RUNNING HEAD: Sleep and emotion dysregulation in BPD

The influence of sleep on emotion dysregulation in borderline personality disorder, generalized anxiety disorder, and healthy controls

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1. Introduction

Borderline personality disorder (BPD) is a debilitating disorder characterized by high rates of suicide attempts and deaths (Goodman et al., 2017). Theories suggest that BPD is characterized by emotion dysregulation (i.e., disruptions to emotion processes as well as difficulties modulating them; Linehan, 1993), which is a key target and mechanism of frontline BPD treatments (e.g., Neacsiu et al., 2010; Stepp et al., 2008). Optimizing BPD treatment may therefore involve identifying and targeting the factors contributing to emotion dysregulation.

1.1 Emotion dysregulation and BPD

Linehan's (1993) Biosocial theory outlines several components of emotion dysregulation including sensitivity to emotionally-salient stimuli, heightened emotional reactivity (i.e., larger changes in emotional intensity following provocation), delayed recovery of emotions, and emotion regulation deficits (i.e., difficulties altering emotion on purpose or automatically; Gross & Thompson, 2007). Several studies also suggest that BPD is characterized by higher baseline negative emotion compared to healthy (HC) and clinical control groups across self-report (e.g., Elices et al., 2012; Fitzpatrick et al., 2020a; Gratz et al., 2010; Kuo & Linehan, 2009; Kuo et al., 2016), parasympathetic (Kuo & Linehan, 2009; Kuo et al., 2016), and sympathetic (e.g., Fitzpatrick et al., 2020a) indices.

Studies are mixed with respect to the presence of heightened emotional reactivity in BPD, with evidence of both heightened and comparable self-reported, sympathetic, or parasympathetic emotion in BPD versus control groups (e.g., Austin et al., 2007; Dixon-Gordon et al., 2013; Elices et al., 2012; Gratz et al., 2019; Fitzpatrick et al., 2020a; Kuo et al., 2016; Rosenthal et al.,

2016). Findings may be mixed because heightened emotional reactivity in BPD may be specific to *interpersonal*, rather than non-interpersonal, stressors (e.g., Limberg et al., 2011).

In addition and contrary to Linehan's (1993) theory, several studies suggest that individuals with BPD can decrease self-reported, sympathetic, and parasympathetic negative emotion comparably to HCs and clinical control groups using emotion regulation strategies such as distraction and mindful awareness (e.g., nonjudgmentally noticing, allowing, and observing emotional responses; Kabat-Zinn, 1990) (e.g., Fitzpatrick et al., 2020a; Krause-Utz et al., 2018; Kuo et al., 2016). However, given the elevated baseline negative emotion that characterizes BPD, those with BPD continue to exhibit high negative emotion relative to controls even after "successful" emotion regulation (Kuo et al., 2016). In sum, BPD is consistently characterized by elevated negative baseline emotion, potentially heightened reactivity to interpersonal stressors, and an intact capacity to regulate emotion that may require further strengthening. It is essential to identify what factors influence these emotion dysregulation components so they can be strategically targeted.

1.2. Sleep and emotion dysregulation

Sleep is pertinent to healthy emotion and emotion regulation and serves a "reset" function for the networks that regulate emotional activity in the brain. Accordingly, sleep problems influence a range of emotion dysregulation components (e.g., see Gruber & Cassoff, 2014, for review). Sleep is ultimately influenced by two semi-independent processes. The *homeostatic system* results in the building of a pressure to sleep (i.e., sleep drive) during increasing wakefulness and activity (Borbély, 1982; Webb, 1988). Poor sleep efficiency (SE; the percentage of time spent actually sleeping while in bed) can disrupt homeostatic sleep by diminishing healthy accumulation of sleep drive and resulting in a conditioned association between

wakefulness and sleep stimuli (e.g., the bed; Spielman et al., 1987). The c*ircadian* s*ystem* reflects biological imperatives to sleep and wake at specific times for optimal sleep (e.g., "body clock"; Borbély, 1982; Webb, 1988). However, it remains unclear which of these specific sleep domains (if any) influence emotion dysregulation in BPD, and which specific emotion dysregulation components are impacted.

1.2.1. Homeostatic processes and emotion dysregulation

Although people with BPD exhibit insomnia-related difficulties (Selby, 2013), studies are mixed with respect to whether BPD involves disrupted homeostatic sleep processes as meta-analyses suggest that individuals with BPD have low SE relative to HCs but similar or high SE relative to clinical controls (e.g., Winsper et al., 2017). However, poor homeostatic sleep processes such as low SE are associated with some components of emotion dysregulation. In adolescents, lower sleep duration is associated with elevated negative mood, reduced positive affect, and a parent-rated measure of emotion dysregulation that comprised several components (i.e., general emotion dysregulation; Becker et al., 2020; Goodhines et al., 2020). Further, *both low and high* amounts of time in bed are associated with elevations on a self-report measure of a range of emotion dysregulation components in elementary students (Rubens et al., 2017). In depressed and healthy groups, lower SE predicts higher emotional reactivity specifically to daily life events (O'Leary et al., 2017). Finally, among those with BPD (but not HCs or individuals with generalized anxiety disorder), *higher* SE predicts higher general emotion dysregulation (Fitzpatrick et al., 2020b). Homeostatic sleep processes such as SE may thus be associated with emotion dysregulation, but the direction of this relationship may be curvilinear. Moreover, most of the studies examining the relationship between homeostatic sleep processes and emotion

dysregulation focus on self- or informant-report measures of general emotion dysregulation, rather than its specific components.

