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Use Your Words: The Role of Emotion Labeling in Regulating Emotion in Borderline Personality Disorder

Skye Fitzpatrick[1], Ph.D., Jennifer Ip[2], B.Sc., Lillian Krantz[2], M.A., Richard Zeifman[2], M.A., Janice R. Kuo[2], Ph.D.

[1]Department of Psychology, York University, 4700 Keele Street, Toronto, ON, M3J 1P3

[2]Department of Psychology, Ryerson University, 350 Victoria Street, Toronto, ON, M5B 2K3

Correspondence regarding this article should be directed to:
Skye Fitzpatrick,
Assistant Professor,
Department of Psychology,
York University,
4700 Keele Street,
Toronto, ON, M3J 1P3
Email: skyefitz@yorku.ca

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Abstract

Borderline personality disorder (BPD) treatments emphasize emotion labeling to decrease negative emotion and facilitate emotion regulation. However, no studies have examined emotion labeling in BPD or its impact on intentional emotion regulation. The present study examined the impact of emotion labeling on emotion and intentional emotion regulation attempts across self-reported and physiological indices (i.e., skin conductance response [SCR], respiratory sinus arrhythmia [RSA]) in BPD and healthy control (HC) groups. Participants listened to emotionally-evocative scripts and were either instructed to type the emotions that they were experiencing (labeling) or the objects they could imagine seeing in the script (control) into a computer. Following this, they were instructed to use either mindfulness or cognitive reappraisal to decrease their emotion. Self-reported, RSA, and SCR indices of negative emotion were collected throughout and analyzed using generalized estimating equations. Findings indicated that the BPD group experienced higher RSA during emotion labeling compared to the control task, but the HC group did not. HCs reported lower negative emotion after emotion labeling when implementing both emotion regulation strategies compared to the control task, but the BPD group did not. These findings suggest that emotion labeling may activate emotion regulatory systems in BPD and can potentiate intentional emotion regulation in HCs.

Keywords: Borderline personality disorder; emotion regulation; emotion dysregulation; emotion labeling; dialectical behavior therapy

Use Your Words: The Role of Emotion Labeling in Regulating Emotion in Borderline Personality Disorder

Borderline Personality Disorder (BPD) is a mental health problem with high rates of self-harming and suicidal behavior (Soloff, Lynch, & Kelley, 2002) and disability (Zanarini, Jacoby, Frankenburg, Reich, & Fitzmaurice, 2009). There are evidence-based treatments for BPD, with dialectical behaviour therapy (DBT; Linehan, 1993) amassing particularly robust evidence (Cristea et al., 2017). However, standard DBT is long (i.e., at least one year; Linehan, 1993) and imperfect, as effect sizes remain small to moderate (Cristea et al., 2017). It is important to identify methods of improving and facilitating DBT outcomes. Linehan (1993) purports that emotion dysregulation, comprised of a biological emotional vulnerability (i.e., a biological predisposition to disrupted emotion processes such as heightened emotional sensitivity, heightened emotional reactivity, and slower reductions in emotion) and emotion regulation deficits (i.e., difficulties modulating emotional intensity; Gross & Thompson, 2007), is the core deficit of BPD. Accordingly, DBT aims to enhance emotion regulation to modulate emotional vulnerability and improve emotion regulation deficits (Linehan, 2015), which is supported by evidence suggesting that emotion regulation skills is associated with treatment gains (e.g., Axelrod, Perepletchikova, Holtzman, & Sinha, 2011; Neacsiu, Rizvi, & Linehan, 2010; Probst et al., 2018; Stepp, Epler, Jahng, & Trull, 2008). Identifying and leveraging the specific strategies that improve emotion regulation is thus pertinent to enhancing BPD treatment outcomes.

Emotion labeling (i.e., using a specific word to describe an emotion) is a heavily emphasized emotion regulation strategy in both DBT and other evidence-based treatments such as cognitive behavioral therapy (CBT; e.g., Beck, Rush, Shaw, & Emery, 1979; O'Donohue & Fisher, 2009). In CBT and DBT, clients are taught emotion labeling as a standalone strategy to

decrease negative emotion and also as a preliminary step before implementing subsequent emotion regulation strategies (Barlow et al., 2011; Beck et al., 1979; O'Donohue & Fisher, 2009; Linehan, 2015). For example, clients are often instructed to use emotion labeling as part of the process of engaging in mindfulness (i.e., observing, describing and accepting emotional responses without judgement; Kabat-Zinn, 1990; Segal, Williams, & Teasdale, 2013), a core DBT emotion regulation strategy (Linehan, 2015). Furthermore, in CBT, emotion labeling is a quintessential first step to implementing cognitive reappraisal (i.e., re-thinking an emotional situation; Gross & John, 2003), a hallmark skill of the treatment (Beck et al., 1979; Barlow et al., 2011). However, it is unknown whether emotion labeling effectively decreases negative emotion, or potentiates other emotion regulation strategies, in BPD. The present study therefore examines the impact of emotion labeling on negative emotion and emotion regulation in BPD.

Why Is Emotion Labeling Important? Lessons from Basic Emotion Science

Emotion labeling is purported to decrease negative emotion by activating the right ventrolateral prefrontal cortex (RVLPFC), which subsequently exerts a downregulating influence on regions associated with the generation of negative emotion such as the amygdala (e.g., Lieberman et al., 2007). Emotion labeling has thus been conceptualized as an "*incidental emotion regulation strategy*" because it dampens emotional responding without necessarily being deployed for an emotion regulatory goal. Conversely, strategies such as cognitive reappraisal are conceptualized as "*intentional emotion regulation strategies*," given their explicit function of regulating emotion (Lieberman, Inagaki, Tabibnia, & Crockett, 2011).

The majority of emotion labeling studies have tested its efficacy in decreasing negative emotion by utilizing a paradigm that presents facial expressions and asking participants to either label the emotional expression conveyed, or engage in a control task by labeling or matching the

emotion or gender conveyed, from a list of options. Research using this paradigm indicates that emotion labeling results in higher RVLPFC responses than emotion matching (Torrisi et al., 2013) and lower amygdala activation than all aforementioned controls (Lieberman et al., 2007; Torrisi et al., 2013). Dynamic causal modeling further reveals that the RVLPFC exerts a dampening influence on the amygdala during emotion labeling, supporting theoretical assertions regarding its neural mechanisms (Torrisi et al., 2013). Furthermore, a recent meta-analysis suggested that emotion tasks that use specific emotion words (e.g., "sad") elicit higher activation in semantic processing regions and lower amygdala responses than non-emotion words (e.g., gender words) and non-specific emotion words (e.g., "unpleasant"). These findings suggest that using precise emotion words, rather than non-specific emotion terms, is crucial for emotion labeling's effect of decreasing negative emotion (Brooks et al., 2017). Emotion labeling also elicits equivalent reductions in self-reported negative emotion (i.e., comparable emotion regulation) compared to the use of intentional emotion regulation strategies, such as cognitive reappraisal and distraction (Lieberman et al., 2011), and comparable amygdala activation to cognitive reappraisal (Payer, Lieberman, & London, 2012). These findings collectively support the status of emotion labeling as an incidental emotion regulation strategy.

