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Predicting the effectiveness of engagement and disengagement emotion regulation based on

emotional reactivity in borderline personality disorder

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Abstract

Improving emotion regulation is central to borderline personality disorder (BPD) treatment, but little research indicates which emotion regulation strategies are optimally effective and when. Basic emotion science suggests that engagement emotion regulation strategies that process emotional content become less effective as emotional intensity increases, whereas disengagement strategies that disengage from it do not. This study examined whether emotional reactivity to emotional stimuli predicts the effectiveness of engagement and disengagement emotion regulation across self-report, general physiologic (heart rate), sympathetic (skin conductance responses), and parasympathetic (respiratory sinus arrythmia) emotion in BPD, healthy, and clinical control (i.e., generalized anxiety disorder; GAD) groups. 120 participants (40 per group) were exposed to emotion inductions and then instructed to implement engagement (mindful awareness) and disengagement (distraction) strategies while self-report and physiological emotion measurements were taken. In the BPD and GAD groups, higher heart rate or respiratory sinus arrythmia reactivity, respectively, predicted improved mindful awareness effectiveness. Higher skin conductance reactivity predicted worsened distraction effectiveness in BPD. Higher reactivity may potentiate engagement emotion regulation, and exacerbate disengagement from emotional content, in BPD. Future research should examine other domains of emotion regulation that may be influenced by emotional intensity, and other forms of emotional intensity that may influence them.

Keywords: Borderline personality disorder; emotion dysregulation; emotion regulation; generalized anxiety disorder; mindfulness

Predicting the Effectiveness of Engagement and Disengagement Emotion Regulation Based on Emotional Reactivity in Borderline Personality Disorder

Borderline Personality Disorder (BPD) is a prevalent public health problem that involves pervasive instability of identity, actions, behaviours, cognitions, and relationships (American Psychiatric Association, 2013). Linehan's (1993) Biosocial Theory suggests that emotion dysregulation is the core of BPD, which involves a biological vulnerability to disrupted emotion coupled with deficits in emotion regulation (i.e., the automatic or volitional manipulation of emotion; Gross & Thompson, 2007). Frontline BPD treatments such as Dialectical Behaviour Therapy (DBT; Linehan, 1993) therefore directly target emotion regulation deficits by training clients in the use of adaptive emotion regulation strategies. Accordingly, among individuals with BPD or high BPD features, the use of DBT skills, and improvements in emotion regulation specifically, predict treatment gains (Axelrod et al., 2011; Neacsiu et al., 2010; Stepp et al., 2008). However, DBT teaches people with BPD hundreds of skills, many of which are emotion regulation strategies, and it is unclear which strategies are optimally effective and when.

Classes of emotion regulation

Given the centrality of emotion regulation deficits to BPD, researchers have begun to more finely delineate the nature of these deficits. Gross' (1998) Process Model delineates between different forms of emotion regulation by highlighting that they can operate at several progressive stages of emotion generation: situation (i.e., selection of an emotional situation or not), attention (i.e., deploying attention towards or away from emotional stimuli), appraisal (i.e., appraising emotional stimuli in more or less emotional ways), and response (i.e., modulating emotional responses and expressions after their elicitation). Basic emotion scientists delineate between two main forms of emotion regulation that operate at distinct stages in this process.

Engagement strategies operate at the "appraisal" stage of the cycle because they involve engaging with or processing the emotional content in some way by representing it in working memory (e.g., thinking about what is upsetting). Conversely, *disengagement strategies* operate earlier in the emotion generative cycle at the attention stage by disengaging with emotional content and diverting attention away from it (e.g., distraction; Sheppes & Gross, 2011; Sheppes, Scheibe, Suri, & Gross, 2011; Sheppes et al., 2014).

Evidence of emotion regulation deficits in BPD. Experimental, ambulatory monitoring, psychophysiological, and neuroimaging research generally suggest that individuals with BPD do not differ from control groups in their ability to reduce emotional responding using engagement strategies. For example, individuals with BPD do not differ from control groups in reductions in self-reported negative emotions or electrophysiological indices of emotional responding while reappraising negative images (e.g., Baczkowski et al., 2016; Lang et al., 2012; Marissen et al., 2010), although research with neural indices is more mixed (e.g., Koenigsberg et al., 2009; Ruocco et al., 2010; Schulze et al., 2011). Similarly, research examining mindful awarenessanother engagement strategy that is emphasized in DBT (Linehan, 2015)- shows similar findings. Mindful awareness involves nonjudgmentally acknowledging, accepting, and embracing the present experience without attempting to modulate or change it (Kabat-Zinn, 1990; Segal et al., 2013). One ambulatory monitoring study suggested that people with BPD exhibit comparable reductions to controls in self-reported emotion using mindful awareness (Chapman et al., 2009). However, emotion is a multi-faceted construct, one with distinct experiential (self-report), sympathetic, and parasympathetic components. Further, these components do not directly and perfectly reflect each other, but rather are more loosely coupled (Berntson et al., 1994; Gross & Thompson, 2007; Mauss et al., 2005). Comprehensively

assessing the impact of this strategy across multiple domains of responding is therefore important to fully probe the existence of emotion regulation deficits or a lack thereof in BPD. Accordingly, experimental studies suggest that individuals with BPD can implement mindful awareness to the same extent as healthy controls (HCs) across self-report, general physiological (i.e., heart rate), sympathetic (i.e., skin conductance responses), and parasympathetic (i.e., respiratory sinus arrythmia) domains (Fitzpatrick et al., 2021; Kuo et al., 2016). These findings indicate that individuals with BPD may not exhibit deficits in the use of engagement emotion regulation strategies.

Research with disengagement strategies similarly suggest that individuals with BPD can implement distraction – another strategy emphasized in DBT (Linehan, 2015)– to reduce selfreported, general physiologic, sympathetic, and parasympathetic indices of emotion to the same extent as control groups (Kuo et al., 2016). However, one study suggested that individuals with BPD are less effective at reducing self-reported emotion, but not sympathetic or parasympathetic emotion, using distraction compared to HC and clinical control (i.e., generalized anxiety disorder) groups (Fitzpatrick et al., 2021). Findings therefore suggest that individuals with BPD may show deficits in the implementation of disengagement strategies in the self-report emotion domain, but not in other physiological ones.

Despite a lack of evidence for emotion regulation deficits in BPD, individuals with BPD are characterized by elevated self-reported negative emotion and physiological emotion at baseline (e.g., Elices et al., 2012; Feliu-Soler et al., 2013; Gratz et al., 2010; Kuo & Linehan, 2009; Kuo et al., 2016; Scott et al., 2013). Individuals with BPD may continue to exhibit heightened negative emotion even after the implementation of an emotion regulation strategy because they "started off" higher, even if their rate of decrease while using the strategy is the same as controls (e.g., Kuo et al., 2016). Thus, although individuals with BPD can regulate, they may need to exhibit even *greater* abilities in emotion regulation than controls to achieve emotional intensity levels that are comparable to non-clinical groups (Kuo et al., 2016). Individuals with BPD thus find themselves at an unfortunate impasse; although they may be able to regulate as well as anyone else, it still might not be *good enough*.