1.2.2. Circadian processes and emotion dysregulation

Little research has investigated circadian processes in BPD. Some studies suggest that adolescents with BPD have later rise times than HCs (Huynh et al., 2016) and adults with BPD have later rise times than those with generalized anxiety disorder but not HCs, although other indices of chronotypes do not differ across these groups (Fitzpatrick et al., 2020b). Regardless of chronotype variability in BPD, there is evidence that having a later chronotype may be associated with elevations in some emotion dysregulation components. Studies in healthy samples show that later chronotypes are associated with higher amygdala reactivity to fearful stimuli and reduced functional connectivity between the amygdala and dorsal anterior cingulate cortex, suggesting poorer emotion regulation (Horne & Norbury, 2018). Conversely, one experimental study in undergraduates suggested that chronotype did not predict self-reported or parasympathetic emotion regulation (Taylor et al., 2020). Further, chronotype did not predict self-reported general emotion dysregulation in BPD, HC, or a generalized anxiety disorder groups (Fitzpatrick et al., 2020b). As evidenced by the mixed findings reviewed, it is possible that chronotypes influence some domains of emotion dysregulation (e.g., emotional reactivity) and not others (e.g., emotion regulation). Whether and which specific domains are influenced by chronotype in BPD, and if these relationships are unique to BPD or pervasive across healthy and clinical groups, is unknown.

1.2.3. Sleep quality and emotion dysregulation

Finally, it is possible that it is the subjective experience of sleep that is particularly impactful to emotion dysregulation. Indeed, people with BPD report low sleep quality (SQ)

relative to HCs (Semiz et al., 2008) and poor SQ has been associated with elevated baseline negative emotional reactivity in both clinical and healthy samples (Bouwmans et al., 2017; Kaurin et al., 2022; Simor et al., 2015; Toschi et al., 2021). Moreover, experimental research indicates that low SQ predicts less capacity to regulate self-reported sadness in a community sample (Mauss et al., 2013). SQ may thus exacerbate emotional reactivity and emotion regulation elements of emotion dysregulation. However, the impact of SQ on specific self-reported and physiological components of emotion dysregulation in BPD is unclear, as is the potential specificity of these relationships to BPD.

1.3. The present study

In summary, homeostatic, circadian, or subjective sleep components may be associated with BPD and influence emotion dysregulation. However, it remains unclear which sleep components influence particular emotion dysregulation components, and whether these relationships are specific to BPD or pervasive across healthy and clinical groups. The present study therefore aimed to determine whether specific homeostatic (i.e., SE), circadian (i.e., chronotype), and subjective (i.e., SQ) sleep components predict unique elements of emotion dysregulation in BPD, HCs, and a clinical control group (i.e., generalized anxiety disorder). Individuals with generalized anxiety disorder (GAD) were selected as the clinical control group because, like those with BPD, this group is also characterized by elevated emotion dysregulation (Mennin et al., 2005) and sleep disruptions (Tsypes et al., 2013). The inclusion of this group therefore facilitates the identification of whether the relationships under study are specific to those with BPD or pervasive across groups with emotion dysregulation and/or a high frequency of sleep disruptions. The emotion dysregulation elements studied were baseline negative emotion, emotional reactivity, and emotion regulation using two strategies emphasized in BPD

treatments (mindful awareness and distraction; Linehan, 1993), and were measured comprehensively across self-reported, sympathetic, and parasympathetic emotion domains. We hypothesized that low and high SE, later chronotypes, and poorer SQ would predict elevated emotion dysregulation across components. Given a dearth of literature, we considered examinations regarding whether these effects were specific to BPD or pervasive across clinical and HC groups to be exploratory.

2. Method

This study utilized data collected as part of a broader study examining emotion processes (Fitzpatrick et al., 2020a) and sleep processes (Fitzpatrick et al., 2020b) in BPD.

2.1. Participants

See Table 1 for demographic and diagnostic information about the sample. Participants with BPD (n = 40), GAD (n = 40), and HCs (n = 40; total N = 120) were recruited from online and physical advertisements. Exclusion criteria are detailed elsewhere (Fitzpatrick et al., 2020a) but involved medical conditions or taking medications that could interfere with task completion/physiological recording, bipolar I disorder, DSM-IV-TR current alcohol or drug dependence (APA, 2000), or severe psychotic-spectrum disorders. Participants in the GAD or HC groups were excluded if they met 4+ diagnostic BPD criteria, or the self-harm/suicidality or impulsivity BPD criterion. Finally, HCs were also excluded if they met full diagnostic criteria for any current psychiatric disorder. The groups did not statistically differ in age or sex.

2.2. Measures

The *International Personality Disorders Examination- BPD Module* (IPDE; Loranger et al., 1994) is a gold standard semi-structured interview that was used to assess for BPD, which correlates highly and in expected directions with other BPD measures (Schroeder et al., 2010).

The *Structured Clinical Interview for DSM-IV-TR disorders* (SCID-IV-TR; First et al., 1995) was used to assess for the presence of exclusion criteria and GAD, and to characterize the study sample. The prevalence-adjusted bias-adjusted kappas (PABAKs; Byrt et al., 1993) between assessors and the gold standard for the IPDE and SCID-IV-TR ranged from .67 to 1.00, with an average PABAK of .95 and .97, respectively. *The Duke Structured Interview for Sleep Disorders-Insomnia Module* (DSISD; Edinger et al., 2009) was administered to assess for the presence of insomnia, and reliability for this measure is typically strong (Carney et al., 2009).

