Identifying specific insomnia components in borderline personality disorder and their influence on emotion dysregulation

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Abstract

Insomnia-related sleep problems are common in borderline personality disorder (BPD) and exacerbate the core of BPD, emotion dysregulation. Insomnia is elicited and maintained through behaviors that disrupt both the homeostatic and circadian sleep systems. However, it is unclear which homeostatic or circadian insomnia behaviors characterize BPD and exacerbate emotion dysregulation, thus warranting clinical attention in this population. This study therefore investigated whether homeostatic (i.e., abnormalities in time in bed (TIB) and sleep efficiency (SE)) and circadian (i.e., abnormalities in risetime variability and chronotypes) behaviors characterize and exacerbate emotion dysregulation in BPD relative to healthy (HC) and generalized anxiety disorder (GAD) groups. Participants from the community who met criteria for BPD, GAD, or no psychological disorders (HCs) were recruited and completed measures of emotion dysregulation. They also completed measures of daily homeostatic and circadian insomnia behavior measures for fourteen days. Generalized estimating equations revealed that the GAD group exhibited lower SE than HCs, and there was a marginally significant effect wherein the BPD group exhibited delayed risetimes relative to the GAD group. Moreover, higher TIB predicted elevated emotion dysregulation in HCs but lower emotion dysregulation in the GAD group. Higher SE predicted higher emotion dysregulation in BPD. These results suggest that the influence of insomnia behaviors on emotion dysregulation is heterogeneous. Idiographic assessments of the influence of insomnia behaviors on emotion dysregulation are advised.

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Borderline Personality Disorder (BPD) affects 5.9% of the population (Pagura et al., 2010), with 10% dying by suicide (Paris & Zweig-Frank, 2001). Effective BPD treatment requires understanding what factors exacerbate it, so that BPD interventions can target them to optimize outcomes. Insomnia-related sleep problems and BPD are highly associated. Insomnia is characterized by difficulty with either: (1) initiating, (2) maintaining, or (3) waking too early from, sleep (American Psychiatric Association, 2013). Up to 63% of those with BPD report at least one of these problems (Selby, 2013). This manuscript aims to specify the nature of insomnia-related sleep problems in BPD and their impact on emotion dysregulation.

Contemporary theories suggest that emotion dysregulation, comprised of abnormalities in both emotion and its modulation (i.e., emotion regulation; Gross & Thompson, 2007), is the core of BPD (Linehan, 1993). Emotion dysregulation involves emotion-related regions (e.g., the amygdala) and prefrontal regions of the brain implicated in emotion regulation. Inadequate sleep is associated with decreased functional relationships between prefrontal emotion regulation regions (i.e., medial prefrontal cortex), and the amygdala, and heightened amygdala activation (e.g., Gruber & Cassoff, 2014; Kahn et al., 2013; Yoo, Gujar, Hu, Jolesz, & Walker, 2007). Accordingly, extensive research suggests that sleep problems are associated with more intense and labile negative emotion (see Kahn, Sheppes, & Sadeh, 2013, for review) and greater difficulties regulating it. For example, in healthy populations, poorer sleep quality predicts higher emotion dysregulation longitudinally (Tavernier & Willoughby, 2014), and worse emotion regulation in a laboratory task (Mauss, Troy, & LeBourgeois, 2013). Reciprocally, evidence suggests that emotion dysregulation can impact the ability to fall asleep and subsequent

sleep quality (Konjarski, Murray, Lee, & Jackson, 2018), perpetuating a destructive cycle between sleep problems and emotion dysregulation over time. Insomnia may therefore account for and contribute to some of the emotion dysregulation and, consequently, BPD symptoms, observed in BPD. Reciprocally, the emotion dysregulation that characterizes BPD may also contribute to insomnia in this group over time. Indeed, those who recover from BPD have better sleep quality, less sleep-related daytime dysfunction, faster sleep onset, and less sleep medication use than those who do not (Plante, Frankenburg, Fitzmaurice, & Zanarini, 2013). Insomnia-related sleep problems in particular may thus exacerbate BPD through its impact on emotion dysregulation, and reducing insomnia symptoms may improve BPD treatment outcomes.

Adequate sleep is governed by two interrelated systems; the homeostatic and circadian systems. The *homeostatic* system balances rest and activity because it involves the accumulation of deep sleep drive during increasing wakefulness. The *circadian* system is a "body clock" that determines optimal timings of sleep and wakefulness (Borbély, 1982; Webb, 1988). Cognitive behavioral models of insomnia emphasize that individuals with insomnia engage in behaviors that disrupt both systems, eliciting and maintaining insomnia (Edinger & Carney, 2015). However, it is unclear whether individuals with BPD exhibit behaviors that disrupt either homeostatic or circadian sleep processes and, if so, whether they influence emotion dysregulation in this population. Therefore, which set of insomnia behaviors, if any, require clinical attention in this population, remains unknown.