Optimizing emotion regulation in BPD

One way of optimizing emotion regulation in BPD involves identifying factors that indicate which *specific* type of emotion regulation strategy (i.e., engagement versus disengagement) is most likely to be effective in a particular moment to guide strategy selection. Basic emotion scientists suggest that engagement strategies require more cognitive resources than disengagement strategies because they involve processing emotional content. Consequently, they suggest that high emotional intensity may compete for these resources, thereby diminishing engagement strategies' effectiveness. Engagement strategies are therefore theorized to be optimally effective under conditions of low to moderate emotional intensity. Conversely, disengagement strategies do not require processing of emotional content and, in fact, involve shifting attention away from emotional content altogether. Theorists therefore suggest that these strategies might be effective regardless of emotional intensity level. Thus, whereas engagement strategies are theorized to be less effective as emotional intensity increases, disengagement strategies may not show a relationship between emotional intensity and effectiveness (Sheppes & Gross, 2011; Sheppes et al., 2011). Accordingly, both HCs and those with BPD are more likely to select disengagement strategies in response to highly emotionally intense stimuli and engagement strategies in response to less emotionally intense stimuli (Sauer et al., 2016; Sheppes et al., 2011ab). Furthermore, in an experimental study in healthy groups, the effectiveness of

engagement and disengagement strategies were comparable following exposure to low intensity emotional stimuli, but distraction (disengagement) was more effective than reappraisal (engagement) following high intensity emotional stimuli (Sheppes et al., 2014).

Few studies have examined the efficacy of engagement and disengagement strategies in BPD as a function of emotional intensity. Sauer et al. (2016) showed that individuals with BPD rate engagement and disengagement strategies as more difficult to implement in response to higher intensity emotional stimuli than lower intensity emotional stimuli, but HCs do not. However, these authors only measured how successfully participants felt that they *could* employ the strategy, rather than the effectiveness of the strategy in altering emotion itself. One experimental study using a similar paradigm showed that, across both BPD and HC groups, disengagement strategies (distraction) become increasingly effective at reducing sympathetic arousal (skin conductance levels) as the intensity of emotional stimuli increased. However, whereas HCs experienced deteriorations in the effectiveness of the engagement strategy (mindful awareness) in decreasing self-reported emotions as intensity of emotional stimuli increased, the BPD group did not (Fitzpatrick & Kuo, 2016). These findings partially support basic emotion theorizing by suggesting that disengagement strategies may be optimally suited to high intensity situations. However, they also suggest that, while healthy groups may be vulnerable to deteriorations in engagement emotion regulation as a result of increasing emotional intensity, individuals with BPD may not be. These works illustrate when specific forms of emotion regulation may be more or less helpful. However, they are limited by their reliance on the intensity of the stimuli used to elicit emotion as the predictor of the differential efficacy of engagement and disengagement emotion regulation, rather than the intensity of the emotion that individuals are experiencing.

One experimental study examined the impact of the intensity of emotional stimuli and self-reported emotional intensity in predicting choice between disengagement and engagement emotion regulation strategies in individuals with varying levels of BPD features. Self-reported emotional intensity was a stronger predictor of differential choice between engagement and disengagement strategies than emotional stimulus intensity (Kuo et al., 2018). Overreliance on the intensity of emotional stimuli as a predictor of the differential effectiveness of engagement versus disengagement strategies may thus obfuscate when individuals should select particular emotion regulation strategies over others. Further, within this study, BPD features and selfreported emotional intensity did not predict the differential effectiveness of engagement versus disengagement strategies (Kuo et al., 2018). However, this study did not examine participants with a BPD diagnosis, and the differential impact of emotional intensity on engagement versus disengagement emotion regulation may be more evident in clinical populations characterized by greater emotion dysregulation like BPD. In addition, although this study examined whether the level of self-reported emotional intensity prior to the implementation of emotion regulation strategies predicted effectiveness of engagement versus disengagement emotion regulation, it did not examine whether one's emotional reaction to a stimulus does.

Heightened emotional reactivity (i.e., larger changes in intensity from baseline in response to emotional stimuli; Linehan, 1993) is theoretically a core component of BPD, although studies are mixed with respect to whether emotional reactivity is (e.g., Austin et al., 2007; Dixon-Gordon et al., 2013; Ebner-Priemer et al., 2005; Elices et al., 2012; Gratz et al., 2010; 2013) or is not (Feliu-Soler et al., 2013; Fitzpatrick et al., 2021; Jacob et al., 2009; Kuo et al., 2009; 2016; Staebler et al., 2009) elevated in BPD relative to control groups. These findings may be mixed because, whereas baseline emotional intensity appears more consistently elevated in individuals with BPD (e.g., Kuo et a., 2009; 2016), emotional reactivity may be more variable in BPD. Unlike previously described studies—which utilized the intensity of an emotion or emotional stimulus to predict the differential effectiveness of engagement versus disengagement emotion regulation— emotional reactivity encapsulates the extent to which emotional intensity has *changed* in response to a stressor. Given that emotion regulation is frequently employed in response to such *changes* in emotional intensity, examining emotional reactivity as a predictor of differential emotion regulation effectiveness provides important information about the use of these strategies in daily life. Further, as people with BPD may have substantial variability in their emotional reactivity, it may be a potentially better predictor than emotional intensity of which type of emotion regulation strategy may be most effective in BPD at a particular moment in time. However, no studies have investigated this possibility.

It is also unclear whether hypothesized relationships are specific to BPD, populations with elevated emotional reactivity, or all individuals. It is possible that individuals with BPD who exhibit higher levels of experiential avoidance (Chapman et al., 2011) might be particularly likely to experience deteriorations in the effectiveness of engagement strategies under conditions of high emotional reactivity. However, it is less clear whether such effects would be specific to BPD, or pervasive across clinical groups, with these emotional features. For example, individuals with generalized anxiety disorder (GAD) are also characterized by heightened emotion dysregulation (Mennin et al., 2005), exhibit higher emotional reactivity relative to control groups (Macatee & Cougle, 2012), and have elevated experiential avoidance (Buhr & Dugas, 2012). Those with GAD may therefore similarly exhibit a deterioration of the effectiveness of engagement strategies specifically in response to their emotional reactions. Comparing the moderating effect of emotional reactivity on engagement and disengagement emotion regulation in BPD and a GAD group specifically would help disentangle whether group differences between BPD and a healthy group are specific to BPD or pervasive in populations characterized by emotion dysregulation and emotional reactivity.

Finally, dissociation is particularly characteristic of BPD (Ross, 2008) and can dampen subjective and physiological emotion processes, including emotion regulation (e.g., Ebner-Priemer et al., 2005; Krause-Utz et al., 2018). State dissociation may therefore obfuscate comparisons of emotion processes in BPD groups and those with high emotion dysregulation who are not characterized by dissociation, such as GAD. Therefore, controlling for state dissociation is important to disentangle whether emotional reactivity differentially influences emotion regulation across BPD and other high emotion dysregulation groups.