2.2.1. Sleep predictors

2.2.1.1. Sleep efficiency and quality

SE and SQ were measured via the Consensus Sleep Diary (CSD; Carney et al., 2012); a daily sleep diary that participants complete as soon as they get out of bed about the sleep episode they just had including when they got into bed, tried to fall asleep, woke up, got out of bed, and how long it took them to fall asleep. SE is calculated by dividing total time spent sleeping by total time spent in the bed per sleep episode. SQ is measured via a single item that asks participants to rate their quality of the past night's sleep on a five-point scale ranging from "very poor" to "very good." The CSD was administered for seven consecutive days leading up to the study experiment, which is usually a sufficient time period for stable estimates (Wohlgemuth et al., 1999).

2.2.1.2. Chronotype

Chronotype was measured via the Morningness-Eveningness Questionnaire (MEQ; Horne & Östberg, 1976); a 19-item measure that assesses individuals' proclivity towards morning versus evening chronotypes (α = .87). Items are summed with higher and lower scores indicating biological imperatives towards morningness and eveningness, respectively.

2.2.2. Emotion outcomes

All emotion outcomes were broken down into 30-second epochs. During phases wherein the last epoch did not equal exactly to 30 seconds, it was averaged into the prior epoch.

2.2.2.1. Self-report

Self-reported emotion was assessed via a rating dial, which allowed for a continuous, moment-to-moment measurement of positive and negative emotion. Participants were instructed to place their hand on the dial and to move it to reflect changes in their emotion. The dial ranged from 0 (very negative) to 9 (very positive).

2.2.2.2. Sympathetic emotion

Skin conductance responses (SCRs) were measured to reflect sympathetic emotion via electrodes on the non-dominant middle and index phalanges. Data were collected with a gain of 1000 at 1000 samples per second. High (.05 Hz) and low (35 Hz) pass filters were used to process SCR data, and a rolling filter edited artifacts. Using Mindware Technologies EDA 2.40 program, SCRs were counted as the number of skin responses in a 30-second epoch exceeding 0.05 μS.

2.2.2.3. Parasympathetic Emotion

Respiratory sinus arrythmia (RSA) was measured as a form of parasympathetic emotion using two electrodes and a respiratory band around the chest. R-R intervals within each 30-second epoch were calculated using Mindware Technologies HRV 2.33. RSA was calculated using spectral analysis which identified three frequency ranges in the electrocardiogram and used a validated algorithm to derive spectral densities in the highest one (>.15 Hz), indicative of parasympathetic influence (Berntson et al., 1997).

2.3. Procedure

Potentially eligible participants underwent diagnostic assessments and, if eligible, were instructed in the use of the CSD and to complete it for the seven days prior to the experiment. Participants were asked to avoid ingesting substances or caffeine on their experiment day.

On the experiment day, participants completed the MEQ, were instructed in the use of the rating dial, and connected to physiological equipment. Figure 1 in Fitzpatrick et al. (2020) provides a visual representation of the experimental paradigm. Participants underwent a 10-minute baseline phase wherein they sat staring at a black computer screen. The experiment followed and was a within-subjects design wherein participants underwent three trials that consisted of four phases each. The first phase in each trial was the "*Training Phase"*, wherein participants learned how to use an emotion regulation strategy in response to a prompt on the screen. For the first trial, participants were told to react as they usually would when they see a "REACT" prompt. In the second trial, participants were either instructed to either distract themselves from their emotions with neutral thoughts when they see the "DISTRACT" prompt, or notice and nonjudgmentally observe their emotions, thoughts, and body sensations without attempting to amplify or reject them when they see the "MINDFUL AWARENESS" prompt (e.g., Kuo et al., 2016). In the training phase of the third trial, participants were trained in the alternative strategy (DISTRACT or MINDFUL AWARENESS). Instruction phases involved research assistants reading from a standardized script that provided detail on how to implement the strategy, followed by clarifying any questions participants have. Research assistants then asked participants to explain their understanding of the strategy to them and provided corrective feedback as needed. Training phases lasted approximately five minutes.

Next, participants underwent another five-minute baseline (i.e., "*Trial-Baseline Phase"*) followed by the "*Emotion Induction Phase"* wherein they listened to an emotionally evocative

story through headphones. Each story lasted approximately two minutes and centered around themes of rejection which is particularly evocative to individuals with BPD (e.g., Limberg et al., 2011) (see Fitzpatrick et al., 2020a and https://osf.io/cqaj2/ for more information). Participants then underwent the "*Emotion Regulation Phase."* In the first trial of this phase, participants were presented with the "REACT" prompt (data from the emotion regulation phase of the REACT condition were not used in this manuscript). In the emotion regulation phase of the second and third trials, individuals were prompted to either "DISTRACT" or use "MINDFUL AWARENESS." Participants repeated a trial with all four phases (training, trial baseline, emotion induction, emotion regulation) three times; first with the REACT training and prompt, then with either the DISTRACT or MINDFUL AWARENESS training or prompt, and last with whichever prompt remained. Order of DISTRACT versus MINDFUL AWARENESS and the pairing of emotion inductions with emotion regulation strategies were counterbalanced. Participant groups did not differ in the extent with which they reported attempting to use DISTRACT and MINDFUL AWARENESS strategies (Fitzpatrick et al., 2020a).

2.4. Data Analytic Strategy

Data were aggregated within each phase prior to analysis to aid convergence for some models and because hypotheses focus on between-person/between-phase differences. For self-report and parasympathetic outcomes, we aggregated within each phase by computing the mean. As SCRs are counts, we summed the total number of SCRS within each phase. Length of time varied across phases (phase baseline = 5 min, induction = 2 min, regulation = 2.5 min), so we adjusted SCR counts to be comparable across phases by identifying the lowest common multiple of time among the three phases (10), then multiplied SCR counts in each phase to be equivalent

to a total phase time of 10 minutes. Finally, baseline models used the last 5 minutes of data from the 10-minute baseline to ensure stable estimates (Jennings et al., 1992).