Insomnia Behaviors and BPD

Homeostatic insomnia behaviors. Theory suggests that individuals with insomnia disrupt the homeostatic system by attempting to make up for lost sleep by increasing time in bed (TIB), which (1) results in reduced sleep efficiency (SE; the ratio of time in bed relative to the

time spent sleeping); (2) diminishes sleep-promoting signals; and (3) promotes conditioned associations between wakefulness and the bed, collectively leading to poorer sleep (Spielman, Caruso, & Glovinsky, 1987). However, it is unclear whether individuals with BPD exhibit altered TIB and SE, and thus whether such homeostatic insomnia behaviors require targeting in this population. No research has examined whether individuals with BPD spend more TIB than healthy or other clinical groups, or whether TIB influences emotion dysregulation. Conversely, studies examining sleep during one or two nights in a laboratory (Asaad, Okasha, & Okasha, 2002; Bell, Lycaki, Jones, Kelwala, & Sitaram, 1983; Lahmeyer et al., 1988) or on an inpatient unit (Battaglia, Ferini-Strambi, Smirne, Bernardeschi, & Bellodi, 1993) show that BPD groups exhibit lower SE than healthy controls (HCs), and higher (Asaad et al., 2002; Lahmeyer et al., 1988) or equivalent (Bell et al., 1983) SE compared to major depressive disorder (MDD) groups. Cross-sectional questionnaire research also suggests that SE is lower in BPD than in HCs (Taherifard, Abolghasemi, & Hajloo, 2015; Weibel et al., 2017), higher than in antisocial personality disorder groups (Taherifard et al., 2015), and comparable to attention deficit and hyperactivity disorder groups (Weibel et al., 2017). A meta-analysis integrating these findings suggests that SE is lower in BPD than in HCs, but comparable to clinical controls (Winsper et al., 2017). However, this research is limited by its sole examinations of SE during either one or two nights of sleep in a laboratory or inpatient unit, or a single questionnaire assessing general sleep tendencies. Such methods do not indicate whether SE is lower, or influences emotion dysregulation, in the real-world, day-to-day lives of individuals with BPD.

One study showed that SE, measured by daily wrist actigraphy for two weeks, was lower in participants with BPD compared to HCs (Bromundt et al., 2013). However, some of the BPD group received light therapy during this study which, along with the study's small sample sizes,

confounds and limits study conclusions. This study is also hampered by its lack of inclusion of a clinical control group, convoluting any inferences that can be drawn about which homeostatic insomnia behaviors uniquely characterize BPD, rather than psychopathology broadly. Finally, this study did not control for the potential influence of depression severity. Insomnia is a diagnostic criterion for depression (APA, 2013), which is highly comorbid with BPD (i.e., 96% of individuals with BPD report a mood disorder; Zanarini et al., 1998). Studies suggest that BPD diagnoses do *not* predict SE once depression severity is controlled for (Weibel et al., 2017), and that BPD without current MDD and HC groups exhibit comparable SE (Philipsen et al., 2005). Depression severity may thus account for SE differences between BPD and controls. In sum, it is unclear whether real-world homeostatic insomnia behaviors, namely TIB and SE, are key clinical foci in BPD because they (1) are elevated compared to HC and clinical control groups, after controlling for depression severity, and (2) exacerbate emotion dysregulation.

Circadian insomnia behaviors. Beyond homeostatic irregularity, insomnia also involves disruption of the circadian system (Schwartz & Carney, 2012). Sleep schedule variability can interfere with circadian timings and perpetuate insomnia over time (Bootzin, 1972). Compared to bedtimes, risetimes more clearly indicate problematic variability in sleep scheduling because insomnia treatments emphasize the importance of rising consistently but refraining from going to bed, even at the scheduled bedtime, if not sleepy (Edinger & Carney, 2015). Therefore, bedtime variability can reflect problematic *or* appropriate sleep scheduling, while risetime variability is more clearly problematic. Individuals also have biological differences in the patterns in which circadian rhythms operate (i.e., chronotypes; McEnany & Lee, 2000). Intrinsic body clocks can be “advanced” or “delayed,” leading to biological imperatives to go to bed and rise earlier, or later, respectively (McEnany & Lee, 2000). Phase delayed chronotypes are associated with a

range of BPD-relevant adverse outcomes such as mood disruption, impulsivity, and destructive behaviours such as aggression and substance use (Taylor & Hasler, 2018). It is important to identify which circadian insomnia behaviors, if any, are present in BPD and exacerbate emotion dysregulation, therefore warranting clinical attention.

No studies have examined risetime variability (RTV) in BPD. One measured sleep-wake cycles via actigraphy, skin temperature, and salivary melatonin in BPD and HCs, while some participants received light therapy. The BPD group was more variable than HCs in sleep-wake cycles, but did not exhibit distinct chronotypes (Bromundt et al., 2013). However, as mentioned, small sample sizes, ongoing light therapy, and a lack of clinical controls limit this study's findings. Whether RTV or chronotype influences emotion dysregulation is also unknown.