The present study therefore aimed to examine whether emotional reactivity predicts the effectiveness of BPD-relevant engagement (mindful awareness) and disengagement (distraction) strategies comprehensively across self-report, general physiologic, sympathetic, and parasympathetic emotion indices. Further, it examined whether these relationships differed across BPD, HC, or clinical (i.e., GAD) control groups. We hypothesized that, across groups, higher reactivity would predict less effectiveness of mindful awareness (engagement) but not distraction (disengagement). Given a dearth of literature, we considered examination of the moderating effects of group to be exploratory.

Method

Participants

Study procedures received approval from institutional review boards and participants in this study provided informed consent for participation. This study utilized data from a parent study examining emotion processes in BPD (Fitzpatrick et al., 2021). See the parent study for

additional details regarding methodology. Consistent with sample sizes from related studies (e.g., Fitzptrick & Kuo, 2016; Sauer et al., 2016), 40 participants with BPD, GAD, and HCs (N = 120) between the ages of 18 and 60 were recruited and did not differ in age or sex (Fitzpatrick et al., 2020). Participants were excluded if they met criteria for bipolar I disorder, severe psychoticspectrum disorders, or a current substance or alcohol dependence per the Diagnostic and Statistical Manual of Mental Disorders-IV-TR (APA, 2000). Participants taking regularly prescribed psychiatric medications other than Selective Serotonin Reuptake Inhibitors (SSRIs) were also excluded, given research suggesting that SSRIs exert less effects on cardiac responding than other psychiatric medications (Licht et al., 2010). Of the total sample, 14.17% of participants reporting taking medications. In addition, any prospective participants with major neurocognitive problems or illnesses likely to interfere with participation were excluded. Participants were also excluded if they were taking medications that were likely to confound physiological recordings or influence alertness (e.g., H1 histamine receptor blockers; Buccelletti et al., 2009; Hou et al., 2007; Lutfi, 2012). Prospective HC or GAD group participants were excluded if they met four or more diagnostic criteria for BPD, or the self-harm/suicidality BPD diagnostic criterion. Finally, prospective HCs were excluded if they met diagnostic criteria for any current psychological diagnosis or were taking psychiatric medications.

Measures of participant demographics and clinical functioning

All interview assessments were administered by MA- and BA-level assessors under the supervision of a clinical psychologist.

The Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders-IV-TR (SCID-IV-TR; First, Spitzer, Gibbon, & Williams, 1995) was used to examine the presence of what was formerly titled "Axis I" disorders. The SCID-IV-TR has excellent psychometric properties with adequate to good interrater reliability (Lobbestael et al., 2011) and strong convergent validity (e.g., Sprinkle et al., 2002). The average prevalence-adjusted biasadjusted kappas (PABAKs; Byrt, Bishop, & Carlin, 1993) of assessors with a gold-standard assessor was .97 across modules.

The International Personality Disorders Examination- BPD Module (IPDE-BPD; Loranger et al., 1994) was used to assess for BPD. The IPDE-BPD is a reliable and valid measure of BPD pathology, which assesses patterns of attitudes, feelings, and behaviour related to BPD pathology over the past five years and prior to age 25. The IPDE-BPD module has high temporal stability (Mann et al., 1999) and strong convergent validity with other self-report measures of BPD (Schroeder et al., 2010). The average PABAK of assessors with a goldstandard assessor was .95.

Indices of emotional responding and dissociation

Self-report. Participants were provided with a continuous rating dial and asked to keep one hand on the dial and consistently adjust it to reflect shifts in general negative and positive emotional intensity (Ruef & Levenson, 2007). The dial had 10 markers on it, ranging from 0 (very negative) to 9 (very positive), with neutral demarcated between 4 and 5. Consistent with physiological data, average self-reported emotional intensity was calculated for each 30-second epoch, and the average of these epochs were used in analyses as study predictors and outcomes.

Psychophysiology. The BIOPAC 6-channel acquisition system (BIOPAC Systems Inc., Model MP150, Goleta, CA) was used to collect all psychophysiological indices of emotional responding.

Heart rate (HR) was examined as a physiological index of emotional responding with input from both parasympathetic and sympathetic systems (Berntson et al., 2007). A twoelectrode configuration with a bioimpedance module serving as a ground reference was used to examine electrocardiography (BIOPAC Systems Inc., MODEL EL503). HR was indexed by intervals between R-spikes in the electrocardiogram. HR data was processed using Mindware Technologies HRV 2.33 software (Mindware Technologies Ltd., 2011A), wherein R-R intervals were calculated across 30-second epochs. All data was visually inspected and double-scored to ensure that R-spikes were being correctly identified by the software.

Skin conductance responses (SCR) were examined as a sympathetic index of emotional responding. Electrolyte gel was placed on the medial non-dominant middle and index finger (Fowles et al., 1981). Low (35 Hz) and high (.05 Hz) pass filters were used to digitize SCRs at 1000 samples per second and a gain of 1000. Data was processed using Mindware Technologies EDA 2.40 program (Mindware Technologies Ltd., 2011B), indexing SCR as the number of responses over $0.05 \,\mu$ S across 30-second epochs. A programmable rolling filter was used to detect and edit artifacts.

Respiratory sinus arrhythmia (RSA) was examined as a parasympathetic emotion index. After R-R intervals were identified in accordance with preparing the HR data, spectral analysis was used to decompose the electrocardiogram into three different frequency ranges. RSA was measured via the frequency band of spectral analysis between .12 Hz to .4 Hz, as cardiac activity below this frequency is posited to reflect sympathetic rather than wholly parasympathetic influence (Berntson et al., 1997). Respiratory patterns were measured via a respiratory band placed around the chest. RSA was calculated using Mindware Technologies HRV 2.33 software (Mindware Technologies Ltd., 2011A). Mindware software applied a validated algorithm to calculate spectral densities within this frequency band across 30-second epochs. *Dissociation.* State dissociation was measured as a covariate via the Dissociative State Scale (DSS; Stiglmayr et al., 2001). This is a 21-item scale that asks people to indicate a range of dissociative experiences that they are having in the present moment. Scores are summed and higher scores reflect higher levels of dissociation. As detailed in the parent study (Fitzpatrick et al., 2021), Cronbach alpha in the present study was .92 to .93 across study phases.

Emotion induction stimuli (imagery scripts)

Given that BPD is associated with heightened emotional reactivity specifically in response to interpersonally-themed stressors (Limberg et al., 2011), we developed three 2-minute rejection-based scripts to use in the present study. The three scripts were narrated and recorded by the same graduate student and involved rejection from either a mother, friends, or a romantic partner. Consistent with Pitman and colleagues (1987), the number of thoughts (e.g., "you think to yourself 'how could they do this?""), physiological sensations (e.g., "your heart beats faster"), and emotions was consistent across script. Piloting these scripts in a sample of 55 undergraduates suggested that they elicited comparable levels of negative emotion (see Fitzpatrick et al., 2021; see https://osf.io/cqaj2/ for more information about scripts and their impact in a pilot sample).