All analyses were conducted using R 4.1.2 (R Core Team, 2021). Ordinary least squares models used base R, negative binomial models used the MASS package (Venables & Ripley, 2002), and multilevel models and generalized linear mixed models used the lme4 package (Bates et al., 2015). Sleep variables were standardized to aid convergence and simplify interpretation.

For baseline phase models, each baseline emotion measure (self-report, sympathetic, and parasympathetic) was separately regressed onto two dummy-coded group variables (GAD or BPD = 1, HC = 0), three sleep variables (chronotype, SQ, and linear and quadratic SE), and interactions between each group and sleep variable. Positive model coefficients represent higher baseline emotion (e.g., higher self-reported, sympathetic, or parasympathetic emotion). Ordinary least squares models were used for self-report and parasympathetic emotion, and negative binomial models were used for sympathetic emotion due to the count nature of SCRs.

Reactivity models used multilevel models and focused on the "react" condition only. This condition always came first (before mindfulness or distraction), which means the trial-baseline was unaffected by any previous emotion inductions. For these models, each emotion measure was separately regressed onto the three-way interactions between group, each sleep variable, and a dummy-coded phase variable (0 = trial-baseline phase, 1 = induction phase) representing change from trial-baseline to induction phase (i.e., emotional reactivity), plus all lower order terms. A positive value for reactivity represents an increase in the emotion outcome from the trial-baseline phase to the induction phase (and vice-versa for negative coefficients). Two-way interactions between sleep and reactivity test whether sleep was associated with reactivity (which

are conditional in three-way models), and three-way interactions between sleep, phase, and group test whether sleep's association with reactivity differed by groups.

Regulation models focused on the two conditions in which participants were instructed to regulate their emotions–"MINDFUL AWARENESS" and "DISTRACT" conditions–which were treated as repeated measures within each person. Regulation models were tested using the same model as reactivity, except for the dummy-coded phase (induction phase = 0, regulation phase = 1), which represents change from induction phase to regulation phase (i.e., emotion regulation). Similar to reactivity models, a positive value for regulation indicates an increase from induction to regulation phase (i.e., less emotion regulation for SCR and more emotion regulation for RSA and self-report). For all models, when three-way interactions were nonsignificant, these terms were then removed to test associations between sleep variables and emotion dysregulation components across groups. Additionally, a quadratic effect for SE was nonsignificant in all models (*p*s>.20)—except emotion regulation models for self-reported emotion—so the term was removed from these models to simplify interpretation of results.

3. Results

3.1. Baseline negative emotion

See Table 2 for means and standard deviations for all sleep variables (means and standard deviations for emotion variables are presented elsewhere; Fitzpatrick et al., 2020a), and Table 3 for correlations among main study variables. See Table 4 for model results for baseline emotional intensity.[1]

3.1.1. Self-reported emotion

[1] Results from models wherein no results related to primary study questions are statistically significant are presented in supplementary tables.

The association between SE and self-reported baseline emotion significantly differed by group, in which both BPD and GAD groups differed from HCs (but not from each other). Specifically, HCs with higher SE had more negative/less positive self-reported baseline emotion (*B*=-0.90, *p*=.004), but this association was not significant for GAD (*B*=0.11, *p*=.546) or BPD (*B*=0.04, *p*=.840) groups. Associations between either chronotype or SQ and self-reported baseline emotion did not significantly differ by group. Across groups, those with higher (earlier) chronotype (*B*=0.36, *p*=.002) and better SQ (*B*=0.28, *p*=.026) had more positive/less negative self-reported baseline emotion.

3.1.2. Sympathetic emotion

None of the associations between sleep measures and sympathetic baseline emotion significantly differed by group (see Table S1; .106<*p*s<.862), nor were any sleep measures significantly associated with sympathetic baseline emotion across groups.

3.1.3. Parasympathetic emotion

Associations between two sleep measures (SE and SQ) and baseline RSA differed between GAD and HC participants. HCs with lower SE (*B*=-0.60, *p*=.042) and higher SQ (*B*=0.51, *p*=.015) had higher baseline RSA (*B*=-0.60, *p*=.042). In contrast, those in the GAD group did not have significant associations between either SE (*B*=0.17, *p*=.329) or SQ (*B*=-0.16, *p*=.442) and baseline RSA. Those in the BPD group also did not have significant associations between either SE (*B*=0.03, *p*=.856) or SQ (*B*=0.09, *p*=.690) and baseline RSA, but this group did not differ from HCs. Finally, associations between chronotype and parasympathetic emotion did not differ by group, and chronotype was not associated with parasympathetic emotion across groups (*B*=-.09, *p*=.409).

3.2. Emotional reactivity

Table S2 shows model results for emotional reactivity. None of the associations between sleep measures and emotional reactivity differed by group, nor were any sleep variables significantly associated with emotional reactivity across groups.

3.3. Emotion regulation

Table 5 shows model results for emotion regulation.

3.3.1. Self-reported emotion

The association between SE and self-reported emotion regulation differed by group, in which both BPD and GAD groups differed from HCs (but not from each other). HC had a curvilinear (inverted-U) association between SE and self-reported emotion regulation (B=-0.700, p=.043; See Figure 1), in which moderate SE was optimal for self-reported emotion regulation. For GAD and BPD groups, SE was not linearly or curvilinearly associated with self-reported emotion regulation. Associations between either chronotype or SQ and self-reported emotion regulation did not significantly differ by group, and neither chronotype nor SQ was associated with self-reported emotion regulation across groups.