External Validity Pitfalls in Emotion Labeling Research

Labeling one's own emotion. There are several gaps in the emotion labeling literature that limit its generalizability to BPD treatments. First, the aforementioned studies typically ask participants to label an emotion that matches a facial expression by selecting a word from a standardized list. Participants therefore do not label their *own* negative emotion, but rather an external facial expression. This approach is inconsistent with real-world therapeutic experiences, which involve labeling one's own negative emotion without necessarily constraining responses

to lists. Several non-clinical studies have employed more generalizable emotion labeling paradigms that address this. For example, one study presented negative images and asked healthy participants to engage in intentional emotion regulation strategies (observe or cognitively reappraise negative emotion), or the incidental emotion regulation strategy of labeling *their own* negative emotion using one of three words provided. Both cognitive reappraisal and emotion labeling were associated with greater activation in prefrontal regions including the RVLPFC, and reductions in amygdala activation and self-reported negative emotion, compared to passively viewing images (Burklund et al., 2014). Another study further increased generalizability by allowing undergraduates to use any word to label emotion. Results revealed that cognitive reappraisal resulted in lower, and emotion labeling resulted in *higher*, self-reported negative emotion, compared to passively viewing emotional images. The conditions did not differ in sympathetic indices of emotion (i.e., skin conductance response [SCR]; Ortner, 2015). Finally, among healthy controls (HCs), one study compared the effects of labeling emotional expressions conveyed in images (i.e., external emotion) to labeling one's own emotion in response to images. When participants labeled emotional pictures, their SCRs decreased over time, but when they labeled their own emotion, SCR *increased* over time (McRae, Taitano, & Lane, 2010). These findings suggest that the efficacy of emotion labeling as an incidental emotion regulation strategy may be dependent on the extent to which individuals generate their own labels. It also suggests that, although labeling one's own emotion is emphasized in clinical interventions, it may not be as effective as the forms of labeling that have typically been studied. These findings underscore the importance of examining emotion labeling in an externally valid, therapeutically informed way to understand its real-world utility. Such externally valid forms of assessment also require controlling for baseline trait differences in the *ability* to identify and label emotions (i.e.,

alexithymia; Sifneos, 1973) in order to avoid conflating the efficacy of emotion labeling with trait differences in difficulty labeling emotion. The aforementioned studies did not control for alexithymia, and it thus remains unclear if findings are accounted for by this construct.

Emotion labeling in BPD. Generating externally valid information about emotion labeling also requires examining the efficacy of this strategy in BPD samples. Studies with other clinical samples reveal a diverse pattern of findings with respect to the effects of emotion labeling: Individuals with bipolar disorder (Almeida et al., 2009; Foland et al., 2008; Howells, Rauch, Ives-Deliperi, Horn, & Stein, 2014) and social anxiety disorder (Burklund, Craske, Taylor, & Lieberman, 2015) show distinct patterns of neural activation during emotion labeling compared to HCs, but individuals with treatment-resistant depression (Ferri et al., 2017) and methamphetamine dependence (Payer, Lieberman, & London, 2011) do not. Individuals with bipolar disorder also do not show differences from HCs in heart rate variability during emotion labeling (Howells, Rauch, Ives-Deliperi, Horn, & Stein, 2014). The efficacy of emotion labeling may thus depend on the clinical group implementing the strategy, and the emotion indices assessed. As individuals with BPD are theorized to have emotion regulation deficits (Linehan, 1993) and exhibit higher alexithymia than healthy and some clinical groups (New et al., 2012; Lysaker et al., 2017), emotion labeling may be uniquely challenging for them. However, the efficacy of emotion labeling in BPD is unknown. In addition, state dissociation is common in BPD (Ross, 2007) and dampens autonomic indices of emotion (Ebner-Priemer et al., 2005). Failing to assess and control for state dissociation can mask real differences in emotional responding between BPD and control groups because the negative emotion of individuals with BPD can consequently appear more attenuated than it is in reality. Failing to control for state dissociation also obfuscates differences between emotion labeling and control conditions in

BPD, as dissociation can promote an illusion of blunted or unresponsive emotion. In alignment with other studies on emotion processes in BPD (e.g., Hazlett et al., 2012; Kuo & Linehan, 2009; Kuo et al., 2016), controlling for state dissociation is thus important to isolate the impact or lack thereof of emotion labeling in BPD.

Emotion labeling as a prerequisite to intentional emotion regulation. Finally, the lack of examination of whether emotion labeling potentiates intentional emotion regulation strategy use for individuals with BPD leaves a gap between research and practice. Evidence-based psychotherapies such as CBT (Beck et al., 1979; O'Donohue & Fisher, 2009) and DBT (Linehan, 2015) encourage the use of emotion labeling prior to intentional emotion regulation strategies like cognitive reappraisal and mindfulness. Consistently, studies with other populations and treatments suggest that emotion labeling may potentiate intentional emotion regulation. For example, individuals with anxiety disorders who label emotion during initial exposures show greater reductions in SCR, but not self-reported negative emotion, during exposures one week later, compared to those who do nothing, cognitively reappraise, or distract during initial exposures (Kircanski, Lieberman, & Craske, 2012; Niles, Craske, Lieberman, & Hur, 2015). If emotion labeling can potentiate exposure effects over time, then it may similarly facilitate intentional emotion regulation strategies such as cognitive reappraisal and mindfulness. However, it remains unclear whether emotion labeling potentiates DBT-specific intentional emotion regulation, or whether its effects are specific to one strategy but not others. Moreover, studies examining the potentiating influence of emotion labeling on exposures suggests that this effect may be specific to *some emotion domains* (e.g., SCR), but not others (e.g., self-report). Emotion is a multi-faceted process with independent behavioral, self-report, sympathetic, and parasympathetic domains (Berntson, Cacioppo, Quigley, & Fabro, 1994). Assessing emotion

labeling comprehensively across multiple domains- both sympathetic and parasympathetic- is therefore pertinent to understanding its effect.

The Present Study

In sum, despite the emphasis on emotion labeling in DBT, it is unclear whether individuals with BPD differ from HCs in their ability to decrease negative emotion across emotion domains using an externally valid form of emotion labeling. Further, it is unknown if emotion labeling potentiates intentional emotion regulation strategies, such as cognitive reappraisal and mindfulness, and whether this effect is specific to strategies or pervasive across them. The present study therefore investigated whether self-generated emotion labeling decreases negative emotion across self-report, sympathetic, and parasympathetic domains and, whether this effect differs across BPD and HC groups. We also examined whether emotion labeling potentiates cognitive reappraisal and mindfulness and if this effect differs across BPD and HC groups, intentional emotion regulation strategies, or whether groups and strategies interact to influence its efficacy. We investigated these questions by instructing BPD and HC groups to listen to emotionally-evocative scripts and either label their own emotion or objects that they could imagine in the script (control), and then implement mindfulness or cognitive reappraisal. Alexithymia and state dissociation were controlled for. Based on extant theory and research (e.g., Brooks et al., 2017; Burklund et al., 2014; Linehan, 1993; Payer et al., 2012; Lieberman et al., 2007; Lieberman et al., 2011; Torrisi et al., 2013), we hypothesized that (1) emotion labeling would decrease negative emotion more than a control task but that (2) this effect would be smaller in BPD compared to HC groups. Drawing upon DBT models of emotion regulation (Linehan, 2015), we hypothesized that (3) emotion labeling would potentiate intentional emotion regulation strategies. Given a dearth of literature indicating whether emotion

labeling would potentiate intentional emotion regulation moreso for a specific group or intentional emotion regulation strategy, we considered these analyses exploratory.

Method

Participants

Thirty participants with BPD and 30 HCs between the ages of 18 and 60 were recruited via fliers, online advertisements, and outreach to previous study participants who expressed interest in future research opportunities. Participants with BPD were excluded if they had current psychological disorders that could significantly interfere with their ability to understand or participate in study tasks: severe psychotic-spectrum disorders, bipolar I disorder, and current alcohol or substance dependence. Given the potential confounding impact of psychiatric medications on psychophysiology, participants were also excluded from the study if they were taking any scheduled psychiatric medications other than selective serotonin reuptake inhibitors, which exert less pronounced effects on cardiac indices (Licht, de Gues, van Dyck, & Penninx, 2010). HCs were age- and sex-matched to the BPD group to account for age- and sex-related differences in cardiac activity (e.g., Antelmi et al., 2004). HCs were excluded if they endorsed four out of nine BPD diagnostic criteria or the self-harm/suicidal behaviors BPD criterion in order to ensure diagnostic divergence between groups. As well, HCs were excluded if they met full diagnostic current criteria for any of the assessed psychological disorders, or were taking psychiatric medications. All prospective participants were excluded on a case-by-case basis if they were taking medications (e.g., H1 histamine receptor blockers, beta-blockers; Hou, Langley, Szabadi, & Bradshaw, 2007; Sandrone et al., 1994) or had medical conditions (e.g., heart problems, epilepsy; Buccelletti et al., 2009; Tomson, Ericson, Ihrman, & Lindblad, 1998) that were likely to interfere with task participation or physiological recordings. Demographics are

presented in Table 1. Diagnostic comorbidities in the BPD group are presented in Table 2.