This study used a mixed design with both between and within-subjects components. Interested participants were contacted by research assistants and briefly screened for inclusion and exclusion criteria. Potentially eligible participants were invited to come to the laboratory for further screening via psychodiagnostic interviews. Eligible participants were invited to return for the experimental procedure and instructed to avoid the ingestion of stimulants such as caffeine and tobacco on the day of their laboratory visit. Electrodes for physiological recordings were attached and participants were instructed in the use of the continuous rating dial.

The experiment consisted of four blocks that repeated across three trials. In Block 1 (*instruction block*), participants were trained in the one of three emotion regulation conditions (REACT, MINDFUL AWARENESS, DISTRACT), depending on the counterbalancing order. In the REACT block, participants were instructed to "act as they normally would" (Kuo et al., 2016) to examine emotional recovery in lieu of an active emotion regulation strategy. Data for this condition is presented elsewhere (Fitzpatrick et al., 2020). The MINDFUL AWARENESS instructions were derived from a combination of scripts by Kuo and colleagues (2016) and Erisman and Roemer (2010) and instructed participants to nonjudgmentally notice any emotional experiences that arise without evaluating them, rejecting them, amplifying them, or attempting to change them in any way. The DISTRACT instructions were derived from past studies and instructed participants to distract themselves from the content of the script by thinking of something that is emotionally neutral (e.g., Kuo et al., 2016; Sheppes & Meiran, 2008). Research assistants asked participants to verbally repeat instructions back to them in order to ensure understanding and clarified misunderstandings as they arose. Participants were instructed to begin to use this strategy when a visual cue appears on the computer screen prompting them to either "REACT", use "MINDFUL AWARENESS", or "DISTRACT".

In Block 2 (*baseline block*), baseline measurements were collected for five minutes without the presentation of stimuli. Immediately following Block 2, in Block 3 (*imagery block*), participants listened to one of three scripts and were instructed to imagine themselves in the scenario depicted. The script terminated after approximately two minutes. Next, in Block 4 (*regulation block*), a prompt on the screen appeared to cue the participant to engage in the emotion regulation strategy determined by the counterbalancing order: "REACT" was always first, followed by either "MINDFUL AWARENESS", or "DISTRACT". Participants

implemented the strategy for two and a half minutes (i.e., five 30-second epochs). After the regulation block, participants completed the DSS measure. Continuous rating dial data and physiological recordings collected throughout.

Participants then repeated this procedure and underwent the next set of instructions, baseline, imagery, and regulation blocks with the next emotion regulation condition. The pairing of imagery scripts with emotion regulation strategy instructions, and the order of MINDFUL AWARENESS and DISTRACT conditions, were counterbalanced across participants. As we detail in the parent manuscript, participants were asked to indicate the percentage of effort that they applied to using emotion regulation strategy after its implementation. These percentage estimates varied from attempting to implement mindful awareness and distraction between 84.32% and 91.93% of the time across groups and strategies, and did not differ across groups (Fitzpatrick et al., 2021).

Data analytic strategy

We conducted a series of univariate analyses of variance with average self-reported emotion, HR, SCR, and RSA during each active regulation block (MINDFUL AWARENESS, DISTRACT) as outcomes. Gender, race, whether or not participants reported being prescribed medications, and age were entered as predictors in order to examine whether these variables were likely associated with outcomes and therefore warranted inclusion in analyses as covariates. These variables did not significantly predict emotion outcomes (*ps* ranged from .138 to .994), with the exception of medication use wherein there was a trend for its prediction of RSA during MINDFUL AWARENESS, F(1, 95) = 3.78, p = .055, and DISTRACTION, F(1, 95) = 3.81, p =.054. As well, race predicted self-reported emotion during MINDFUL AWARENESS implementation, F(11, 97) = 1.94, p = .044. Medication use was included as a study covariate given its theoretical relationship to study outcomes. However, as race was more inconsistent in predicting emotion outcomes and there was not a strong theoretical rationale for its variability with study outcomes, we did not include it as a covariate.

Generalized Estimating Equations (GEE; Burton et al., 1998; Diggle et al., 2013; Hubbard et al., 2010) was used to analyze the data with SPSS version 27 software. GEE is a semi-parametric extension of generalized linear modelling approaches and, like these approaches, optimizes statistical power by allowing for examinations of outcome variables with multiple data points over continuous time courses. GEE also offers the benefit of accommodating and retaining participants who have missing data using the all-available-pairs method. Unlike other multi-level modelling approaches, GEE uses a semi-parametric approach to modeling covariance structures, and therefore yields robust parameter estimates even if they are misspecified or correlations between repeated measures varies across individuals (Burton et al., 1998). GEE analyses were run separately for each condition and emotional index, yielding eight total analyses. Across each analysis, epoch was entered as a within-subjects predictor, and group (BPD, HC, GAD) was entered as a between-subjects predictor. Emotional reactivity of the emotion domain that was being analyzed as the outcome (e.g., RSA, SCR) was computed as the mean emotion in that domain from the induction period minus the mean emotion in that domain from the trial-baseline that immediately preceded it and was entered as a within-subjects predictor. Dissociation (mean centered) and medication use were entered as covariates. Group × Time x Reactivity three-way interactions were entered to examine whether reactivity predicted emotion regulation over time differentially across groups. All lower-level, two-way interactions required to build this higher-order three-way interaction were also entered into the model. 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 a positively skewed count variable (Atkins & Gallop, 2007). Continuous predictors were mean-centered. 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).

Results

Table 1 presents mean self-reported, SCR, and RSA reactivity and emotion across groups and mindful awareness and distraction phases. *[Table 1 near here]*.

Table 2 presents self-report GEE analyses examining the impact of emotional reactivity emotion regulation. There were not statistically significant Group × Time × Reactivity interactions in either mindful awareness or distraction conditions. However, in both conditions, there were statistically significant Time × Reactivity interactions such that, across groups, higher emotional reactivity predicted greater decreases in negative emotion during emotion regulation phases (i.e., improved emotion regulation). *[Table 2 near here]*.

Table 3 presents HR GEE analyses examining the impact of emotional reactivity on emotion regulation. There was a statistically significant Group × Time × Reactivity interaction predicting HR in the mindful awareness condition. Inspection of parameter estimates suggested that there was not a statistically significant Time × Reactivity interaction predicting HR for the HC, $\chi^2(1) = 2.204$, p = .138, or GAD, $\chi^2(1) = .329$, p = .567, groups for the mindful awareness phase. However, for the BPD group, there was a significant Time × Reactivity interaction predicting HR such that higher HR reactivity predicted greater reduction in HR over the mindful awareness phase (B = -.186, *SE* = .047), $\chi^2(1) = 15.647$, p < .001 (i.e., improved emotion regulation). There were no statistically significant interactions between group, time, or reactivity predicting changes in HR in the distraction condition. *[Table 3 near here]*.