3.3.2. Sympathetic emotion

Associations between chronotype and sympathetic emotion regulation differed between GAD and HC participants. HCs with earlier chronotypes had greater sympathetic regulation (i.e., greater decreases in sympathetic response; B=-0.42, p=.001). In contrast, those in the GAD group did not have significant associations between chronotype and sympathetic regulation (B=0.13, p=.401). Those in the BPD group also did not have significant associations between chronotype and sympathetic regulation (B=-0.24, p=.105), but this group did not differ from HCs. Finally, the association between either SE or SQ and sympathetic emotion regulation did

not differ by group, and these sleep variables were not associated with sympathetic emotion regulation across groups.

3.2.3. Parasympathetic emotion

None of the associations between sleep measures and parasympathetic emotion regulation significantly differed by group. Across groups, those with higher SQ had greater parasympathetic regulation (i.e., greater increases in RSA; i.e., increases in emotion regulation; Porges et al., 1994) (B=0.12, p=.023). Neither SE nor chronotype was associated with parasympathetic emotion regulation across groups.

4. Discussion

The current study aimed to determine whether and which homeostatic, circadian, and subjective sleep components predict emotion dysregulation elements (i.e., baseline emotion, emotional reactivity, and emotion regulation) in individuals with BPD, HCs, and a clinical control group (GAD). It was hypothesized that low and high SE, later chronotypes, and poorer SQ would predict elevated emotion dysregulation across components and groups.

4.1. Baseline emotion

In line with our hypotheses, across groups, earlier chronotype and higher SQ predicted more positive/less negative self-reported baseline emotion. Due to demands of daily life (e.g., work beginning at 9:00 AM), people with later chronotypes often experience a misalignment between their physiological sleep drive and daily obligations. This misalignment can lead to chronic sleep deprivation, which may disrupt emotional processes (Wittmann et al., 2006). As people with early chronotypes are less likely to experience circadian misalignment, it is possible that they do not accumulate as much sleep debt as those with later chronotypes. Therefore, across

groups, proclivities towards later circadian rhythms and the consequences it may entail may exacerbate baseline emotion, at least in the experiential domain.

The effect of chronotype on baseline emotion may also be intertwined with the relationship between higher SQ and more positive/less negative self-reported baseline emotion across groups. Indeed, perhaps individuals with later chronotypes experience lower quality sleep if they are frequently sleeping outside of the timeframes dictated by their circadian rhythm. SQ may therefore reflect a broader subjective experience of not being restored from sleep, which may interfere with the functions of sleep that improve baseline emotion. Alternatively, because the effect of SQ on baseline emotion was only evident in the self-report domain, some individuals may have a negative reporting bias that may lower both their ratings of SQ and baseline emotion. Indeed, studies suggest that at least some ratings of subjective SQ are influenced by mood state and may reflect generally negative or pessimistic cognitive patterns (Buysse et al., 2008; Grandner et al., 2006). It is notable that, in the HC group only, higher SQ also predicted higher baseline RSA. Studies suggest that the construct of SQ may be particularly biased by general subjective distress in psychiatric relative to non-psychiatric groups (Hartmann et al., 2015). Therefore, perhaps the relationship between SQ and baseline emotion is distinct for HCs and clinical groups, wherein some aspect of SQ actually exacerbates baseline emotion for the former but reflects a more general negative subjective bias for the latter.

Our findings also suggested that HCs specifically are vulnerable to the effects of high SE as it predicted lower baseline RSA (i.e., higher baseline negative emotional intensity) and more negative self-reported baseline emotion in this group. Low SE may reflect problems driven by insomnia such as spending large portions of time in bed and not being able to sleep. On the other hand, high SE may be more indicative of sleep deprivation, as individuals fall asleep abnormally

quickly when presented with the opportunity to do so (Perlis et al., 2005). The HC group is defined by lack of psychopathology, and thus individuals in this group did not have insomnia. Therefore, the influence of low SE on baseline emotion may not be detectable in such a group. Conversely, HCs may be sleep deprived for a range of reasons, which may be reflected in SE. The relationship between high SE and poorer self-reported and parasympathetic baseline emotion in HCs ultimately corroborates a broader literature suggesting that adequate sleep is essential to restore and replenish emotion generating and regulatory networks (e.g., Gruber & Cassoff, 2014).

Finally, there was no relationship between sleep processes and baseline sympathetic emotion across groups. Therefore, impaired sleep may produce subjective distress and interfere with emotion regulatory networks (e.g., RSA; Beauchaine, 2001; Berntson et al., 1997; Porges et al., 1994), but may not directly elicit sympathetic arousal.

4.2. Emotional reactivity

Numerous studies demonstrate associations between emotional reactivity and sleep components (e.g., Altena et al., 2016; Baglioni et al., 2010; O'Leary et al., 2017; Tempesta et al., 2020). Despite this, and in contrast with our predictions, there were no relationships between sleep processes and emotional reactivity across groups and emotion domains. It is possible that disrupted sleep influences emotional reactivity in variable ways, wherein individuals with poor sleep exhibit heightened or blunted reactivity (e.g., Nota et al., 2021), and such opposing effects obfuscate the detection of either of them. In addition, across groups, average rates of sleep efficiency and chronotypes were approximating "normal" ranges, and average rates of sleep quality provided ratings of "fair" to "good". Therefore, the current sample may also have lacked severe enough sleep problems to impact emotional reactivity. Furthermore, the parent study

showed that, although all groups exhibited increases in self-reported negative emotion from beginning to after the emotion inductions, only some groups showed increases in physiological indices of emotion (Fitzpatrick et al., 2020). Therefore, the study paradigm may not have elicited high enough emotional reactivity to detect potential relationships.