In sum, healthy sleep is governed by homeostatic and circadian processes, both of which can be disrupted through a series of behaviors in insomnia. However, it is not clear which specific homeostatic or circadian insomnia behaviors are present in BPD, or influence emotion dysregulation, and thus need clinical attention. The present study examined which homeostatic and circadian insomnia behaviors (i.e., TIB, SE, RTV, chronotype) (1) are elevated in BPD compared to a HC and a generalized anxiety disorder (GAD) clinical control group, and (2) predict emotion dysregulation, after controlling for depression severity. GAD was selected as a clinical control because, like BPD, insomnia is prevalent (85% endorse it; Navarrete, Páramo, Ordoño, & Gómez, 2017) and emotion dysregulation is elevated (Mennin, Heimberg, Turk, & Fresco, 2005) in GAD. A GAD group thus allows for an examination of whether insomnia behaviors are specific to BPD or pervasive across high emotion dysregulation groups. Given associations between BPD and insomnia (Selby, 2013), we hypothesized that individuals with BPD would exhibit higher TIB and RTV, and lower SE, than HCs. Insomnia behavior

comparisons between BPD and GAD groups are considered exploratory given a lack of research in these areas. We also hypothesized that higher TIB, lower SE, and higher RTV would predict elevated emotion dysregulation across groups. We considered the influence of chronotype on emotion dysregulation, and examinations of group differences in these relationships, exploratory.

Method

Participants

Participants between ages 18 to 60 (N = 120; 40 per BPD, GAD, or HC group) were recruited with fliers and online postings as part of a broader parent study examining sleep and emotion processes in BPD. Groups were sex- and age- (+/- five years) matched. Clinical group participants were excluded if they were taking medications known to influence psychophysiological responding such as psychiatric mediations other than selective serotonin reuptake inhibitors (SSRIs; Licht, de Gues, van Dyck, & Penninx, 2010), H1 histamine receptor blockers, and beta-blockers (Hou, Langley, Szabadi, & Bradshaw, 2007; Sandrone et al., 1994). However, it remained possible that those taking more allowable psychiatric medications may have exhibited greater sleep problems or higher emotion dysregulation severity, and we therefore controlled for number of psychiatric medications in analyses. Participants were also excluded if they endorsed having medical conditions that could interfere with the parent study such as major neurocognitive disorders, mental retardation, organic brain syndromes, traumatic brain injuries, epilepsy, heart/respiratory conditions, and metabolic syndromes (e.g., Lutfi, 2012; Malpas & Maling, 1990; Tomson, Ericson, Ihrman, & Lindblad, 1998).

Participants were also excluded if they endorsed diagnostic criteria for psychiatric disorders that are likely to interfere with understanding study tasks: current alcohol or drug dependence, bipolar I disorder, and severe psychotic-spectrum disorders. Finally, research

suggests that comorbid BPD is rare in GAD, with an estimated prevalence of 3.3% (Dyck et al., 2001), whereas comorbid GAD is common in BPD, with an estimated prevalence of 13.5% (Zanarini et al., 1998). To ensure that groups were at least somewhat distinct, prospective GAD and HC participants who endorsed four or more BPD criteria, or self-harm/impulsivity BPD criteria were excluded. However, to retain some external validity, individuals in the BPD group were not excluded if they had comorbid GAD. Prospective HCs were excluded if they endorsed full criteria for a current psychiatric disorder. Eligibility was confirmed with psychodiagnostic interviews following screening. Demographics and psychiatric diagnoses are presented in Table 1 and 2, respectively. Based on similar past work (Asaad et al., 2002), we conservatively estimated an effect size difference in insomnia behaviors of $d = 1.00$ across groups. Power analyses in G*power indicated that a sample size of 17 individuals per group would be required to detect a statistically significant effect of this size between two groups with 80% power. We thus deemed our sample size of n = 40 per group to be adequate.

Measures

Screening and sample characterization. Participants were screened by phone or online for probable BPD using the *McLean Screening Inventory* (MSI; Zanarini et al., 2003), a 10-item measure that can detect 81% and 85% of BPD cases and non-cases, respectively (Zanarini et al., 2003). Prospective GAD participants were screened for probable GAD using the *Penn-State Worry Questionnaire* (PSWQ; Meyer, Miller, Metzger, & Borkovec, 1990), which assesses the applicability of 16 statements about worry on a five-point scale from 1 (“not at all typical of me”) to 5 (“very typical of me”). The PSWQ discriminates between GAD and other diagnostic groups (Meyer et al., 1990). Participants’ psychiatric diagnoses were assessed in the laboratory with three semi-structured interviews. The *Structured Clinical Interview for Diagnostic and*

Statistical Manual of Mental Disorders-IV-TR (SCID-IV-TR; First, Spitzer, Gibbon, & Williams, 1995) was used to assess for the presence of psychiatric diagnoses other than BPD and insomnia. Supervised undergraduate and masters-level assessors administered all interviews reliably in alignment with a gold-standard assessor, with prevalence-adjusted bias-adjusted kappas (PABAKs; Byrt, Bishop, & Carlin, 1993) ranging from .67 to 1.00 across modules (average PABAK between assessors and goldstandard = .97). The *International Personality Disorders Examination- BPD module (IPDE-BPD;* Loranger et al., 1994) was administered to assess for BPD. The IPDE-BPD module assesses BPD-relevant patterns of thinking, feeling, and acting over the past five years and prior to age 25. It has strong temporal reliability (kappas in the range of .82; Mann et al., 1999) and convergent validity with other BPD measures (Schroeder, Andresen, Naber, & Huber, 2010). Interrater reliability between assessors and the gold-standard assessor was strong, with PABAKS ranging from .67 to 1.00 (average PABAK = .95). The *Duke Structured Interview for Sleep Disorders-Insomnia Module* (Edinger et al., 2009) was used to assess for insomnia. In this measure, interviewers assess whether participants have difficulty falling asleep, staying asleep, or waking up too early and, if so, whether it has caused a variety of problems in daytime functioning. This measure has strong reliability (Kappa values tend to range between .71 and .86 across modules; Carney, Ulmer, Edinger, Krystal, & Knauss, 2009).