Table 4 presents SCR GEE analyses examining the impact of emotional reactivity on emotion regulation. There were no statistically significant interactions between group, time, or reactivity predicting changes in SCR in the mindful awareness condition. However, there was a statistically significant Group × Time × Reactivity interaction predicting SCR in the distraction condition. Inspection of parameter estimates suggested that there was not a statistically significant Time × Reactivity interaction predicting SCR for the HC, $\chi^2(1) = 1.351$, p = .245, or GAD, $\chi^2(1) = 2.783$, p = .095, groups for the distract phase. However, for the BPD group, there was a significant Time × Reactivity interaction predicting SCR such that higher SCR reactivity predicted less reduction in SCR over the distraction phase (**B** = .068, *SE* = .027), $\chi^2(1) = 6.215$, p = .013 (i.e., worsened emotion regulation). [*Table 4 near here*].

Table 5 presents RSA GEE analyses examining the impact of emotional reactivity on emotion regulation. There was a statistically significant Group × Time × Reactivity interaction predicting RSA in the mindful awareness condition. Inspection of parameter estimates suggested that there was not a statistically significant Time × Reactivity interaction predicting RSA for the HC, $\chi^2(1) = 2.009$, p = .156, or BPD, $\chi^2(1) = 1.026$, p = .311, groups for the distract phase. However, for the GAD group, there was a significant Time × Reactivity interaction predicting RSA such that greater increases in RSA from the baseline to the emotion induction (i.e., lower reactivity) predicted less increase in RSA (i.e., worsened emotion regulation) over the mindful awareness phase (B = -.133, SE = .053), $\chi^2(1) = 6.217$, p = .013.

There were no statistically significant interactions between group, time, or reactivity predicting changes in RSA in the distraction condition. *[Table 5 near here]*.

Discussion

This manuscript aimed to examine whether emotional reactivity predicts the effectiveness of two BPD-relevant engagement (mindful awareness) and disengagement (distraction) strategies across self-report and physiologic domains of emotion in BPD, HC, and clinical control (i.e., GAD) groups. We hypothesized that higher reactivity would predict less effective mindful awareness, but would not predict the effectiveness of distraction. Contrary to our hypotheses, our results suggested that higher emotional reactivity appears to *potentiate* the effectiveness of mindful awareness and, if anything, deteriorate the effectiveness of distraction.

Potentiated effectiveness of all emotion regulation strategies for all groups

For self-reported emotion, higher emotional reactivity generally predicted greater emotion regulation effectiveness across groups and strategies. This finding conflicts with theory and research suggesting that higher emotional reactivity may deteriorate emotion regulation, at least for engagement strategies (Sheppes & Gross, 2011; Sheppes et al., 2011). It is possible that the experiential domain is particularly vulnerable to study demand characteristics, such that participants tended to increase and decrease their rating dials by proportionate amounts during the imagery and regulation blocks. Such demand characteristics may not be group- or emotion regulation strategy-specific. Conversely, it is possible that emotion regulation strategies "work best" when individuals perceive that there is some emotion to "work with", regardless of the type of strategy that they are. Specifically, perhaps participants with higher self-reported emotional reactivity perceived their elevated emotion to a greater extent than they would with physiological emotion indices, and consequently were particularly motivated to use both types of emotion regulation strategies to downregulate it. Moreover, those without elevated self-reported emotional reactivity may not have perceived a genuine need for emotion regulation strategies, and therefore may have exerted less effort in their implementation than those with higher selfreported reactivity. Such effects may be unique to the experiential domain wherein emotion is most readily perceived and interpreted.

Potentiated effectiveness of engagement emotion regulation

Converse to the self-report findings, the impact of emotional reactivity on emotion regulation was both group and strategy specific in the physiological indices. For individuals with BPD, higher emotional reactivity potentiated the effectiveness of engagement emotion regulation for heart rate. Similarly, for those with GAD, higher emotional reactivity potentiated the effectiveness of engagement emotion regulation for RSA. Thus, in both clinical groups characterized by high emotion dysregulation, higher emotional reactivity *facilitated* engagement emotion regulation in cardiac domains. These findings are in direct contrast to basic emotion theorizing, which suggests that the effectiveness of engagement strategies deteriorate as a result of increasing reactivity and, if anything, are facilitated for disengagement strategies (Sheppes & Gross, 2011; Sheppes et al., 2011). However, this finding is partially consistent with prior work that suggests that, while HCs experience deteriorating effectiveness of engagement strategies (mindful awareness) in response to increasing emotional stimuli intensity, individuals with BPD do not (Fitzpatrick & Kuo, 2016).

The findings in the cardiac domains directly contradict the assertion that the more intense an emotional response is, the "harder" it is to regulate. Perhaps the reason for these findings is that the "higher an emotion goes", the "farther it has to fall"; Indeed, it is possible that larger emotional responses produce more emotion to "work with" during emotion regulation, which facilitates the emotion regulation process. This may be particularly important for engagement emotion regulation strategies that may require at least some inner emotional content to engage and interact with in order to work. Further, this effect may be unique to the clinical groups that involve high emotion dysregulation because they are characterized by having heightened emotional reactions in their daily lives. Indeed, the emotional reactivity that can be observed in a laboratory setting using a standardized emotion induction for those with BPD or GAD may not approximate the high levels of emotional reactivity that they can experience in an idiographic, evocative, real-life situation. Therefore, perhaps the higher emotional reactivity elicited for individuals with BPD and GAD in a laboratory fell into an "optimal range" for engagement emotion regulation strategy effectiveness for these groups. By contrast, HCs may be less acclimatized to high emotional reactions and thus the emotional reactivity elicited in the laboratory setting may have been at the higher end of their "range". In this case, the effectiveness of engagement strategies would not be potentiated for HCs in the same way as it was for the clinical groups in response to higher emotional reactivity.

Related, it may also be that higher emotional reactivity does not improve emotion regulation effectiveness per se, but lower emotional reactivity in BPD and GAD groups inhibits it. Given the importance of flexibility both in emotional reactivity and regulation to emotional health (e.g., Aldao et al., 2015; Hollenstein et al., 2013; Kashdan & Rotternberg, 2010), perhaps individuals with BPD or GAD with lower cardiac emotional reactivity possessed blunted emotional response systems that are less responsive to both emotional provocation and regulation. Indeed, emotional reactivity in response to a stimulus designed to elicit emotion is not necessarily a marker of emotional dysfunction and may in fact indicate a responsive, sensitive, and fluid emotional system (Porges, 1995). This may be particularly true for BPD and GAD groups, given that both are characterized by a tendency to attempt to avoid emotional experiences (e.g., Buhr & Dugas, 2012; Chapman et al., 2011). Individuals in these groups with higher emotional reactivity may therefore have more flexible emotional response systems that also respond more readily to engagement emotion regulation attempts.