Measures

Diagnostic assessments. Participants were screened for BPD in online or phone formats based on their preferences using the McLean Screening Inventory (MSI; Zanarini et al., 2003). The MSI asks participants 10 "yes" or "no" questions about BPD-related criteria. If prospective BPD participants endorsed five or more questions, they were invited to come into the laboratory for further assessment. Our experiences indicated that participants endorsed more criteria online than on the phone. Therefore, prospective HCs were excluded based on this measure if they endorsed four (phone) or six (online) or more questions, or the self-harm question. Thresholds for screening HCs were based off our previous observations that prospective HCs endorsed more questions online than over the phone, possibly because phone screeners would further probe participant's endorsements to assess if they met clinically-relevant thresholds, which is not possible online. We therefore determined that applying phone screening thresholds to those who completed online screenings likely ruled out eligible participants.

Diagnostic interviews. MA- and undergraduate-level assessors under the supervision of a licensed clinical psychologist administered the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders-IV-TR (SCID-IV-TR; First, Spitzer, Gibbon, & Williams, 1995) to assess for what was formerly known as "Axis-I" psychopathology. The SCID-IV-TR is a well-established semi-structured interview that reliably assesses for disorders based on DSM-IV-TR diagnostic criteria. The SCID-IV-TR has strong convergent validity. For example, the depression module is highly correlated with validated self-report measures of depression symptoms (Sprinkle et al., 2002). In the present study, inter-rater reliability with a gold-standard assessor as indexed by the prevalence-adjusted bias-adjusted kappa (PABAK;

Byrt, Bishop, & Carlin, 1993) ranged from .67 to 1.00 across assessors and modules, with an average of .97.

The International Personality Disorders Examination- BPD (IPDE-BPD) Module (Loranger et al., 1994) was used to assess for BPD. The IPDE assesses attitudes, feelings, and behavior patterns over the past five years and before 25 years old. Research supports its interrater reliability (kappa = .90) and temporal stability (kappa = .82; Mann et al., 1999), and shows that it correlates highly with self-report BPD measures (Schroeder, Andresen, Naber, & Huber, 2010). In the current study, inter-rater reliability with the gold-standard assessor as indexed by the PABAK ranged from .67 to 1.00 across assessors, with an average of .95.

Covariates. The Toronto Alexithymia Scale (TAS; Bagby, Parker, & Taylor, 1994) was administered to control for individual differences in trait difficulty identifying and labeling emotions. The TAS is a 20-item measure that asks participants to indicate the extent of agreement with various statements from 1 ("strongly disagree") to 5 ("strongly agree"). It has strong psychometric properties. For example, higher scores on the TAS have strong negative correlations with related measures such as psychological mindedness (Bagby, Taylor, & Parker, 1994). In the present study, the TAS also had a Cronbach alpha of .89.

The Dissociative State Scale (DSS; Stiglmayr, Shapiro, Stieglitz, Limberger, & Bohus, 2001) was used to measure state dissociation. The DSS is a 21-item measure that asks participants to rate the extent of a range of dissociative phenomena (e.g., "I have the impression that my body or a part of it is insensitive to pain") that they are experiencing "just now" from 0 ("none") to 9 ("very strong"). Higher scores indicate more severe current dissociation. The DSS also has good convergent validity, as higher scores correlate strongly and positively with aversive inner tension (Stiglmayr et al., 2001). In the present study, the DSS exhibited strong

internal reliability, with Cronbach alphas at each timepoint ranging from .93 to .95.

Dependent variables. Emotion was measured across self-report, sympathetic, and parasympathetic domains.

Emotion: self-report. Visual analogue scales (VAS) ranging from 0 (not at all) to 100 (very) were used to assess participants' current self-reported negative emotion across 11 basic and BPD-relevant emotions: fear, loneliness, anger, anxiety, shame, disgust, emptiness, guilt, hopelessness, sadness, and tension. Scores from each emotion were averaged to yield a composite of negative emotion. Research suggests that VAS' reliably examine psychological states (McCormack, Horne, & Sheather, 1988). For example, baseline levels of sadness, anger, and fear as acquired via the VAS distinguish between BPD and HC groups (Kuo & Linehan, 2009). The negative emotions assessed in this study were all moderately to highly positively correlated (see section and table S2 in supplemental information, for correlation matrix and its interpretation), supporting the validity of combining them into one composite. Their combination was further supported by their excellent internal reliability, with Cronbach alphas at each timepoint ranging from .94 to .96.

***Emotion: sympathetic responding*.** Skin conductance responses (SCR) were collected as a measure of sympathetic nervous system responding (Dawson, Schell, & Filion, 2007) using the BIOPAC 6-channel acquisition system (Model MP150, Goleta, CA). We elected to collect SCRs as a measure of sympathetic responding over skin conductance levels because the former is less vulnerable to drift over prolonged stimulus presentations such as the ones in the present study (Dawson et al., 2007). Research assistants placed electrodes on participants' medial phalanges of the non-dominant middle and index finger, consistent with gold standard recommendations (Fowles, Christie, Edelberg, Grings, Lykken, & Venables, 1981). SCRs were digitized at a rate

of 1000 samples per second and a gain of 1000 with low (35 Hz) and high (.05 Hz) pass filters in accordance with standard SCR conventions in psychological research (e.g., Braithwaite, Watson, Jones, & Rowe, 2015). Data were separated into 30-second epochs and were processed using Mindware Technologies EDA 2.40 program and a programmable rolling filter that detects and edits artifacts. The number of responses exceeding 0.05 μS within a 30-second epoch was indexed as SCR. This threshold is consistent with field standards and conventions for recording SCR (e.g., Braithwaite et al., 2015; Dawson et al., 2007; Fowles et al., 1981).

***Emotion: parasympathetic responding*.** Respiratory sinus arrhythmia (RSA) was collected as a measure of parasympathetic responding (Beauchaine, 2001; Porges, Doussard-Roosevelt, & Maiti, 1994) using the same BIOPAC system. A two-electrode configuration with a bioimpedance module for grounding was used to collect heart rate data, and a respiratory band was placed around the chest. Mindware Technologies HRV 2.33 software was used to process the data into 30-second epochs. The data was visually inspected and double-scored by two independent research assistants to ensure that the correct R-spikes in the electrocardiogram were identified and to remove artifacts. Following this, spectral analysis decomposed the electrocardiogram into three frequency ranges, retaining the highest frequency band (greater than .15 Hz), conceptualized to reflect parasympathetic responding (Berntson et al., 1997). A validated algorithm was then implemented to calculate spectral densities within this high frequency band across 30-second epochs.

Emotion induction stimuli. Four emotionally-evocative auditory scripts (one for each trial) were used as emotion inductions in the present study. These scripts were piloted in order to ensure that they effectively elicited general negative emotion. In developing the scripts, we used Pitman, Orr, Forgue, de Jong, and Clairborn's (1987) method by identifying that the number of

thoughts, physiological sensations, and emotions described in each script were consistent (five for each category in each script). Some scripts were inspired by scenarios in emotion inductions from Cook, Hawk, Davis, and Stevenson's (1991) work, but were rewritten and edited to ensure sufficient length and consistency of number of thoughts, sensations, and emotions. The scripts involved the death of one's mother ("mother"), a betrayal of one's romantic partner with one's best friend ("betrayal"), a hit-and-run car accident ("car"), and the death of a dog ("dog"). These scripts were piloted on 54 undergraduates who were played each one through headphones in counterbalanced order and completed self-report measurements of fear, sadness, and anger. Generalized estimating equations (see description, below) showed that the scripts resulted in increases in pre- to post negative emotion (i.e., the sum of intensity of fear, sadness, and anger), (B = 101.30, SE = 7.92), Wald $\chi^2(1) = 163.59$, $p < .001$, fear (B = 27.06, SE = 2.65), Wald $\chi^2(1) = 104.11$, $p < .001$, sadness, (B = 36.07, SE = 3.05), Wald $\chi^2(1) = 139.44$, $p < .001$, and anger (B = 38.07, SE = 3.24), Wald $\chi^2(1) = 138.13$, $p < .001$. However, the scripts varied in the intensity of each emotion elicited (see Table S1 in supplemental information for means of emotions elicited by each script and Section S1 comparisons of elicitation of these emotions by script). The final scripts were approximately two minutes in length[1]. Each script is spoken through second-person narration (e.g., "you hear wheels screeching"), and participants are told multiple times to imagine that they are the protagonist and that the events described are occurring to them,

[1] Three of the four auditory scripts varied in length between 1-minute and 47-seconds to 1-minute and 55-seconds. In order to consistently yield four epochs for each induction, participants continued to be monitored for the remaining time after the auditory script ended (i.e., for between 5 and 13 seconds). One script was 2-minutes and 7-seconds in length. For this script, physiological data from the last epoch was scored in a 37-second segment.

making the events and their sensations as vivid as possible.