Approximately 389, 294, and 200 screenings took place for prospective participants in the BPD, HC, and GAD groups, respectively. Of these, 133 (BPD group), 130 (HC group), and 131 (GAD group) prospective participants were deemed potentially eligible and invited to attend an in-person psychodiagnostic assessment. Following this stage of screening, 76 (BPD group), 53 (HC group), and 85 (GAD group) psychodiagnostic assessments were conducted and indicated that 41 (BPD group), 49 (HC group), and 43 (GAD group) participants were eligible

for the study and invited to complete its next phases. Of this group, the participants who attended the next phase of the study and could be matched to individuals in the other groups on sex and age were included in the final sample of 40 individuals per group.

The Insomnia Severity Index (ISI; Bastien, Vallieres, & Morin, 2001) is a 7-item measure that was used to measure subjective ratings of insomnia severity. The ISI assesses insomnia-related sleep problems (e.g., "to what extent do you consider your sleep problem to INTERFERE with your daily functioning?") on five-point scales that vary in their anchors. Higher scores indicate higher insomnia severity. Scores above 10 typically indicate probable insomnia (Morin, Belleville, Bélanger, & Ivers, 2011). Research supports this measure's psychometric properties, as it has high internal reliability (α = .87 in the present study) and convergent validity, correlating with other sleep-related measures (Morin et al., 2011).

Emotion dysregulation was measured via the *Difficulties with Emotion Regulation Scale (DERS;* Gratz & Roemer, 2004), a 36-item measure that asks participants to indicate the extent to which various emotion dysregulation based statements generally apply to them (e.g., "When I'm upset, I become angry with myself for feeling that way") on a five-point scale ranging from 1 (almost never/0-10% of the time) to 5 (almost always/91-100% of the time). The DERS has high predictive validity and can distinguish between BPD and HC groups (Kuo & Linehan, 2009). Internal reliability is also high, and Cronbach alphas were .97 in the present study.

Depression severity was measured as a covariate via the depression subscale of the Depression, Anxiety, and Stress Scales (DASS; Lovibond & Lovibond, 1995). This subscale asks participants to rate the extent to which 14 statements about their past week (e.g., "I felt that I had nothing to look forward to") apply to them on a scale from 0 (does not apply to me at all) to 3 (applied to me very much or most of the time). The DASS subscales can distinguish those with

and without diagnoses of MDD (Kemp, Quintana, Felmingham, Matthews, & Jelinek, 2012), and the Cronbach alpha for the depression subscale was .97 in the present study.

Insomnia behaviors. TIB, SE, RTV, and chronotype were measured via *The Consensus Sleep Diary (CSD*; Carney et al., 2012), a daily measure of information about a respondent's last sleep episode (e.g., time went to bed, fell asleep, woke, arose, and the number and durations of times awakenings). Participants were asked to complete it each morning immediately after rising, as this time of day provides the most reliable estimates (Carney et al., 2012). Participants completed the CSD from home for 14 consecutive days to ensure robust stability of sleep estimates (Wohlgemuth, Edinger, Finns, & Sullivan, 1999). TIB reflects the number of hours and minutes between the times individuals went to bed and rose per night. SE is calculated by dividing the total time spent sleeping by the time spent in bed per night, with higher SE indicating that participants spent almost all TIB asleep. RTV is computed by subtracting the latest risetime recorded on the CSD from the earliest one. Chronotype was measured via participant's bedtimes (i.e., times they got into bed) and risetimes (i.e., times they woke up). We also measured chronotype via the *Morningness-Eveningness Questionnaire (MEQ;* Horne & Östberg, 1976). The MEQ is a 19-item measure that asks participants about experiences (e.g., "at what time of day do you think that you reach your "feeling best" peak?") that indicate a tendency towards "morningness" versus "eveningness." Higher and lower scores reflect greater biological imperatives towards morning and evening "types", respectively (Horne & Östberg, 1976). The MEQ has robust psychometric properties, with a Cronbach alpha of .87 in the present study and high convergent validity with related scales (Caci, Deschaux, Adan, & Natale, 2009).

Procedure

All study procedures received approval from relevant institutional review boards. If eligible based on phone or online screens, participants were invited to come into the laboratory for further psychodiagnostic assessments, after which eligible participants then completed the CSD for 14 consecutive days. As part of the parent study, mid-way through CSD completion, participants returned to the laboratory for an experiment that utilized brief emotion inductions to examine psychophysiological responses to emotion evocation and attempts to regulate them (data not reported here). Participants completed questionnaires assessing BPD severity, depression severity, insomnia severity, and chronotype on this experiment day.