That these effects were evident in the heart rate domain for people with BPD and the RSA domain for people with GAD underscores the importance of comprehensive assessment of emotion across domains. Indeed, discrepancies in emotion indices are common in BPD research (e.g., Baschnagel et al., 2013; Reitz et al., 2012; Scott et al., 2013). Further, given that heart rate is influenced by both sympathetic and parasympathetic systems, but RSA is wholly parasympathetic in nature, these findings suggest that parasympathetic emotion regulation specifically may be influenced by parasympathetic/vagal reactivity in the GAD group. Conversely in BPD, emotional reactivity, when collectively influenced by both sympathetic and parasympathetic changes during engagement emotion regulation rather than reactivity and regulation in specific parasympathetic systems per se.

The parasympathetic domain is theoretically central to the regulation of emotion (e.g., Beauchaine, 2001) and research documents that individuals with BPD have low baseline RSA, including compared to clinical control groups (e.g., Kuo & Linehan, 2009; Kuo et al., 2016). Although the parent study did not find such a group difference (Fitzpatrick et al., 2021), it is possible that individuals with BPD have particularly unresponsive parasympathetic systems that are less likely to be influenced by a range of moderators including emotional reactivity. Alternatively, the parent study showed that individuals with GAD experienced worse emotion regulation in sympathetic domains compared to BPD and HC groups (Fitzpatrick et al., 2021). As an anxiety disorder, perhaps those with GAD have particular difficulties modulating sympathetic arousal and their attempts to do so through emotion regulation strategies are not as responsive to moderators such as emotional reactivity. Accordingly, those with GAD would be less likely to exhibit improved emotional reactivity as a consequence of higher emotional reactivity in the HR domain which involves more sympathetic influence than RSA. This pattern of findings may therefore reveal that, though both BPD and GAD groups may be characterized by emotion dysregulation and experiential avoidance, individuals with BPD and GAD may have particular rigidity in emotion regulation processes in the parasympathetic and sympathetic cardiac domains, respectively. However, this theorizing is largely speculative and more research probing distinct physiological indicators of emotion dysregulation across these two groups is needed.

Deteriorated effectiveness of disengagement emotion regulation

Converse to the cardiac findings, higher SCR reactivity predicted less effectiveness of disengagement (distraction) emotion regulation in the BPD group specifically. This result directly conflicts with basic emotion theory that suggests that the effectiveness of disengagement strategies would not be influenced by rising emotional reactivity (Sheppes & Gross, 2011; Sheppes et al., 2011). It is important to note that the defining characteristic of disengagement strategies like distraction is that they operate on attention by diverting it away from emotional content. Research documents that individuals with BPD have an attentional bias towards emotional stimuli and content (Kaiser et al., 2017). This may be particularly true under conditions of rising emotional reactivity. Indeed, the Emotional Cascades Model of BPD suggests that individuals with BPD respond to rising emotion by ruminating on emotional content which further escalates emotion dysregulation over time (Selby & Joiner, 2009). Thus, while emotional reactivity does not influence disengagement emotion regulation for other groups, it may be particularly challenging for individuals with BPD because it results in them engaging in ruminative processes that inhibit their ability to disengage.

Finally, perhaps these unexpected findings with respect to both engagement and disengagement strategies can be attributed to emotional reactivity being a suboptimal predictor of the differential effectiveness of disengagement or engagement strategies. It may be possible that the magnitude of *change* in an emotional reaction (i.e., reactivity) is less impactful of the differential effectiveness of engagement and disengagement strategies than the *absolute intensity* of that emotion before regulating it. Indeed, as mentioned, research is mixed with respect to whether individuals with BPD exhibit heightened reactivity, under-reactivity, or neither, compared to clinical and healthy control groups (e.g., Austin et al., 2007; Baschnagel et al., 2013; Herpertz et al., 1999; 2000; Kuo et al., 2016; Pfaltz et al., 2015; Rosenthal et al., 2016). Individuals with higher emotional reactivity may therefore still exhibit lower absolute emotional intensity relative to others if their baseline emotional intensity was also lower. Future studies are advised to examine whether absolute levels of emotional intensity predict the differential effectiveness of engagement and disengagement strategies in a similar way as emotional reactivity does, or in a way that is more consistent with basic emotion theory.

Limitations

This work has several limitations. First, as with all laboratory studies, it remains unclear to what extent the emotional reactivity and regulation observed in the laboratory setting generalizes to individuals' daily lives. Perhaps heightened emotional reactivity in response to daily life stressors may influence emotion regulation in entirely unique ways than the reactivity induced in the artificial laboratory environment, and future researchers are advised to investigate this. Related, although participant groups did not differ in the effort they reported putting into emotion regulation strategies, many experimental paradigms assessing emotion regulation strategy implementation, including this one, are limited by a lack of assessment of *how*

participants actually implemented those strategies. It is possible that distinct groups implement emotion regulation strategies in unique ways even when they perceive their application similarly, and future studies should investigate this. Second, this work utilized reductions in emotion as a proxy for emotion regulation. However, there are several potential forms of emotion regulation deficits, including problematic emotion regulation goals, the selection of inappropriate strategies (Gross and Jazaieri, 2014), and rigid inflexibility with which various emotion regulation strategies are applied (Aldao & Nolen-Hoeksema, 2012). It remains unclear how these forms of emotion regulation are influenced by emotional reactivity in BPD, and we therefore encourage future studies to utilize a broader definition of emotion regulation in similar pursuits. Third and related, it is possible that emotional reactivity as computed in the present study does not predict the differential effectiveness of engagement and disengagement strategies in the expected directions, but other emotion indices do. As mentioned, absolute levels of emotional intensity rather than relative changes in intensity may indicate to what extent engagement versus disengagement strategies are likely to be fruitful. Similarly, perhaps high intensity of specific emotions (e.g., anger, shame) predict the differential effectiveness of engagement versus disengagement emotion regulation to a greater extent than general emotional intensity or emotional reactivity. It is important for future research to examine a broad range of emotional experiences that can predict the effectiveness of engagement and disengagement emotion regulation strategies. Examination of a broader range of potential covariates that were not measured in this study (e.g., body mass index) that could be meaningfully related to physiological emotion outcomes is also advised for future work.