4.3. Emotional regulation

As with baseline emotion and consistent with hypotheses, higher SQ predicted improved parasympathetic emotion regulation across groups. This finding acts in concert with research suggesting that high SQ is associated with enhanced emotion regulation (Mauss et al., 2013). As this result was specific to the parasympathetic domain, it may also suggest that SQ enhances emotion regulation more than it directly dampens negative emotion. However, given the subjectivity of SQ ratings, it is unclear what specific element of it may be helpful to this end. For example, it is possible that SQ is interpreted as a general assessment of sleep that is influenced by another, unmeasured sleep process which directly influences emotion regulation, and additional research is needed to this end.

Also similar to our baseline emotion findings, some sleep components influenced emotion regulation in HCs specifically. Earlier chronotypes were associated with improved sympathetic emotion regulation in HCs. It is unclear whether the influence of chronotype on emotion regulation in HCs was observed because early chronotypes themselves promote optimized emotion regulation, or because they provide opportunity for alignment between chronotype and social rhythms, resulting in more restorative sleep. Future researchers are advised to probe such distinctions.

Second, in addition to chronotypes and partially consistent with our hypotheses, SE influenced self-reported emotion regulation in HCs only. In particular, SE had a quadratic

relationship with self-reported emotion regulation for this group wherein high and low levels predicted worsened subjective emotion regulation. This finding suggests that self-reported emotion regulation may be impaired by low sleep efficiencies that may result from spending extended periods of time in bed and consequentially inadequate sleep drive, and high sleep efficiencies that could result from an excessive drive to sleep that builds in the setting of sleep deprivation.

It is unclear why this effect was specific to the self-reported domain. Perhaps SE levels were not extreme enough in either direction to influence physiological emotion regulation processes. Alternatively, perhaps participants were more likely to *report* benefits from emotion regulation strategies– possibly due to better mood states– in the setting of moderate SE, regardless of the actual benefits they observed, which would be evident in self-report but not physiological emotion domains. It is also unclear why the effects of SE on baseline emotion and emotion regulation, as well as chronotype on sympathetic emotion regulation, were specific to HCs. The lack of sleep problems in the HC group may make this group particularly vulnerable to atypical deviations in sleep processes (e.g., changes in SE). Furthermore, HCs may have more fluid emotion systems that are more responsive to external influence such as sleep deprivation, whereas emotion in clinical groups may be pervasively negative and less responsive to sleep parameters.

5. Limitations and Clinical Implications

There are several clinical implications of these findings. First, assessing and optimizing SQ may facilitate the reduction of baseline emotion and enhancement of emotion regulation regardless of diagnostic status. Moreover, clinicians are advised to assess client's chronotypes and their potential alignment or lack thereof with their life demands as those with later

chronotypes may be particularly likely to struggle with negative self-reported baseline emotion and improving such alignment may help ameliorate this. Finally, healthy populations may benefit from finding a balance between excessive time in bed and deprived sleep, ensuring that their SE is moderate, to facilitate emotion regulation.

These findings are not without limitations. First, while the sleep measures used are empirically-supported, they are still vulnerable to recall bias. Future studies should incorporate objective measures of sleep processes in relation to emotion dysregulation. Second, sleep problems (insomnia) are a diagnostic criterion of the clinical control group (GAD), but not the BPD group (APA, 2022). Consequently, the BPD group may have had more variability in sleep processes compared to the clinical control group, obfuscating group-specific effects across them. Alternatively, rates of insomnia were higher in the BPD (77.5%) than GAD (27.5%) group. Insomnia is conflated with several of the sleep components under study as it inherently involves low sleep efficiency and subjective sleep quality. It is possible that study results were conflated by the uneven prevalence of insomnia across groups, which was not accounted for in analyses due to its overlap with sleep constructs of interest. Third, individuals taking most psychiatric medications including sleeping medications were excluded from this study. While this enhanced the validity of the psychophysiology, it also potentially restricts the sample to those with no to mild sleep problems. Therefore, the potential influence of sleep processes on emotion dysregulation in people who take sleep medications remains unclear.

Despite these limitations, this study presents a comprehensive assessment of the impact of sleep problems on emotion dysregulation in BPD. Ultimately, findings indicate that sleep and emotion are intertwined, but this relationship may generally be more prominent for healthy populations than those with BPD. Despite this, some selective pathways may exist to facilitate

emotion dysregulation interventions by addressing specific sleep processes such as addressing chronotype misalignments and targeting sleep quality.

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Table 1

Demographics and current diagnoses across participant groups

	Healthy controls	Borderline personality disorder	Generalized anxiety disorder
Age [Mean(standard deviation)]	24.70 (6.88)	25.35 (6.77)	25.95 (7.20)
Gender Identity (%)			
Woman	77.5%	77.5%	76.3%
Man	20%	17.5%	21.1%
Transgender/gender queer/other	2.5%	5%	2.6%
Ethnicity (%)			
Chinese or Chinese-Canadian/Korean or Korean-Canadian/Other Asian or Asian-Canadian	55%	37.5%	23.7%
White/Caucasian/European Origin	15%	40%	34.2%
Black-Canadian/Black/Caribbean Origin	10%	5%	5.3%
Mexican or Mexican-Canadian/ Hispanic/Latinx	10%	0%	5.3%
Middle Eastern	5%	0%	5.3%