Data Analytic Strategy

Skew statistics for most outcome variables indicated that they were generally normally distributed (skew statistics ranged from -.05 to 1.04) with three exceptions: TIB (skew statistic = 1.52), SE (skew statistic = -2.37), and RTV (skew statistic = 2.49). We squared the negatively skewed variables and applied a logarithmic transformation (base 10) to the positively skewed variables to yield more normal distributions for TIB (transformed skew statistic = -1.09), SE (transformed skew statistic = -1.36), and RTV (transformed skew statistic = -.18), and entered these variables as outcomes for their respective analyses.

Group differences on time-varying outcomes from the CSD (i.e., TIB, SE, bedtime and risetime) were examined using generalized estimating equations (GEE; Burton, Gurrin, & Sly, 1998; Diggle, Heagerty, Liang, & Zeger, 2013; Hubbard et al., 2010) with SPSS version 25. GEE is a semi-parametric analysis that can assess continuous variables over time. It is robust to covariance structure misspecification because it derives individual point estimates and estimates models based on repeated measure covariance structures (Burton et al., 1998). GEE analyses were run separately for each time-varying outcome (i.e., TIB, SE, bedtime, and risetime). Group

was entered as a between-subjects predictor, and depression severity and number of psychiatric medications as a within-subjects covariates. For time-invariant outcomes (i.e., RTV and MEQ), univariate analyses of covariance (ANCOVAs) were conducted with group as a between-subjects predictor and depression severity and number of psychiatric medications as covariates. Depression severity and number of psychiatric medications were median-centered.

GEE analyses were also run to examine the influence of insomnia behaviors on emotion dysregulation. Total DERS score was entered as the outcome. Group was entered as a between subjects predictor, and depression severity and number of psychiatric medications were entered as time-invariant covariates, TIB and SE as time-varying predictors, and RTV as a time-invariant predictor. There were three possible variables that could be entered to index chronotype: risetime, bedtime, and chronotype per the MEQ. We choose one to avoid duplicating predictors that reflect the same construct. Given the conflation of bedtime with behavioral sleep patterns (e.g., setting an alarm), and that risetime values were utilized in the RTV predictor, we entered the MEQ as a time-invariant chronotype predictor. We then entered two-way interactions between group and each insomnia behaviors to examine whether their influence on emotion dysregulation varied across groups. For all GEE analyses, unstructured, autoregressive, and exchangeable covariance structures were considered and the one with the lowest Quasilikelihood under the Independence Model Criterion (QIC) was retained.

Results

See Table 3 for means and standard deviations of study variables across groups. Only .32% of questionnaire data, and 5.6% of CSD entries, were missing. A Little's Missing Completely At Random Test was non-significant, χ^2 (6436) = 187.12, p = 1.00, indicating that data were missing completely at random. A univariate ANOVA revealed that there was a

significant main effect of group for insomnia severity, F (2, 115) = 13.91, p < .001, partial η^2 = .20. Post-hoc contrasts revealed that both the BPD and GAD groups exhibited higher insomnia severity than the HCs, F (1, 115) = 15.89, p < .001, partial η^2 = .12, and F (1, 115) = 27.21, p < .001, partial η^2 = .19, respectively, but did not differ from each other, F (1, 115) = .15, p = .70.

See Table 4 for GEE analyses examining group differences in insomnia behaviors. There was a statistically significant main effect of group on SE and a marginally significant main effect of group on risetime, but not on TIB or bedtime. For SE, the GAD group exhibited lower SE than the HC group, (β = -.07, *SE* = .03), χ^2 (1) = 6.25, p = .01, but did not differ from the BPD group, χ^2 (1) = 2.71, p = .10. Further, the BPD and HC groups did not differ in SE, χ^2 (1) = .28, p = .60. For risetime, HCs did not exhibit distinct risetimes from either the BPD or GAD groups, χ^2 (1) = 2,41, p = .12 and χ^2 (1) = 4.70, p = .49, respectively. However, the BPD group exhibited significantly later risetime than the GAD group (β = .95, *SE* = .39), χ^2 (1) = 5.87, p = .02. ANCOVAs revealed that there were no statistically significant main effects of group on RTV, F (2, 115) = .39, p = .68, or chronotype as measured by the MEQ, F (2, 115) = 2.13, p = .12.

Table 5 presents GEE analyses of insomnia behaviors predicting emotion dysregulation. There were statistically significant interactions between group and both TIB and SE on emotion dysregulation. Higher TIB did not predict emotion dysregulation in the BPD group, (χ^2 (1) = ,27, p = .60), predicted higher emotion dysregulation in HCs (B = .004, SE = .001, χ^2 (1) = 8.50, p < .01), and lower emotion dysregulation in the GAD group (B = -.01, SE = .002, χ^2 (1) = 7.16, p = .01). Higher SE predicted higher emotion dysregulation in the BPD group (B = .06, SE = .03, χ^2 (1) = 4.99, p = .03), but not in HCs (χ^2 (1) = 3.32, p = .07) or the GAD group (χ^2 (1) = -.10, p = .06). No other insomnia behaviors predicted emotion dysregulation.