Procedures. Institutional ethics review boards approved all study procedures. Interested participants either completed an online or phone MSI (Zanarini et al., 2003) screening to assess for potential study eligibility, in addition to generic questions about demographic factors and medical and psychiatric histories. If they remained eligible following initial screening, they were invited for in-person full informed consent procedures and a psychodiagnostic assessment (SCID-IV-TR and IPDE). Eligible participants then either immediately underwent the laboratory procedure or were scheduled to return on a separate day. Participants were instructed to avoid consuming caffeine, nicotine, or mood-altering substances on the day of their assessment and experiment to minimize their effects on psychophysiological recordings or study procedures.

On the testing day, participants first completed the TAS and physiological recording equipment was then attached. The entire experiment was a within-subjects design, and each experimental trial consisted of three blocks: a BASELINE, an EMOTION INDUCTION, and an INTENTIONAL EMOTION REGULATION block. The direct effect of emotion labeling on negative emotion was measured during the EMOTION INDUCTION block, which varied depending on whether participants were instructed to label their emotions (LABEL condition), or engage in a control task (CONTROL condition). The impact of emotion labeling on intentional emotion regulation was measured during the INTENTIONAL EMOTION REGULATION block, which varied depending on whether participants were instructed to use MINDFULNESS or REAPPRAISAL.

Experimental paradigm training. After the physiological sensors were attached, participants were then trained in the experimental paradigm. For the emotion induction, participants were told that they would hear an emotional script in their headphones and the

computer screen would simultaneously either present the question “what are you feeling right now?” (LABEL condition) or “what objects can you imagine seeing in the scene?” (CONTROL condition). For the LABEL condition, participants were told to type the emotions that they were feeling into the computer. They were also provided with a list of example emotion responses on the screen throughout the paradigm (i.e., anxious, angry, afraid, sad, ashamed, guilty, disgusted, surprised, interested, happy, neutral), but were free to deviate from this list. To help participants identify emotions specifically, research assistants reviewed the difference between thoughts and emotions and asked participants to constrain each emotion response to one word only. Research assistants also emphasized that participants may not experience negative emotion, and encouraged them to label any emotional experience that they observed, including positive emotions, neutrality, or boredom. For the CONTROL condition, participants were told to type objects that they could imagine seeing in the script that was being played. Participants were provided with a list of example responses (e.g., pens, chairs, cars), and instructed that they could use these examples or deviate from this list. This control condition allowed for the examination of the unique effect of emotion labeling while controlling for the behaviour of typing on the computer during simultaneous script presentation. This control condition was selected over common distraction-based controls (e.g., type words that begin with the letter ‘S’) because it avoids the conflation of labeling emotion with distracting from emotion, which is also conceptualized as an emotion regulation strategy (e.g., Webb, Miles, & Sheeran, 2012). Participants were told to provide as many responses in each condition as felt accurate for them and to continue typing new emotions or objects as they arise, but that they must provide at least one response during emotion EMOTION INDUCTION block.

For the INTENTIONAL EMOTION REGULATION block, participants were told that

they would be presented with a screen that asked them to either practice MINDFULNESS or REAPPRAISAL. Participants were not presented with any stimuli during the INTENTIONAL EMOTION REGULATION block other than an instruction on the computer screen to implement the intentional emotion regulation strategy. Rather, participants were instructed to sit quietly and utilize the strategies to reduce any negative emotions that they were presently experiencing. For mindfulness, participants were instructed to nonjudgmentally notice and observe their current emotional experiences without attempting to reject or cling to them. For reappraisal, participants were instructed to attempt to change their emotions by interpreting the emotion induction in a different way (e.g., telling oneself that the script is fictional). Prior to the beginning of the experimental procedure (after training), participants verbally demonstrated their understanding to research assistants who provided corrective feedback until it was clear that participants understood the instructions. Research assistants repeatedly emphasized the importance of labeling accurate emotion and reporting accurate emotion intensity on VAS', even if the emotion experienced was not negative or intense, in order to minimize demand characteristics.

Experimental procedure. Following training, participants underwent the experimental procedure, which consisted of four trials. In each trial, participants first engaged in a 5-minute baseline period during which they sat quietly with no computerized stimuli (i.e., a black screen) while physiologically monitored (BASELINE block), after which they completed a VAS. Next, participants underwent the EMOTION INDUCTION block, with either the LABEL or CONTROL condition, for approximately two minutes, after which they completed another VAS. Finally, participants underwent the INTENTIONAL EMOTION REGULATION block, with either the MINDFULNESS or REAPPRAISAL condition, for 2.5 minutes, after which they completed another VAS. Physiological data were collected throughout. They completed the DSS

after each intentional emotion regulation block. Participants repeated this sequence with a different pairing of emotion induction and intentional emotion regulation conditions. This procedure used a 2×2 design, such that all participants received each combination of emotion induction conditions (i.e., LABEL or CONTROL) and intentional emotion regulation strategies (i.e., MINDFULNESS or REAPPRAISAL), yielding four trials for each participant (LABEL-MINDFULNESS, LABEL-REAPPRAISAL, CONTROL-MINDFULNESS, CONTROL-REAPPRAISAL). The order of the trials was counterbalanced across participants with the constraint that the two LABEL conditions (i.e., LABEL-MINDFULNESS, LABEL-REAPPRAISAL) and the two CONTROL conditions (i.e., CONTROL-MINDFULNESS, CONTROL-REAPPRAISAL) were presented consecutively in order to minimize spillover from the prior condition. Thus, there were four versions of the experiment, which varied whether LABEL or CONTROL conditions occurred in the first two trials and the order in which MINDFULNESS and REAPPRAISAL were presented, which was counterbalanced across participants. Furthermore, participants never heard the same script twice, and the matching of scripts to conditions was also counterbalanced across participants so that each script was matched to each condition. Figure 1 provides an example of an experimental trial. At the end of each condition, participants were asked to indicate the percentage of time that they attempted to follow the instruction that was given to them for that condition (e.g., to label emotion, to implement mindfulness).

Data Analytic Strategy

Participant entries during the LABEL and CONTROL conditions were visually inspected. Trials where participants did not provide any responses, typed objects in the LABEL

conditions, or internal states in the CONTROL conditions, were omitted from analyses. This was not applied to instances where participants wrote incomplete or indecipherable words.