Aboriginal-Canadian/First Nations/Metis/Inuit	0%	2.5%	0%
East Indian	0%	2.5%	5.3%
Other/bi-racial/multi-racial	5%	12.5%	21.1%
Marital Status			
Single	71.8%	35%	55.3%
Dating	15.4%	47.5%	26.3%
Married/common law/life partner	12.8%	7.5%	18.4%
Separated/Divorced/Other	0%	10%	0%
Current Comorbid Diagnoses			
Major Depressive Disorder/Dysthymic Disorder		47.5%	22.5%
Bipolar II Disorder		5%	2.5%
Substance induced mood disorder		2.5%	0%
Psychotic disorder not otherwise specified		2.5%	0%
Alcohol abuse/substance abuse disorder		17.5%	2.5%
Panic disorder/Agoraphobia/ Agoraphobia without a history of panic disorder		20%	10%
Social anxiety disorder		45%	42.5%
Specific phobia		17.5%	25%

Obsessive compulsive disorder	32.5%	15%
Posttraumatic stress disorder	15%	2.5%
Generalized anxiety disorder	40%	100%
Eating disorder	17.5%	7.5%
Insomnia	77.5%	27.5%

Table 2

Means (standard deviations) of sleep variables

	Healthy controls	Borderline personality disorder	Generalized anxiety disorder
Sleep efficiency	.901 (.064)	.814 (.108)	.788 (.199)
Chronotype	49.325 (12.031)	41.400 (10.466)	47.200 (9.879)
Sleep quality	2.870 (.651)	2.144 (.647)	2.200 (.695)

Table 3

Correlations among main study variables

	1	2	3	4	5	6	7	8	9	10	11	12	13
1. GAD group	-												
2. BPD group	-.500***	-											
3. Sleep efficiency	-.190*	-.196*	-										
4. Chronotype	.077	-.289**	.065	-									
5. Sleep quality	-.198*	-.251**	.439***	.058	-								
6. Self-report (Baseline)	-.006	-.293**	.121	.349***	.288**	-							
7. Sympathetic (Baseline)	.054	.155	-.015	.013	.063	.058	-						
8. Parasympathetic (Baseline)	-.058	-.108	.075	-.036	.204*	.154	-.038	-					
9. Self-report (Reactivity)	-.061	.022	-.095	.099	-.103	-.103	-.123	-.126	-				
10. Sympathetic (Reactivity)	.023	.055	.07	.14	-.029	.013	-.084	.202*	-.018	-			
11. Parasympathetic (Reactivity)	-.059	.095	-.021	-.159	-.038	-.111	-.069	-.062	.04	-.082	-		
12. Self-report (Regulation)	.095	.107	-.218*	-.041	-.094	.006	.190*	-.005	-.16	-.038	-.027	-	
13. Sympathetic (Regulation)	.044	.000	.023	-.146	.037	-.017	.313***	-.229*	-.228*	-.541***	.114	.243**	-
14. Parasympathetic (Regulation)	.088	-.072	.058	.093	.244**	.081	.168	-.103	-.039	.107	-.252**	-.029	.083

Note. GAD = generalized anxiety disorder, BPD = borderline personality disorder, GAD group = dummy-coded variable for group (GAD group = 1, healthy controls and BPD group = 0), BPD = dummy-coded variable for group (BPD group = 1, healthy controls and GAD group = 0). Variables 6-14 are emotion variables, which were aggregated at the person level in this table. All correlations therefore represent between-persons correlations. Reactivity = mean during induction phase subtracted by mean during trial-baseline ("react" condition only), regulation = mean during regulation phase subtracted by mean during induction phase ("mindfulness" and "distract" conditions only).

Table 4

Model results examining influence of sleep variables on self-reported and parasympathetic baseline emotion

Self-reported emotion

Variable	B	SE	t	p
Intercept	5.551	0.27	20.64	<.001
Group_GAD	-0.608	0.33	-1.84	.068
Group_BPD	-0.914	0.34	-2.69	.008
SE	-0.898	0.31	-2.91	.004
Chronotype	0.426	0.18	2.40	.018
SQ	0.387	0.22	1.75	.083
Group_GAD×SE	1.011	0.36	2.80	.006
Group_BPD×SE	0.935	0.36	2.59	.011
Group_GAD×chronotype	-0.063	0.27	-0.23	.819
Group_BPD×chronotype	-0.150	0.27	-0.55	.582
Group_GAD×SQ	-0.212	0.31	-0.68	.499
Group_BPD×SQ	-0.037	0.32	-0.12	.908

Respiratory sinus arrythmia

Variable	B	SE	t	p
Intercept	6.644	0.25	26.19	<.001
Group_GAD	-0.324	0.31	-1.03	.305
Group_BPD	-0.338	0.32	-1.05	.295
SE	-0.601	0.29	-2.06	.042

Chronotype	-0.071	0.17	-0.43	.671
SQ	0.514	0.21	2.46	.015
Group_GAD×SE	0.774	0.34	2.27	.025
Group_BPD×SE	0.633	0.34	1.86	.066
Group_GAD×chronotype	-0.239	0.27	-0.89	.375
Group_BPD×chronotype	0.252	0.26	0.98	.328
Group_GAD×SQ	-0.678	0.30	-2.28	.025
Group_BPD×SQ	-0.427	0.30	-1.42	.158

Note. SE=Sleep efficiency; SQ=Sleep quality

Table 5

Model results examining influence of sleep variables on emotion regulation

Self-reported emotion				
variable	**B**	**SE**	**t**	**p**
(Intercept)	4.381	0.42	10.333	<.001
Group_GAD	-1.194	0.50	-2.38	.019
Group_BPD	-1.526	0.51	-2.98	.003
Regulation	0.24	0.32	0.76	.450
SE	-0.394	0.44	-0.89	.377
SE^2	-0.265	0.47	-0.57	.570
Chronotype	0.374	0.24	1.55	.123
SQ	-0.154	0.30	-0.51	.609
Group_GAD×Regulation	0.023	0.37	0.06	.952
Group_BPD×Regulation	0.138	0.38	0.36	.718
Group_GAD×SE	0.12	0.62	0.19	.847
Group_BPD×SE	0.885	0.63	1.40	.165
Group_GAD×SE^2	0.175	0.48	0.36	.717
Group_BPD×SE^2	0.415	0.49	0.84	.400
Group_GAD×chronotype	-0.202	0.38	-0.54	.591
Group_BPD×chronotype	-0.094	0.38	-0.25	.806
Group_GAD×SQ	0.323	0.43	0.75	.455
Group_BPD×SQ	-0.079	0.44	-0.18	.859