Clinical implications

Despite these limitations, the present work provides important information regarding when various forms of emotion regulation may or may not be helpful to people with BPD. Taken together, these findings add nuance to basic emotion theory and clinical lore that people with BPD cannot engage with emotion when emotional reactivity is high. Indeed, they suggest that healthy and clinical groups may exhibit better experiential emotion regulation when their emotional reactivity is higher. For physiological emotion, they further suggest that individuals with BPD may actually exhibit better emotion regulation using mindful awareness in response to higher emotional reactivity than using distraction. Clinicians are thus advised to evaluate assumptions that high emotional reactivity requires disengagement from emotion, especially in light of basic emotion theory that engaging with emotion over time facilitates better emotional health and well-being. Further, disengaging from emotion under conditions of high emotional reactivity may be challenging for individuals with BPD. It is possible that individuals with BPD require more external distractions than their own mental stimuli in order to do this effectively (e.g., movies, sensory information). Clinicians who choose to encourage people with BPD to utilize disengagement strategies are therefore encouraged to appreciate the difficulty of distraction under conditions of high emotional reactivity for this group and seek out ways to optimize its efficacy

Conclusion

In conclusion, this study sought to identify the impact of emotional reactivity on engagement and disengagement emotion regulation in BPD, healthy, and clinical control groups. Contrary to extant research, findings suggested that higher reactivity potentiates the effectiveness of engagement emotion regulation in at least some cardiac domains for those with BPD and GAD, and deteriorates the effectiveness of disengagement emotion regulation in sympathetic domains for individuals with BPD. Researchers are encouraged to extend these findings by further probing which specific domains of emotion regulation are influenced by which specific types of emotional intensity. Ultimately, this study joins with others in the pursuit of adding nuance to an understanding of what "good emotion regulation" is in populations that are theoretically characterized by deficits in it.

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Table 1

Means (standard deviations) of emotional reactivity and emotion during the regulation phases

across groups

	Mindful aw	areness		Distraction		
	BPD	GAD	НС	BPD	GAD	НС
Dissociation	30.553	23.800	8.158	31.290	22.075	6.263
	(4.711)	(3.757)	(2.299)	(4.811)	(4.332)	(1.955)
Self-report reactivity	-1.306	-1.179	-1.102	-1.196	-1.344	-1.181
	(1.367)	(1.501)	(1.258)	(1.320)	(1.347)	(1.487)
Self-report	3.185	3.404	3.670	3.345	3.541	3.791
	(2.138)	(1.955)	(1.905)	(1.958)	(2.098)	(1.956)
HR reactivity	-1.409	-1.561	-1.663	-2.040	-1.756	-1.452
	(2.804)	(3.857)	(3.803)	(3.172)	(2.666)	(3.392)
HR	76.143	72.833	71.385	75.450	73.840	70.301
	(10.735)	(10.342)	(8.181)	(10.631)	(11.349)	(7.774)
SCR reactivity	.142	231	.176	079	.098	.019
	(3.655)	(1.735)	(.922)	(1.400)	(1.883)	(1.188)
SCR	1.790	1.755	1.284	1.868	1.980	1.415
	(1.871)	(2.041)	(1.732)	(2.490)	(2.321)	(1.838)
RSA reactivity	023	045	.012	.009	027	069
	(.571)	(.662)	(.907)	(.649)	(.670)	(.546)
RSA	6.354	6.607	6.561	6.251	6.309	6.624
	(1.464)	(1.163)	(1.071)	(1.289)	(1.155)	(1.139)

Table 2

Generalized estimating equations analysis examining emotion regulation in the self-report

domain

Mindful Awareness						
	Parameter estim	ates		Overall tests of r	nodel effects	
	В	SE	95% CI	Wald Chi-Square	df	<i>p</i> -value
Intercept	2.879	.258	2.373, 3.385	136.944	1	<.001
Medication Use	.098	.489	860, 1.055	.040	1	.842
Dissociation	009	.007	024, .005	1.555	1	.212
Time	.210	.053	.106, .313	72.210	1	<.001
Group ^a				1.082	2	.582
Group = GAD	268	.411	-1.073, .538			
Group = BPD	451	.440	-1.313, .411			
Reactivity	1.192	.226	.750, 1.634	27.804	1	<.001
Group × Time	$\langle \rangle$	Y		.949	2	.622
Group = GAD	.058	.072	803, .199			
Group = BPD	.066	.074	079, .211			
Group × Reactivity	Y			6.893	2	.032
Group = GAD	818	.314	-1.434,202			
Group = BPD	550	.356	-1.247, .147			
Time × Reactivity	238	.061	358,118	42.013	1	<.001
Group × Time ×						
Reactivity				3.423	2	.181

Group = GAD	.109	.069	026, .245
Group = BPD	.024	.083	139, .188

Distraction

	Parameter estim	ates		Overall tests of 1	nodel effects	
	В	SE	95% CI	Wald Chi-Square	df	<i>p</i> -value
Intercept	2.805	.331	2.156, 3.453	123.617	1	<.001
Medication Use	.302	.500	677, 1.281	.365	1	.546
Dissociation	007	.007	021, .008	.840	1	.359
Time	.293	.055	.185, .401	90.943	1	<.001
Group ^a				1.153	2	.562
Group = GAD	448	.487	-1.401, .506			
Group = BPD	026	.510	-1.027, .974			
Reactivity	.792	.282	.239, 1.344	7.151	1	.007
Group × Time)		7.031	2	.030
Group = GAD	.083	.076	066, .233			
Group = BPD	104	.073	247, .039			
Group × Reactivity				3.117	2	.210
Group = GAD	713	.404	-1.505, .079			
Group = BPD	330	.404	-1.123, .462			
Time × Reactivity	187	.049	282,091	18.732	1	<.001
Group × Time ×						
Reactivity				4.907	2	.086
Group = GAD	.149	.067	.017, .281			

Group = BPD	.083	.060	035, .200
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Note. Significant effects relevant to primary study hypotheses are bolded. Reactivity is computed as mean self-reported emotion during the emotion induction that immediately preceded the strategy implementation minus the mean self-reported emotion from the baseline that immediately preceded that induction. Wald chi-squares, degrees of freedom, and *p*-values reflect overall model effects whereas B, standard error, and confidence interval statistics are presented in the context of the reference category. Reference categories = no medication use and healthy control.

Table 3

Generalized estimating equations analysis examining emotion regulation in the heart rate

domain

Mindful Awareness						
	Parameter estimation	ates		Overall tests of n	nodel effects	
	В	SE	95% CI	Wald Chi-Square	df	<i>p</i> -value
Intercept	70.996	1.434	68.185,			
			73.808	2996.517	1	<.001
Medication Use	-1.213	2.735	-6.574, 4.147	.197	1	.657
Dissociation	050	.037	123, .023	1.799	1	.180
Time	053	.202	449, .343	.648	1	.421
Group ^a				6.289	2	.043
Group = GAD	2.630	2.315	-1.907, 7.167			
Group = BPD	6.00	2.399	1.298, 10.702			
Reactivity	581	.343	-1.253, .090	.033	1	.856
Group × Time				.423	2	.810
Group = GAD	100	.244	578, .379			
Group = BPD	.027	.252	467, .522			
Group × Reactivity				2.826	2	.243
Group = GAD	.504	.473	423, 1.431			
Group = BPD	1.101	.691	252, 2.455			
Time × Reactivity	078	.053	181, .025	5.907	1	.015