Discussion

This study aimed to specify the insomnia behaviors that are elevated and exacerbate emotion dysregulation in BPD. Two homeostatic (TIB, SE) and two circadian (RTV, chronotype) insomnia behaviors were investigated. We hypothesized that individuals with BPD would exhibit higher TIB, lower SE, and higher RTV than HC groups. Contrary to study hypotheses and extensive research (Winsper et al., 2017), we did not identify differences in TIB, SE, or RTV in BPD groups relative to HCs. Although individuals with BPD did not exhibit SE distinct from HCs or individuals with GAD, the GAD group exhibited lower SE than HCs. Unlike in BPD, insomnia is a diagnostic criterion of GAD (APA, 2000; 2013). Individuals in the GAD group may have more "classic" insomnia profiles, including lower SE, than the BPD group, who may present with a confluence of sleep problems including but not limited to insomnia. Alternatively, prior research shows that those with BPD may exhibit comparable SE to HC groups when controlling for depression (Weibel et al., 2017). This study corroborates those findings and suggests that disrupted SE in BPD may be accounted for by high rates of comorbid depression in BPD. Furthermore, the GAD group was recruited based on DSM-IV-TR criteria, which stipulates that depressive disorders must not be primary relative to GAD (APA, 2000). Therefore, the GAD group had lower rates of current MDD (20% versus 30%) and dysthymia (2.5% versus 22.5%) than the BPD group. This feature of GAD criteria may have inadvertently resulted in the recruitment of two clinical groups with "distinct profiles" of insomnia – one that is primarily accounted for by depression in the BPD group, and one that is not in the GAD group. Controlling for depression severity may have thus eliminated significant differences between BPD and HC groups more than between GAD and HC groups. Comparing BPD to other clinical groups could illuminate the relative impact of depression on insomnia behaviors across groups.

Although there were no group differences in RTV or chronotype as measured by the MEQ, marginally significant findings suggest that individuals with BPD may have risetimes than those with GAD. The discrepant findings between the risetime variable and the MEQ may be attributed to statistical power given that the MEQ was only measured at one time point and thus the potential power to detect effects would be considerably lower for analyses with it as the outcome rather than risetime. These findings suggest that individuals with BPD and GAD get into bed at equivalent times, but those with BPD may wake up later, potentially exhibiting a phase delayed chronotype. Research has previously associated such phase delayed chronotypes with adverse BPD-relevant outcomes (e.g., mood disruptions, impulsivity, substance use, aggression; Taylor & Hasler, 2018). There may be a shared underlying biological vulnerability for phase delayed chronotypes and BPD that could account for this association, and/or failing to accommodate a phase delayed chronotype may disrupt sleep and, consequently, lead to BPD-relevant sequelae and criteria. However, this effect was only marginally significant, and future researchers are thus advised to investigate these possibilities further.

There were no group differences in RTV suggesting that, although individuals with BPD may rise later than individuals with GAD, they do so consistently. Alternatively, potential abnormalities in RTV and TIB in those with BPD may have been masked because participants were excluded if they were taking psychiatric medications other than SSRIs, eliminating those who were prescribed sleep medications and may have had particularly severe insomnia.

The influence of insomnia behaviors on emotion dysregulation

Findings partially supported our hypothesis that, across groups, higher TIB, lower SE, and higher RTV would predict higher emotion dysregulation. Specifically, higher TIB predicted lower emotion dysregulation in the GAD group, higher emotion dysregulation in HCs, and did

not predict emotion dysregulation in the BPD group. Further, higher SE predicted higher emotion dysregulation in the BPD group but not the other groups. These findings highlight the importance of examining the impact of insomnia behaviors on emotion dysregulation in specific groups. They are also in direct opposition with theory and research suggesting that lower TIB and higher SE would predict *lower* emotion dysregulation across populations (Gruber & Cassoff, 2014, Kahn et al., 2013). Consistent with insomnia models (Spielman et al., 1987), higher TIB in HCs may reflect increased insomnia behaviors that could exacerbate emotion dysregulation over time. In GAD, a disorder characterized by avoidance (e.g., Borkovec, Alcaine, & Behar, 2004), higher TIB may reflect greater attempts to avoid engaging in life, its stressors, and the broader external world. Consequently, higher TIB may result in the appearance of lower emotion dysregulation in this group, because they have refrained from encountering stimuli or situations that would elicit it. Alternatively, adequate sleep is instrumental in replenishing emotion regulation capacity (Gruber & Cassoff, 2014, Kahn et al., 2013). The heightened arousal and worry associated with GAD (APA, 2013) may entail particularly high emotion regulatory demands, and consequently require more sleep to meet them. Thus, individuals with GAD may need more TIB in order to manage their heightened emotion dysregulation. However, such assertions oppose established insomnia and emotion theories that increasing TIB amidst high arousal is likely to promote insomnia and poor sleep, exacerbating emotion dysregulation (Gruber & Cassoff, 2014; Kahn et al., 2013; Spielman et al., 1987). The aforementioned speculation also fails to explain why this finding would be observed in the GAD group and not the BPD group, which is also characterized by heightened emotion dysregulation. Future research should examine the impact of reducing TIB on emotion dysregulation in GAD.