Regulation×SE	0.134	0.33	0.41	.683
Regulation×SE^2	-0.7	0.35	-2.03	.043
Regulation×chronotype	-0.193	0.18	-1.09	.279
Regulation×SQ	-0.072	0.22	-0.32	.749
Group_GAD×Regulation×SE	-0.077	0.46	-0.17	.866
Group_BPD×Regulation×SE	-0.325	0.47	-0.70	.487
Group_GAD×Regulation×SE^2	0.781	0.36	2.19	.029
Group_BPD×Regulation×SE^2	0.747	0.36	2.05	.041
Group_GAD×Regulation×chronotype	0.415	0.28	1.50	.134
Group_BPD×Regulation×chronotype	0.127	0.28	0.45	.653
Group_GAD×Regulation×SQ	-0.022	0.32	-0.07	.945
Group_BPD×Regulation×SQ	0.362	0.33	1.11	.270

Skin conductance responses

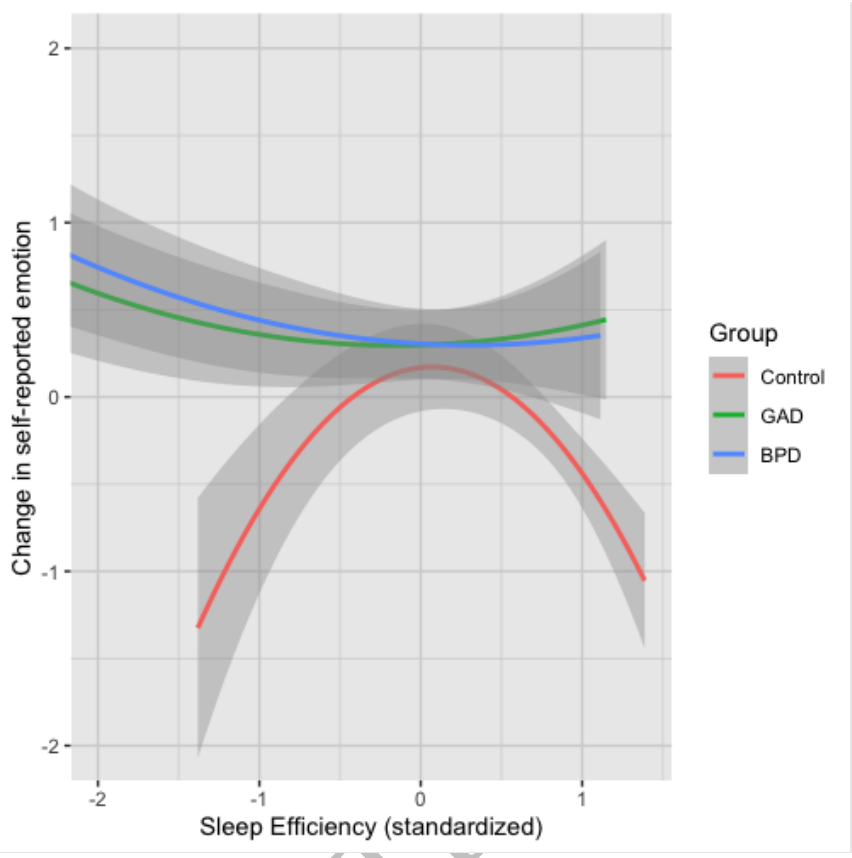
variable	**B**	**SE**	**z**	**p**
(Intercept)	2.602	0.27	9.61	<.001
Group_GAD	0.620	0.33	1.87	.061
Group_BPD	0.846	0.34	2.48	.013
Regulation	0.032	0.19	0.17	.869
SE	0.213	0.31	0.68	.496
Chronotype	0.531	0.18	2.92	.003
SQ	-0.132	0.22	-0.60	.551
Group_GAD×Regulation	-0.004	0.24	-0.02	.987
Group_BPD×Regulation	0.018	0.25	0.07	.941

Group_GAD×SE	0.167	0.36	0.46	.648
Group_BPD×SE	-0.476	0.36	-1.31	.191
Group_GAD×chronotype	-0.929	0.28	-3.32	.001
Group_BPD×chronotype	-0.229	0.27	-0.84	.402
Group_GAD×SQ	0.097	0.31	0.31	.756
Group_BPD×SQ	0.448	0.32	1.41	.158
Regulation×SE	0.055	0.23	0.24	.808
Regulation×chronotype	-0.425	0.13	-3.22	.001
Regulation×SQ	0.041	0.16	0.25	.802
Group_GAD×Regulation×SE	-0.120	0.26	-0.46	.649
Group_BPD×Regulation×SE	0.166	0.26	0.63	.527
Group_GAD×Regulation×chronotype	0.555	0.20	2.72	.006
Group_BPD×Regulation×chronotype	0.186	0.20	0.94	.348
Group_GAD×Regulation×SQ	-0.015	0.23	-0.07	.948
Group_BPD×Regulation×SQ	-0.090	0.23	-0.39	.700

Note. SE=Sleep efficiency; SQ=Sleep quality

Figure 1

Curvilinear associations between sleep efficiency and model-predicted self-reported emotion regulation



Note. Error bands represent 95% confidence intervals.