Generalized Estimating Equations (GEE; Burton, Gurrin, & Sly, 1998; Diggle, Heagerty, Liang, & Zeger, 2002; Hubbard et al., 2010) analyses were run using SPSS version 25 software. GEE is a semi-parametric method derived from generalized linear modelling analyses that allows for the study of outcome variables over multiple time points and uses within-cluster similarity of residuals to derive regression parameter estimates. Similar to other multilevel modeling approaches, GEE examines predictors nested within varying levels of analysis. GEE also accommodates missing data using an all-available pairs approach to maximize statistical power. However, distinct from other multilevel modeling approaches, GEE utilizes a semi-parametric method of estimation that first derives individual point estimates and then conducts a second estimation based on the covariance structure of the repeated measures. This semi-parametric method is unique to GEE and makes it robust to misspecification of covariance structures. In addition, given that several outcome variables in the present study were not normally distributed, GEE offers the advantages of allowing the specification of various forms of distributions (Burton et al., 1998; Diggle et al., 2002; Hubbard et al., 2010). Exchangeable, Autoregressive, and Unstructured covariance structures were considered for the data, and the model with the lowest corrected Quasilikelihood under the Independence Model Criterion (QIC) value was chosen. Analyses examining SCR data as the outcome were modelled with a negative binomial distribution to accommodate for the positively-skewed and count nature of this data (Atkins & Gallop, 2007; Hilbe, 2011). Given concerns about potential of Bonferroni alpha corrections to over-inflate of Type II error (e.g., Cabin & Mitchell, 2000; Moran, 2003; Nakagawa, 2004;

O'Keefe, 2003; Rothman, 1990) and assertions that alpha corrections are overly stringent for hypothesis-driven research (e.g., Rothman, 1990), they were not employed.

Preliminary analyses were conducted to determine whether the emotion inductions sufficiently elicited negative emotion (i.e., a manipulation check). Three separate GEE analyses were run, one for each index of negative emotion. Negative emotion (i.e., self-report, SCR, and RSA) across the ten epochs of the baseline block and four epochs of the emotion induction block for each trial was entered as the outcome. Block (baseline versus emotion induction) was entered as a predictor, with self-reported dissociation from each trial entered as a covariate.

Primary analyses. GEE models were run separately for analyses examining whether emotion labeling (1) decreases negative emotion itself and (2) potentiates intentional emotion regulation (i.e., results in greater decreases in negative emotion while implementing emotion regulation strategies) for each emotion index (i.e., self-reported negative emotion, SCR, and RSA), leading to six primary study analyses. In order to examine whether emotion labeling decreases negative emotion itself, VAS scores from immediately after the EMOTION INDUCTION block, and SCR and RSA across four epochs of the block (i.e., four 30-second epochs across the 2-minute EMOTION INDUCTION block) were entered as outcomes. For analyses examining whether emotion labeling potentiates intentional emotion regulation, VAS scores from immediately after the INTENTIONAL EMOTION REGULATION BLOCK, and SCR and RSA across five epochs of the block (i.e., five 30-second epochs across the 2.5-minute INTENTIONAL EMOTION REGULATION block) were entered as outcomes. Given that individuals with BPD exhibit elevated baseline negative emotion intensity (Kuo, Fitzpatrick, Metcalfe, & McMain, 2016), we entered mean baseline emotion (i.e., self-report, SCR, or RSA for self-report, SCR, and RSA analyses, respectively) from the 5-minute baseline immediately

preceding each emotion induction as a covariate for the analyses examining whether labeling directly decreases negative emotion. In order to avoid conflating elevated negative emotion in general with a lack of intentional emotion regulation, mean emotion (i.e., self-report, SCR, or RSA for self-report, SCR, and RSA analyses, respectively) from the 2-minute emotion induction immediately preceding each INTENTIONAL EMOTION REGULATION block was entered as a covariate for the intentional emotion regulation analyses. Next, self-reported dissociation from each trial and alexithymia scores were entered as covariates. Group status (BPD versus HC) and condition (labeling versus control) were entered as predictors, followed by a group × condition interaction term. For the analyses examining the impact of emotion labeling on intentional emotion regulation, emotion regulation strategy (i.e., mindfulness versus cognitive reappraisal) was entered as a categorical predictor. A two-way condition × strategy interaction was entered to examine whether the effect of labeling on intentional emotion regulation varied across strategies, and a three-way condition × group × strategy interaction was added to examine whether the condition × strategy interaction differed across groups. All subsidiary two-way interactions required to build the three-way interaction were also added. All continuous predictors were grand mean-centered.

Results

Power analyses. We conducted power analyses in G*Power to determine the sample size required to identify a statistically significant condition × group interaction, given its primacy to study hypotheses. Few existing studies provided meaningful estimates for what an expected effect size for this interaction might be. To be conservative, we specified a small effect size (f = .10) for this interaction. We conducted power analyses for a repeated measures analysis of variance with a within-between interaction with two groups and 20 repeated measurements (i.e.,

five epochs per INTENTIONAL EMOTION REGULATION block across four trials). This analysis specified that a sample size of 48 (i.e., 24 individuals per group) would yield a power of .95 to detect a small effect size (f = 0.1). Therefore, we determined that a sample size of 60 (i.e., 30 individuals per group) would be sufficiently powered to detect this interaction.

Descriptive information. Descriptive statistics for the main study variables are presented in Table 3. The BPD group had statistically higher total alexithymia scores (mean = 56.33, standard deviation = 12.36) relative to the HC group (mean = 38.37, standard deviation = 11.48), t (58) = -5.85, $p < .001$. All participants listed at least one internal state in the LABEL condition and one external descriptor/object in the CONTROL condition except for one, whose data from the EMOTION INDUCTION and EMOTION REGULATION blocks were excluded from analyses. One participant listed external descriptors/objects instead of internal states in one of the LABEL conditions, and five participants listed internal states in the CONTROL conditions. Therefore, data from these individuals for the EMOTION INDUCTION and INTENTIONAL EMOTION REGULATION blocks of these trials were omitted from analyses.[2] Participants in the BPD group reported attempting to follow the instructions in the LABEL-MINDFULNES, LABEL-REAPPRAISAL, CONTROL-MINDFULNESS, and CONTROL-REAPPRAISAL conditions 83.43% (SD = 16.82), 81.00% (SD = 18.64), 84.76% (SD = 14.77), and 81.33% (SD = 20.08) of the time, respectively. Participants in the HC group reported attempting to follow the

[2] One participant wrote "pain" amidst a long list of external descriptors/objects in one of the CONTROL condition trials. It was unclear whether this term was referring to their internal state or something that they imagined observing in the script, which was about a funeral. Given that this term was only one in a long list of external descriptors/objects, it was retained in analyses.

instructions in the LABEL-MINDFULNES, LABEL-REAPPRAISAL, CONTROL-MINDFULNESS, and CONTROL-REAPPRAISAL conditions 86.30% (SD = 13.32), 87.77% (SD = 12.72), 81.17% (SD = 18.87), and 82.63% (SD = 22.22) of the time, respectively. These two groups did not differ in their reported adherence to the instruction across any condition (*p*-values range from .11 to .81). Average dissociation was not significantly correlated with average SCR, r (57) = .11, p = .39, or average RSA, r (57) = .04, p = .74. However, it was significantly positively correlated with average self-reported negative emotion, r (57) = .57, $p < .001$, supporting its inclusion as a covariate.

Manipulation Check

There were statistically significant main effects of block (baseline to induction) for self-reported negative emotion (B = 25.39, *SE* = 2.84), Wald $\chi^2(1)$ = 80.18, $p < .001$ and SCR, (B = .71, *SE* = .07), Wald $\chi^2(1)$ = 116.59, $p < .001$, and a trend towards a main effect of block for RSA, (B = -.11, *SE* = .06), Wald $\chi^2(1)$ = 3.13, p = .08. These findings indicated that the emotion induction resulted in increases in negative emotion from across indices (trend for RSA), groups, and conditions, and was therefore successful.

Does Emotion Labeling Decrease Negative Emotion?

Self-report. Primary study analyses examining the role of emotion labeling on negative emotion and intentional emotion regulation are presented in Tables 4 and 5, respectively. There was a trending but ultimately not statistically significant condition × group interaction on self-reported negative emotion during the emotion induction. The main effect of condition on self-reported negative emotion during the emotion induction was also not statistically significant.

Sympathetic responses. There were no main effects of condition, or a condition × group interaction on the number of SCRs across 30-second epochs during the emotion induction.

Parasympathetic responses. There was a statistically significant condition × group interaction on RSA during the emotion inductions. Post-hoc simple effects tests indicated that the HC group did not exhibit differences in RSA between the label and control conditions, Wald $\chi^2(1) = .67$, $p = .41$, but the BPD group exhibited higher RSA (i.e., increased activation of emotion regulatory systems during the emotion inductions) in the label condition than the control condition (estimated marginal mean difference = .13, SE = .06), Wald $\chi^2(1) = 4.51$, $p = .03$.