Group × Time ×						
Reactivity				8.270	2	.016
Group = GAD	.113	.081	046, .273			
Group = BPD	108	.070	246, .030			
Distraction						
	Parameter estim	ates		Overall tests of	f model effec	ts
	В	SE	95% CI	Wald Chi-Square	e df	<i>p</i> -value
Intercept	69.781	1.420	66.999,	2UT		
			72.564	2722.824	1	<.001
Medication Use	-1.314	3.064	-7.319, 4.691	.184	1	.668
Dissociation	041	.040	119, .036	1.089	1	.297
Time	.002	.146	283, .287	4.080	1	.043
Group ^a				4.558	2	.102
Group = GAD	3.365	2.287	-1.118, 7.849			
Group = BPD	5.388	2.579	.334, 10.443			
Reactivity	.113	.367	606, .831	.000	1	.995
Group × Time				2.488	2	.288
Group = GAD	.304	.225	137, .745			
Group = BPD	.330	.266	192, .852			
Group × Reactivity				.575	2	.750
Group = GAD	532	.910	-2.317, 1.250			
Group = BPD	.202	.597	967, 1.371			
Time × Reactivity	103	.045	191,015	2.311	1	.128

Group \times Time \times

Reactivity

Group = GAD	.113	.097	076, .303
Group = BPD	003	.098	196, .190

Note. Significant effects relevant to primary study hypotheses are bolded. Reactivity is computed as mean heart rate during the emotion induction that immediately preceded the strategy implementation minus the mean heart rate from the baseline that immediately preceded that induction. Wald chi-squares, degrees of freedom, and *p*-values reflect overall model effects whereas B, standard error, and confidence interval statistics are presented in the context of the reference category. Reference categories = no medication use and healthy control.

1.461

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.482

2

Table 4

Generalized estimating equations analysis examining emotion regulation in the skin

Mindful Awareness						
	Parameter estim	ates		Overall tests of r	nodel effects	
	В	SE	95% CI	Wald Chi-Square	df	<i>p</i> -value
Intercept	.518	.213	.101, .934	6.966	1	.008
Medication Use	347	.223	784, .090	2.426	1	.119
Dissociation	.001	.003	004, .007	.165	1	.685
Time	091	.058	205, .023	.408	1	.523
Group ^a		,		.898	2	.638
Group = GAD	082	.265	601, .438			
Group = BPD	.132	.271	399, .664			
Reactivity	.033	.213	385, .450	.031	1	.860
Group × Time		Y		2.880	2	.237
Group = GAD	.131	.078	021, .282			
Group = BPD	.085	.073	057, .227			
Group × Reactivity	Y			.258	2	.879
Group = GAD	050	.226	493, .394			
Group = BPD	007	.216	430, .416			
Time × Reactivity	.010	.043	075, .095	.190	1	.663
Group × Time ×						
Reactivity				1.190	2	.552

conductance responses domain

Group = GAD	040	.050	138, .058
Group = BPD	013	.045	101, .076

Distraction

	Parameter estim	ates		Overall tests of 1	nodel effects	
	В	SE	95% CI	Wald Chi-Square	df	<i>p</i> -value
Intercept	.670	.175	.327, 1.104	19.981	1	<.001
Medication Use	237	.202	632, .159	1.378	1	.240
Dissociation	.004	.004	003, .011	1.133	1	.287
Time	071	.050	169, .028	.432	1	.511
Group ^a				.101	2	.951
Group = GAD	010	.266	531, .512			
Group = BPD	.066	.262	447, .580			
Reactivity	.317	.133	.057, .577	3.071	1	.080
Group × Time		<u>)</u>		2.311	2	.315
Group = GAD	.103	.068	030, .236			
Group = BPD	.053	.072	089, .194			
Group × Reactivity				5.231	2	.073
Group = GAD	206	.160	520, .108			
Group = BPD	402	.176	748,057			
Time × Reactivity	072	.062	193, .049	.475	1	.490
Group × Time ×						
Reactivity				10.294	2	.006
Group = GAD	.025	.068	108, .158			

$Group = BPD .140 \qquad .06$	58 .008, .273
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Note. Significant effects relevant to primary study hypotheses are bolded. Reactivity is computed as mean skin conductance responses during the emotion induction that immediately preceded the strategy implementation minus the mean skin conductance responses from the baseline that immediately preceded that induction. Wald chi-squares, degrees of freedom, and *p*-values reflect overall model effects whereas B, standard error, and confidence interval statistics are presented in the context of the reference category. Reference categories = no medication use and healthy control.

Table 5

Generalized estimating equations analysis examining emotion regulation in the respiratory

Mindful Awareness						
	Parameter estim	ates		Overall tests of r	nodel effects	
	В	SE	95% CI	Wald Chi-Square	df	<i>p</i> -value
Intercept	6.630	.190	6.257, 7.002	1292.191	1	<.001
Medication Use	638	.337	-1.299, .024	3.572	1	.059
Dissociation	.004	.004	004, .012	1.048	1	.306
Time	.000	.036	070, .070	3.576	1	.059
Group ^a				1.748	2	.417
Group = GAD	.308	.255	193, .808			
Group = BPD	.035	.298	550, .619			
Reactivity	158	197	543, .228	1.307	1	.253
Group × Time		<i>Y</i>		2.055	2	.358
Group = GAD	060	.049	155, .036			
Group = BPD	074	.062	196, .048			
Group × Reactivity	Y			5.591	2	.061
Group = GAD	.805	.342	.135, 1.475			
Group = BPD	.393	.572	729, 1.514			
Time × Reactivity	.067	.047	025, .159	1.891	1	.169
Group × Time ×						
Reactivity				8.396	2	.015

sinus arrythmia domain

Group = GAD	199	.071	338,060
Group = BPD	154	.098	346, .039

Distraction

	Parameter estim	ates		Overall tests of r	nodel effects	
	В	SE	95% CI	Wald Chi-Square	df	<i>p</i> -value
Intercept	6.811	.183	6.453, 7.170	1412.570	1	<.001
Medication Use	587	.312	-1.199, .025	3.538	1	.060
Dissociation	.007	.004	001, .016	3.031	1	.082
Time	032	.032	094, .030	3.360	1	.067
Group ^a				3.697	2	.158
Group = GAD	116	.262	629, .397			
Group = BPD	519	.279	-1.066, .029			
Reactivity	066	.311	675, .543	.001	1	.978
Group × Time) í		2.192	2	.334
Group = GAD	055	.052	156, .046			
Group = BPD	.030	.053	073, .132			
Group × Reactivity				.246	2	.884
Group = GAD	.021	.473	905, .947			
Group = BPD	.192	.423	638, 1.021			
Time × Reactivity	075	.060	193, .042	.017	1	.897
Group × Time ×						
Reactivity				2.355	2	.308
Group = GAD	.106	.083	056, .267			

.100 .000.000	Group = BPD	.136	.104	068, .339
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Note. Significant effects relevant to primary study hypotheses are bolded. Reactivity is computed as mean respiratory sinus arrythmia during the emotion induction that immediately preceded the strategy implementation minus the mean respiratory sinus arrythmia from the baseline that immediately preceded that induction. Wald chi-squares, degrees of freedom, and *p*-values reflect overall model effects whereas B, standard error, and confidence interval statistics are presented in the context of the reference category. Reference categories = no medication use and healthy control.