Similarly, it is unclear why higher SE in BPD is associated with higher emotion dysregulation. Sleep deprived individuals may benefit from *extending* TIB in order to allow for longer sleep durations. Given that the average SE values in the BPD population approximated normal, sleep problems in this group may be highly heterogeneous, wherein some individuals present with low SE caused by extended TIB, and others present with sleep deprivation and high SE. Sleep deprivation exacerbates emotion dysregulation (Gruber & Cassoff, 2014). If individuals with BPD and high SE are sleep deprived, then it is logical that higher SE would predict heightened emotion dysregulation for them. Indeed, several individuals in the BPD group may be more sleep deprived because they have a phase delayed chronotype which is not accommodated well, perpetuating sleep deprivation and consequently high SE. Heightened SE variability in the BPD group could also explain why this group did not exhibit differences in SE relative to HCs, and TIB did not influence emotion dysregulation for them. If some individuals with BPD exhibit low SE (due to high TIB), whereas others report high SE (due to sleep deprivation), then the average SE in this group may appear "normal" and mask important heterogeneity. In the context of such heterogeneity, increasing TIB could worsen emotion dysregulation for some, but improve it for others. These opposing effects may have collectively resulted in the appearance of a lack of a relationship between TIB and emotion dysregulation in BPD. Future research should further investigate the potential heterogeneity of sleep in BPD.

In addition, circadian insomnia behaviors did not predict emotion dysregulation across groups, suggesting that homeostatic insomnia behaviors may be particularly influential in exacerbating emotion dysregulation. However, there are several ways to measure emotion dysregulation including direct assessment of specific emotion processes (e.g., emotional intensity, emotional reactions) and the selection and use of specific emotion regulation strategies

(e.g., Gross & Jazaieri, 2014). In the present study, emotion dysregulation was measured as a multifaceted construct reflecting broad problems with emotion (e.g., difficulties with emotional acceptance, impulsivity). Circadian insomnia behaviors may influence emotion dysregulation domains not assessed in the present study, and future research should continue to examine this.

Limitations and Clinical Implications

It is possible that the sample size in the present study was too small to detect its hypothesized effects, and future work should therefore examine the present questions in larger samples. In addition, given the vast number of ways that emotion dysregulation can be measured, the use of one self-report measure of it is a limitation. There are also other non-behavioral factors that influence insomnia not examined in this study such as worry about sleep (Harvey, 2002) and hyperarousal (Riemann et al., 2010), which future work should examine in relation to BPD. Other core aspects of BPD pathology may also be affected by insomnia behaviors, such as impulsivity and cognitive/identity disturbances, but their relationship to insomnia behaviors in BPD is unclear. Future researchers are therefore advised to investigate the relationship between insomnia behaviors and BPD pathology broadly. Furthermore, although the sleep diaries used to measure insomnia behaviors are an empirically supported assessment method (Maich, Lachowski, & Carney, 2016), they are nonetheless subjective. Future studies should examine insomnia behaviors broadly across objective measures (e.g., polysomnography). Future research should also examine insomnia behaviors in individuals taking medications other than SSRIs, and clinical groups who are not characterized by emotion dysregulation, as they may exhibit distinct insomnia profiles than the groups examined in this study. Finally, it is notable that, although prospective individuals were excluded from the GAD group if they had high BPD pathology, individuals were not excluded from the BPD group if they had comorbid GAD, although many

did. GAD and BPD groups may thus not be entirely distinct, which may account for the null findings observed between them. Moreover, given that GAD is fundamentally characterized by sleep difficulties (American Psychiatric Association, 2013), the high rates of GAD in the BPD group may account for the insomnia related behaviours observed this group. However, the majority of our findings indicate that the presence of insomnia behaviors, or their influence on emotion dysregulation, is distinct in BPD and GAD groups, suggesting that GAD likely does not fully account for these effects. Despite this, future researchers are advised to disentangle GAD and BPD with greater precision in order to identify whether insomnia behaviours are unique to one group versus the other, or pervasive across them.

Despite these limitations, this study bears key clinical implications. Study findings emphasize the importance of addressing extended TIB in individuals with GAD and high SE in those with BPD, but suggest that thorough investigations delineating the function of TIB, and the cause of abnormal SE, may be important. Assuming that SE *must* be increased in BPD populations to improve emotion dysregulation is likely to lead to imprecise and ineffective treatment; we recommend a patient-specific and case formulation-driven approach to insomnia behaviors with this population. Results also indicate that it may be important to examine potential phase delayed chronotypes in BPD in both clinical practice and research and identify whether failing to accommodate them may exacerbate BPD pathology. In addition, study findings suggest that overemphasis of establishing routine wake and risetimes may not influence emotion dysregulation in BPD, although more research is needed to examine the influence of circadian processes on emotion dysregulation. Ultimately, this study highlights the importance of idiographic and comprehensive assessment of insomnia behaviors across clinical groups, which may present a significant opportunity to improve BPD treatment.