Does Emotion Labeling Potentiate Intentional Emotion Regulation?

Self-report. There was a statistically significant condition × group interaction. Post-hoc simple effects tests indicated that, across intentional emotion regulation strategies, the HC group reported lower self-reported negative emotion (i.e., greater emotion regulation) following the label than the control condition (estimated marginal mean difference = 4.89, SE = 1.89), Wald $\chi^2(1) = 6.69$, $p = .01$, but the BPD group did not exhibit differences following the label or control conditions in self-reported negative emotion when implementing intentional emotion regulation strategies, Wald $\chi^2(1) = .91$, $p = .34$. There was no statistically significant condition × group × strategy interaction on self-reported intentional emotion regulation.

Sympathetic responses. There were no statistically significant main effects of condition, condition × group, or condition × group × strategy interactions on the number of SCRs across 30-second epochs during intentional emotion regulation strategy use.

Parasympathetic responses. There were no statistically significant main effects of condition or condition, condition × group, or condition × group × strategy interactions on RSA intentional emotion regulation.[3]

Discussion

This study was the first to examine the effects of labeling one's own emotion on negative emotion and intentional emotion regulation strategy use across self-reported, sympathetic, and parasympathetic indices of negative emotion in a population with BPD. Findings suggested that the effect of emotion labeling varied based on the emotion index and the population (i.e., BPD or HC). Specifically, emotion labeling increased parasympathetic incidental emotion regulation in BPD, and self-reported intentional emotion regulation in HCs.

Does Emotion Labeling Regulate Negative Emotion?

We hypothesized that individuals with BPD and HCs would experience a decrease in self-reported, sympathetic, and parasympathetic negative emotion following emotion labeling relative to the control condition. Further, we hypothesized that individuals with BPD would benefit less from emotion labeling than HCs. Partially consistent with hypotheses, emotion labeling resulted in higher RSA, but only in the BPD group, during its implementation compared to the control task, suggesting that emotion labeling is effective in activating the parasympathetic system in BPD. Theory suggests that BPD is characterized by emotion regulation deficits (Linehan, 1993) and increases in RSA reflect the activation of emotion regulatory systems (Beauchaine, 2001; Stange, Hamilton, Fresco, & Alloy, 2017). Thus, perhaps emotion labeling

[3] We re-ran study analyses without the dissociation and alexithymia covariates, and the statistical significance of main effects and interactions that are pertinent to primary study questions did not change.

activates emotion regulatory systems specifically in those who have pre-existing emotion regulation deficits, such as those with BPD. Such theorizing is consistent with neural research showing that emotion labeling activates emotion regulatory regions which then subsequently downregulate the amygdala (e.g., Lieberman et al., 2007; Torrisi et al., 2013). In contrast, emotion labeling may have little to no impact on emotion regulatory systems in those who are already regulating emotions effectively, as HCs may be.

In contrast with our hypotheses, there were no differences between the emotion labeling and control tasks with respect to self-reported or sympathetic indices of negative emotion. Such differential effects underscore the independence of emotion domains (Lang, 1988; Mauss & Robinson, 2009) and the importance of studying the effects of emotion labeling across them. Although inconsistent with theory (e.g., Lieberman et al., 2011), the lack of effect of emotion labeling on self-reported and sympathetic negative emotion domains in both groups is consistent with research suggesting that emotion labeling did not differ from intentional emotion regulation strategies in reducing sympathetic negative emotion (Ortner, 2015). Our finding, in concert with such prior work, suggests that the emotion regulatory effect of emotion labeling on these domains may not be universal but rather may depend on *how* the emotion is labeled. Several past works that documented the emotion regulatory effect of emotion labeling asked participants to label emotional displays rather than their own emotions (Constantinou, Van Den Houte, Bogaerts, Van Diest, & Van den Bergh, 2014; Lieberman, Inagakim Tabibnia, & Crockett, 2011). Labeling others' emotion may serve as a distraction that allows individuals to disengage from their own, producing decreases in negative emotion that are distinct from the regulatory effect (or lack thereof) of emotion labeling. Compared to labeling other's emotion, labeling one's

own may simply not be as effective in downregulating self-reported or sympathetic negative emotion.

Alternatively, the emotion regulatory impact of emotion labeling may take longer to unfold (e.g., minutes or hours) in subjective and sympathetic negative emotion domains than the brief two-minute window for it provided. According to the process model of emotion regulation (Gross, 1998) and current basic emotion research (Sheppes & Gross, 2011; Thiruchselvam et al., 2011), strategies that involve engaging with emotion may not act as quickly to reduce it than strategies that involve disengaging with it. Since emotion labeling involves engaging with emotion, the process through which negative emotion then decreases may take longer to occur. Although other studies have shown that emotion labeling effectively reduces emotions in even briefer time frames than that of the present study, these works either asked participants to label external stimuli (e.g., Lieberman et al., 2007; Torrisi et al., 2013) or to select a label from a list of three words (Burklund et al., 2014). Generating labels for one's own emotion may be a slower process that requires more time to influence negative emotion, which would account for why other studies that asked participants to label their own emotion have found that emotion labeling either does not impact or increases negative emotion (McCrae et al., 2010; Ortner et al., 2015). Assessing the impact of labeling one's own emotion over longer periods of time would be pertinent to fully understanding its effect on specific emotion domains. Finally, it is possible that our emotional scripts were not sufficiently evocative to illuminate differences between emotion labeling and control tasks. Future work should examine the labeling of emotion of varying intensities to understand whether certain emotional contexts moderate its impact.

Does Emotion Labeling Potentiate Intentional Emotion Regulation?

We also hypothesized that emotion labeling would potentiate the effectiveness of intentional emotion regulation strategies (mindfulness and cognitive reappraisal) relative to the control task. Our findings suggested that the impact of emotion labeling, or lack thereof, was similar across intentional emotion regulation strategies. Self-report findings for HCs supported our hypothesis, as HCs reported lower negative emotion during intentional emotion regulation strategy use following emotion labeling compared to following the control task. Consistent with exposure-based research (Kircanski et al., 2012; Niles et al., 2015), our findings thus suggest that, in HCs, emotion labeling potentiates the effectiveness of intentional emotion regulation.

It is notable that the potentiating effect of emotion labeling on self-reported negative emotion was present in the HC group only, and for the self-report domain only. HCs may simply be more effective at implementing intentional emotion regulation strategies, and, perhaps, emotion labeling "refines" a strategy that is already mastered. If individuals with BPD cannot master intentional emotion regulation strategies to the same extent as HCs, they may not yet be able to refine and improve their efficacy through the use of incidental emotion regulation, needing to focus instead on its implementation. Related, the potentiating effect of emotion labeling may be specific to individuals with low to moderate self-reported negative emotion intensities. Research suggests the efficacy of intentional emotion regulation strategies that involve engaging with emotion content deteriorates as negative emotion intensity increases (e.g., Sheppes & Gross, 2011; Sheppes, Scheibe, Suri, & Gross, 2011; Sheppes et al., 2014). Incidental emotion regulation that involves engaging with emotion content, such as emotion labeling, may similarly be deleteriously impacted by high negative emotional intensity. The higher self-reported negative emotion intensity that characterizes BPD (e.g., Kuo et al., 2016) may thus block its capacity to potentiate intentional emotion regulation in this group. Finally, it's possible

that emotion labeling may potentiate self-reported incidental emotion regulation in the BPD group, but the lower emotional awareness associated with BPD (Derks, Westerhof, & Bohlmeijer, 2017) obstructed their ability to notice and report such changes.

