Trial-Baseline	1.19(.99)	1.12(1.23)	1.28(1.27)
	Induction	2.18(2.26)	1.84(1.66)	2.16(1.95)
	Regulation	1.84(1.73)	1.46(1.12)	1.84(1.59)
Mindfulness	Trial-Baseline	2.03(2.35)	1.30(1.52)	1.81(2.12)
	Induction	2.20(2.94)	1.52(1.59)	1.58(1.54)
	Regulation	1.76(1.20)	1.26(1.41)	1.76(1.54)
Distraction	Trial-Baseline	1.67(1.09)	1.42(1.26)	2.04(2.15)
	Induction	1.59(1.57)	1.49(1.63)	2.14(2.20)
	Regulation	1.87(1.76)	1.40(1.48)	1.98(1.91)
Respiratory Sinus Arrythmia (ms^2)				
Initial Baseline		6.20(1.30)	6.62(.99)	6.28(.92)
React	Trial-Baseline	6.28(1.09)	6.58(1.06)	6.36(.94)
	Induction	6.29(1.16)	6.48(1.05)	6.24(.97)
	Regulation	6.38(1.05)	6.57(1.04)	6.49(.99)
Mindfulness	Trial-Baseline	6.24(1.18)	6.51(.90)	6.31(.93)
	Induction	6.21(1.29)	6.44(.96)	6.27(1.04)
	Regulation	6.46(1.17)	6.55(.94)	6.61(.99)
Distraction	Trial-Baseline	6.28(1.08)	6.45(1.02)	6.23(.94)
	Induction	6.29(1.27)	6.35(.96)	6.20(1.11)
	Regulation	6.25(1.12)	6.59(1.04)	6.31(.96)

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Table 4
Primary generalized estimating equations analyses

	Self-report					Skin conductance responses					Respiratory sinus arrythmia				
	B	*SE*	χ^2	df	*p*	B	*SE*	χ^2	df	*p*	B	*SE*	χ^2	df	*p*
Baseline Emotion															
Intercept	5.40	.27	290.69	1	<.001	-.62	.24	6.55	1	.01	6.56	.23	470.46	1	<.001
Race/Ethnicity			2.58	2	.28			3.71	2	.16			.12	2	.94
Dysthymic disorder	-.11	.38	.08	1	.78	.46	.47	.97	1	.32	-.26	.55	.23	1	.63
Alcohol abuse	.08	.60	.02	1	.90	.75	.46	2.70	1	.10	-.06	.80	.01	1	.94
Dissociation	-.002	.01	.10	1	.75										
Group			**8.91**	**2**	**.01**			**8.48**	**2**	**.01**			2.69	2	.26
Emotional Reactivity															
Intercept	5.19	.22	500.56	1	<.001	.76	.16	49.71	1	<.001	5.98	.16	3122.14	1	<.001
Dysthymic disorder	.44	.33	1.81	1	.18	.24	.36	.45	1	.50	-.30	.16	3.60	1	.06
Alcohol abuse	.63	.56	1.28	1	.26	-.05	.32	.03	1	.87	.10	.16	.35	1	.56
Race/Ethnicity			5.57	2	.06			.49	2	.78			3.11	2	.21
Epoch	-.68	.08	212.42	1	<.001	-.11	.03	16.35	1	<.001	.03	.03	.05	1	.83

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Baseline emotion	.60	.07	70.90	1	<.001	.17	.04	14.27	1	<.001	.90	.06	220.27	1	<.001
Dissociation	-.002	.003	.37	1	.55										
Group			7.32	**2**	**.03**			.28	2	.87			1.85	2	.40
Group×epoch			.80	2	.67			.03	2	.98			2.07	2	.36
Emotional Recovery															
Intercept	3.54	.29	77.74	1	<.001	.45	.20	15.76	1	<.001	6.66	.19	2182.28	1	<.001
Dysthymic disorder	-.65	.52	1.53	1	.22	-.24	.33	.55	1	.46	-.18	.20	.80	1	.37
Alcohol abuse	.49	.63	.59	1	.44	.38	.30	1.56	1	.21	-.24	.23	1.09	1	.30
Race/Ethnicity			.62	2	.73			2.96	2	.23			2.35	2	.31
Epoch	.11	.04	28.61	1	<.001	-.15	.05	9.38	1	.002	-.03	.04	7.43	1	.01
Induction emotion	.60	.08	55.39	1	<.001	.21	.03	43.45	1	<.001	.83	.06	186.96	1	<.001
Dissociation	-.004	.01	.58	1	.45										
Group			3.53	2	.17			.47	2	.79			.53	2	.77
Group×epoch			3.84	2	.15			1.94	2	.38			1.18	2	.55
Emotion Regulation: Mindfulness															
Intercept	2.54	.34	53.06	1	<.001	.38	.24	14.50	1	<.001	6.38	.20	1683.88	1	<.001

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Dysthymic disorder	-.62	.35	3.20	1	.07	.12	.34	.12	1	.73	.15	.21	.53	1	.47
Alcohol abuse	-.08	.49	.03	1	.87	.29	.24	1.44	1	.23	-.002	.35	.00	1	.99
Race/Ethnicity			1.86	2	.39			1.55	2	.46			1.32	2	.52
Epoch	.20	.07	44.23	1	<.001	-.16	.06	2.05	1	.15	-.03	.03	6.57	1	.01
Induction emotion	.69	.09	65.22	1	<.001	.21	.04	29.99	1	<.001	.76	.06	142.33	1	<.001
Dissociation	-.01	.01	.80	1	.37										
Group			.61	2	.74			.60	2	.74			1.64	2	.44
Group×epoch			1.45	2	.49			**6.97**	**2**	**.03**			1.59	2	.45
Emotion Regulation: Distraction															
Intercept	2.24	.32	90.50	1	<.001	.37	.24	2.43	1	.12	6.58	.20	2501.49	1	<.001
Dysthymic disorder	-.14	.39	.13	1	.72	.26	.35	.55	1	.46	-.21	.23	.81	1	.37
Alcohol abuse	.39	.40	.95	1	.33	-.33	.29	1.33	1	.25	-.07	.30	.05	1	.83
Race/Ethnicity			.78	2	.68			1.29	2	.52			1.58	2	.45
Epoch	.28	.06	86.29	1	<.001	-.10	.08	.07	1	.79	-.02	.04	2.03	1	.15
Induction emotion	.81	.07	126.93	1	<.001	.30	.03	98.16	1	<.001	.76	.06	182.44	1	<.001
Dissociation	-.002	.01	.13	1	.72										

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Group	2.13	2	.34	1.24	2	.54	3.00	2	.22
Group×epoch	**8.56**	**2**	**.01**	2.31	2	.32	4.17	2	.12

Note. Statistically significant effects relevant to study hypotheses are bolded. SE = standard error; df = degrees of freedom

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