It is also notable that neither group exhibited differences in physiological indices of intentional emotion regulation as a function of emotion labeling. Perhaps emotion labeling does not influence physiological intentional emotion regulation. Alternatively, perhaps the physiological effects of labeling one's own emotion on subsequent emotion regulation take longer to unfold than the time period examined in the present study. Although labeling one's own emotion may potentiate intentional emotion regulation in the self-reported domain immediately, its effects on physiological domains may occur over minutes to hours, or even weeks to months, following repeated use. Future research should examine the time course of labeling one's own emotion across emotion domains, and whether it differs from that of labeling external stimuli. It is also possible that the emotion inductions utilized in the present study were not sufficiently evocative and thus did not present an emotion regulatory "challenge" for participants. Perhaps emotion labeling only potentiates physiological intentional emotion regulation in situations where negative emotion is particularly intense, which may not have occurred in our experimental setting. Future research should examine the impact of emotion labeling on intentional emotion regulation in participant's daily life and over longer time periods.

Limitations, Implications, and Future Directions

There are a number of important limitations within this study. The sample size of 30 individuals per group is small. It is possible that more group differences in the impact of emotion labeling would be apparent with a larger sample size and more statistical power, and future research should investigate this. In addition, this study allocated only a two-minute period for

participants to engage in emotion labeling and a two-and-a-half minute period for intentional emotion regulation. It is possible that this brief window did not allow for a full assessment of emotion labeling, emotion regulation, and the impact of emotion labeling on intentional emotion regulation, and future work should thus extend the time frames utilized in the present study. Related, the potential effects of emotion labeling over the long-term (i.e., after repeated use over days and months) remain unclear and more directly reflect its use in therapeutic contexts. Longitudinal research should study the effect of emotion labeling to provide clinically-relevant information about the impact of this strategy over time. As well, given that emotion labeling was always followed by the implementation of an intentional emotion regulation strategy, the subsequent effects of emotion labeling by itself over time remain unclear and should be examined in future work. Furthermore, given that participants were told that they would hear emotionally evocative scripts prior to the experiment, they may have over-reported their negative emotion in response to the emotional scripts across conditions due to demand characteristics. Although we attempted to mitigate this by emphasizing that participants should provide accurate responses even if they reflect positive, no, or neutral emotion, such demand characteristics may be particularly prevalent in the labeling condition given that participants are directly told to focus on emotional responses. Furthermore, although our control condition controlled for the act of typing and interacting with emotion induction content without emotion labeling, our study would have been improved by the addition of a control condition that did not involve engaging in any tasks whatsoever. It is possible that the control condition in the present study served to distract participants from the emotional components of the scripts and therefore masked potential emotion regulatory effects of emotion labeling. Future studies should incorporate control

conditions that do not instruct participants to engage in any tasks in order to better isolate the effect of emotion labeling.

Our study is strengthened by its use of an externally valid emotion labeling methodology that asks participants to label their own emotions rather than that of external stimuli. However, such a methodology also entails potential compromises to internal validity, as it is unclear how to measure task performance (i.e., how can the performance of emotion labeling be measured?). Indeed, it is possible that the BPD group did not engage in emotion labeling as accurately or competently as HCs. Future research should attempt to continue to refine methodologies of coding the accuracy of emotion labels to help identify whether there are group differences in emotion labeling competency. Finally, the lack of a clinical control group prohibits conclusions about whether observed group differences are specific to BPD pathology or pervasive across groups with psychopathology, which is particularly compounded by the fact that there were several comorbidities in the BPD group. Clinical control groups in future research could help to clarify whether group differences are specific to BPD, some of its comorbidities, or psychopathology more broadly.

Clinical Implications and Conclusion

Although this study's limitations are notable, there are several key clinical implications of the present findings. Namely, our findings suggest that emotion labeling activates emotion regulatory systems among individuals with BPD. They also suggest that, in HCs, emotion labeling can potentiate the effectiveness of intentional emotion regulation in some domains (i.e., self-report), but not necessarily others. These preliminary findings encourage the use of emotion labeling in BPD treatments such as DBT, but also suggest that its effects may be restricted to parasympathetic domains of emotion and more research is needed to support the extent to which

it is currently used. The discrepancy between self-report and physiological findings further underscores the importance of comprehensively studying negative emotion processes such as emotion labeling across several independent domains. It also suggests that clinicians should assess the impact of strategies such as emotion labeling in ways that extend beyond client's subjective report, as these strategies may affect other (physiological) domains. Further research is also needed to clarify whether the efficacy of emotion labeling is dependent on specific moderators, such as the emotional intensity or emotion labeled. Taken together, our results collectively indicate that the impact of emotion labeling is nuanced and specific to the negative emotion domain studied and the group employing it. Despite these nuances, this study adds to a growing literature suggesting that emotion labeling holds some promise as a therapeutic strategy.

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Table 1

Participant demographics

		Healthy controls	Borderline personality disorder
Age [Mean (Standard deviation)]		23.37 (5.01)	24.17 (4.62)
Sex			
	Female	86.7%	86.7%
	Male	13.3%	13.3%
Gender identity			
	Woman	86.7%	76.7%
	Man	13.3%	16.7%
	Gender queer	0%	6.7%
Ethnicity			
	Other Asian or Asian-Canadian	33.3%	20%
	Chinese or Chinese-Canadian	23.3%	23.3%
	Black-Canadian/Black/Caribbean Origin	13.3%	3.3%
	White/Caucasian/European Origin	13.3%	36.7%
	Other Hispanic	6.7%	0%
	Mexican or Mexican-Canadian	3.3%	0%
	Middle Eastern	3.3%	3.3%
	Bi-Racial/Multi-Racial	3.3%	6.7%
	Korean or Korean-Canadian	0%	3.3%

	Other	0%	3.3%
Level of Education			
	High school graduate	10%	6.7%
	Some college/university	36.7%	43.3%
	College diploma	6.7%	3.3%
	Undergraduate degree	43.3%	40%
	Master’s degree	3.3%	6.7%
Marital Status			
	Single/Never married	80%	50%
	Dating/Never married	13.3%	43.3%
	Married/Common law/Life partner	6.7%	6.7%

Table 2

Current and past co-morbid participant diagnoses within the borderline personality disorder group

	Current	Past
Major depressive disorder	23%	57%
Dysthymic disorder	20%	
Bipolar II disorder	7%	0%
Other bipolar disorder	0%	5%
Substance induced mood disorder	3%	0%
Brief psychotic disorder	0%	2.5%
Psychotic disorder not otherwise specified	0%	0%
Alcohol abuse	3%	7%
Alcohol dependence	3%	24%
Substance abuse	3%	10%
Substance dependence	0%	10%
Panic Disorder	10%	6%
Agoraphobia	3%	7%
Agoraphobia without a history of panic disorder	3%	4%
Social anxiety disorder	43%	14%
Specific phobia	17%	3%
Obsessive compulsive disorder	23%	20%

Posttraumatic stress disorder	27%	10%
Generalized anxiety disorder	50%	0%
Anorexia nervosa	3%	7%
Bulimia nervosa	2%	3%
Binge eating disorder	1%	0%
Eating disorder not otherwise specified	1%	0%

Table 3

Means and standard deviations (in brackets) for primary study variables across conditions, blocks, and groups

Emotion induction condition	Intentional emotion regulation strategy	Block	Self-report		SCR		RSA		DSS	
			BPD	HC	BPD	HC	BPD	HC	BPD	HC
Label										
	Mindfulness								26.10 (28.80)	8.20 (15.65)
		Baseline	20.80 (19.68)	2.11 (3.18)	1.41 (1.96)	1.77 (2.87)	6.55 (1.34)	6.69 (1.21)		
		Emotion induction	42.96 (23.92)	33.21 (31.75)	2.80 (2.09)	3.24 (3.04)	6.39 (1.20)	6.60 (1.22)		
		Emotion regulation	31.25 (22.10)	18.05 (25.88)	2.10 (2.48)	1.73 (2.33)	6.63 (1.27)	6.80 (1.20)		
Label										
	Reappraisal								24.46 (30.89)	5.50 (10.16)
		Baseline	19.52 (19.78)	1.83 (3.15)	1.68 (2.57)	1.37 (1.88)	6.46 (1.50)	6.83 (1.22)		

	Emotion induction	43.30 (25.22)	31.83 (28.65)	2.86 (2.29)	2.95 (2.57)	6.56 (1.28)	6.57 (1.03)		
	Emotion regulation	30.26 (24.28)	9.44 (10.54)	1.89 (2.35)	1.91 (2.45)	6.56 (1.52)	6.74 (1.15)		
Control									
Mindfulness								25.82 (32.42)	9.33 (18.57)
	Baseline	20.89 (20.61)	2.02 (3.34)	1.53 (2.01)	1.47 (2.32)	6.51 (1.36)	6.74 (1.33)		
	Emotion induction	43.91 (24.13)	28.07 (30.21)	3.21 (2.47)	3.30 (3.12)	6.50 (1.17)	6.55 (1.18)		
	Emotion regulation	31.09 (21.47)	18.75 (26.67)	2.54 (3.18)	1.59 (2.25)	6.75 (1.36)	6.80 (1.13)		
Control									
Reappraisal								23.29 (31.52)	8.38 (16.51)
	Baseline	20.68 (19.81)	1.89 (4.28)	1.73 (2.48)	1.13 (1.98)	6.63 (1.44)	6.74 (1.26)		
	Emotion induction	40.51 (28.27)	25.41 (27.36)	3.25 (2.55)	3.11 (3.30)	6.48 (1.37)	6.62 (1.11)		
	Emotion regulation	24.93 (19.90)	11.43 (16.83)	2.64 (3.12)	2.03 (2.43)	6.46 (1.27)	6.83 (1.12)		

Note. BPD = borderline personality disorder; HC = healthy controls; SCR = skin conductance responses; RSA = respiratory sinus arrhythmia; DSS = Dissociative State Scale (Stiglmayr et al., 2001). Self-report reflects the average intensity of negative emotions ranging from 0 to 100. SCRs reflect the average number of skin conductance responses per 30-second epoch. RSA is in $ms^{2.}$

Table 4

Generalized estimating equations analyses examining the impact of emotion labeling on emotion reactivity

		B	Standard error	Wald chi-square	Degree of freedom	p-value
Self-Report						
	Intercept	**32.41**	**4.59**	**184.06**	**1**	**<.001**
	Dissociation	**.45**	**.09**	**25.37**	**1**	**<.001**
	Alexithymia	.12	.25	.22	1	.64
	Baseline Self-Report	.18	.11	2.78	1	.10
	Condition	6.93	3.59	1.81	1	.18
	Group	4.92	7.39	.02	1	.89
	Condition × Group	-7.76	4.55	2.91	1	.09
SCR						
	Intercept	**1.23**	**.15**	**246.22**	**1**	**<.001**
	Dissociation	.00	.00	.01	1	.94
	Alexithymia	.01	.01	1.28	1	.26
	Baseline SCR	.07	.04	2.85	1	.09
	Condition	-.06	.09	2.58	1	.11
	Group	-.14	.22	.88	1	.35
	Condition × Group	-.11	.14	.62	1	.43
RSA						
	Intercept	**6.55**	**.09**	**16508.01**	**1**	**<.001**

Dissociation	-.00	.00	2.65	1	.10
Alexithymia	.00	.01	.76	1	.38
Baseline RSA	**.76**	**.06**	**190.77**	**1**	**<.001**
Condition	-.06	.07	.48	1	.49
Group	-.08	.15	.01	1	.92
Condition × Group	**.19**	**.09**	**4.04**	**1**	**.04**

Note. Significant effects are bolded. Condition = labeling versus control condition; SCR = skin conductance responses; RSA = respiratory sinus arrhythmia.

Table 5

Generalized estimating equations analyses examining the role of potentiating labeling on intentional emotion regulation

		B	Standard error	Wald chi-square	Degree of freedom	p-value
Self-Report						
	Intercept	**24.18**	**2.37**	**409.09**	**1**	**<.001**
	Dissociation	**.23**	**.08**	**9.74**	**1**	**.002**
	Alexithymia	-.04	.11	.18	1	.67
	Induction Self-Report	**.54**	**.04**	**164.06**	**1**	**<.001**
	Condition	-3.20	2.07	.74	1	.39
	Group	.76	3.88	3.33	1	.07
	Strategy	**-5.70**	**2.80**	**7.28**	**1**	**.01**
	Condition × Group	**3.80**	**2.97**	**4.58**	**1**	**.03**
	Group × Strategy	2.72	3.65	2.05	1	.15
	Condition × Strategy	-1.54	2.70	.04	1	.85
	Condition × Group × Strategy	3.78	3.75	1.02	1	.31
SCR						
	Intercept	**1.57**	**.35**	**140.88**	**1**	**<.001**
	Dissociation	.00	.01	.05	1	.83
	Alexithymia	.01	.03	.26	1	.61
	Induction SCR	**.42**	**.09**	**24.31**	**1**	**<.001**

	Condition	.20	.36	1.04	1	.31
	Group	.88	.85	.11	1	.75
	Strategy	.66	.33	.92	1	.34
	Condition × Group	-.54	.52	1.95	1	.16
	Group × Strategy	**-.78**	**.51**	**4.71**	**1**	**.03**
	Condition × Strategy	-.26	.42	.44	1	.51
	Condition × Group × Strategy	.09	.65	.02	1	.89
RSA						
	Intercept	**6.79**	**.09**	**16458.23**	**1**	**<.001**
	Dissociation	.00	.00	.38	1	.54
	Alexithymia	.00	.01	.01	1	.94
	Induction RSA	**.89**	**.05**	**309.41**	**1**	**<.001**
	Condition	-.05	.10	.16	1	.69
	Group	.00	.19	.51	1	.47
	Strategy	**-.05**	**.10**	**9.51**	**1**	**.002**
	Condition × Group	.01	.16	.03	1	.87
	Group × Strategy	**-.26**	**.15**	**5.92**	**1**	**.02**
	Condition × Strategy	.03	.16	.10	1	.75
	Condition × Group × Strategy	.02	.24	.01	1	.92

Note. Significant effects are bolded. Condition = labeling versus control condition; SCR = skin conductance responses; RSA = respiratory sinus arrhythmia.

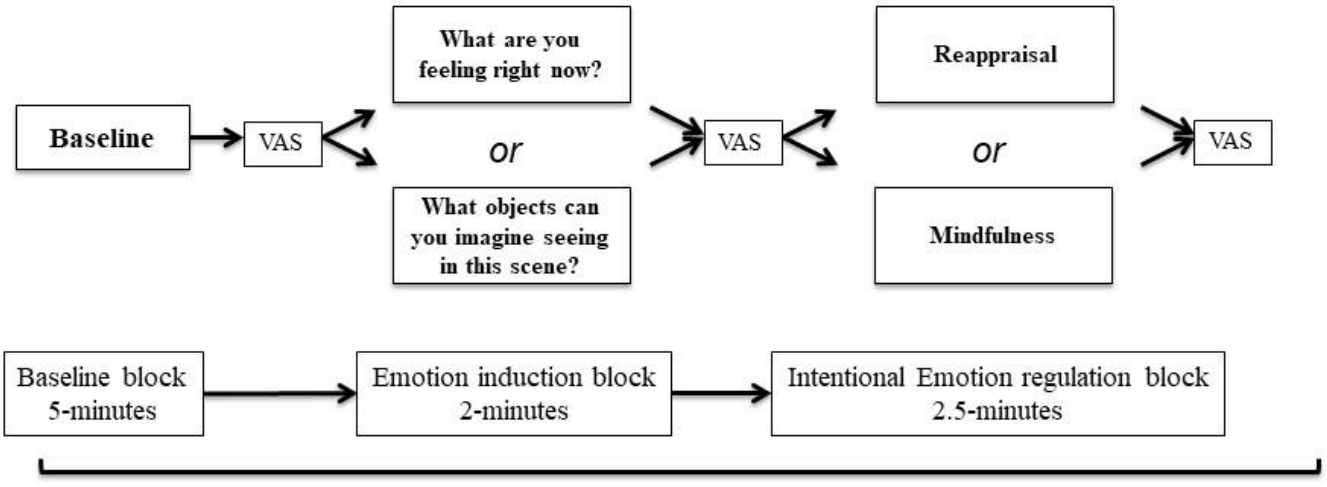

Figure 1. Visual depiction of an experimental trial.

Note. VAS = Visual Analogue Scale