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Table 1

Demographic breakdown for participants across groups

	HC	BPD	GAD	Tests of group differences
Age [Mean(standard deviation)]	24.70 (6.88)	25.35 (6.77)	25.95 (7.20)	F (2, 114) = .31, p = .73
Number of medications [Mean(standard deviation)]	**.00 (.00)**	**.25 (.49)**	**.23 (.48)**	**F (2, 117) = 4.80, p = .01**
Sex (%)				$\chi^2(2)$ = .17, p = .92
Female	80%	82.5%	78.9%	
Male	20%	17.5%	21.1%	
Gender (%)				$\chi^2(8)$ = 5.07, p = .75
Woman	77.5%	77.5%	76.3%	
Man	20%	17.5%	21.1%	
Transgender	2.5%	0%	0%	
Gender queer	0%	2.5%	2.6%	
Other	0%	2.5%	0%	
Ethnicity (%)				$\chi^2(22)$ = 28.20, p = .17
Chinese or Chinese-Canadian	30%	22.5%	10.5%	
Other Asian or Asian-Canadian	22.5%	12.5%	13.2%	
White/Caucasian/European Origin	15%	40%	34.2%	
Black-Canadian/Black/Caribbean Origin	10%	5%	5.3%	
Mexican or Mexican-Canadian	5%	0%	0%	
Other Hispanic/Latino	5%	0%	5.3%	
Middle Eastern	5%	0%	5.3%	
Korean or Korean-Canadian	2.5%	2.5%	0%	
Bi-racial/multi-racial	2.5%	2.5%	7.9%	
Aboriginal-Canadian/First Nations/Metis/Inuit	0%	2.5%	0%	
East Indian	0%	2.5%	5.3%	
Other	2.5%	10%	13.2%	
Marital Status				**$\chi^2(10)$ = 21.55, p = .02**
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	0%	5%	0%	
Divorced	0%	2.5%	0%	
Other	0%	2.5%	0%	

Note. BPD = borderline personality disorder; HC = healthy controls; GAD = generalized anxiety disorder group

Table 2

Diagnostic breakdown across BPD and GAD groups

	BPD		GAD		Tests of Group Differences	
	Current	Past	Current	Past	Current	Past
Major Depressive Disorder	30%	40%	20%	55%	$\chi^2(1) = 1.07$, $p = .30$	$\chi^2(1) = 1.81$, $p = .18$
Dysthymic Disorder	**22.5%**		**2.5%**		**$\chi^2(1) = 7.31$, $p = .01$**	
Bipolar II Disorder	5%	0%	2.5%	0%	$\chi^2(1) = .35$, $p = .56$	
Other Bipolar Disorder	0%	5%	0%	0%		$\chi^2(1) = 2.05$, $p = .15$
Substance induced mood disorder	2.5%	0%	0%	0%	$\chi^2(1) = 1.01$, $p = .31$	
Brief psychotic disorder	0%	2.5%	0%	0%		$\chi^2(1) = 1.01$, $p = .31$
Psychotic disorder NOS	2.5%	0%	0%	0%	$\chi^2(1) = 1.01$, $p = .31$	
Alcohol abuse	**12.5%**	**2.5%**	**0%**	**5%**	**$\chi^2(1) = 5.33$, $p = .02$**	**$\chi^2(1) = .35$, $p = .56$**
Alcohol dependence	0%	27.5%	0%	12.5%		$\chi^2(1) = 2.81$, $p = .09$
Substance abuse	5%	7.5%	2.5%	0%	$\chi^2(1) = .35$, $p = .56$	$\chi^2(1) = 3.12$, $p = .08$
Substance dependence	0%	20%	0%	7.5%		$\chi^2(1) = 2.64$, $p = .11$
Panic Disorder	15%	5%	7.5%	15%	$\chi^2(1) = 1.13$, $p = .29$	$\chi^2(1) = 2.22$, $p = .14$
Agoraphobia	10%	0%	7.5%	7.5%	$\chi^2(1) = .16$, $p = .69$	$\chi^2(1) = 3.12$, $p = .08$
Agoraphobia without a history of panic disorder	5%	2.5%	2.5%	0%	$\chi^2(1) = .35$, $p = .56$	$\chi^2(1) = 1.01$, $p = .31$
Social anxiety disorder	45%	12.5%	42.5%	10%	$\chi^2(1) = .05$, $p = .82$	$\chi^2(1) = .13$, $p = .72$
Specific phobia	17.5%	5%	25%	2.5%	$\chi^2(1) = .67$, $p = .41$	$\chi^2(1) = .35$, $p = .56$
Obsessive compulsive disorder	32.5%	17.5%	15%	7.5%	$\chi^2(1) = 3.38$, $p = .07$	$\chi^2(1) = 1.83$, $p = .18$
Posttraumatic stress disorder	15%	7.5%	2.5%	2.5%	$\chi^2(1) = 3.91$, $p = .05$	$\chi^2(1) = 1.05$, $p = .31$
Generalized anxiety disorder	**40%**	**0%**	**100%**	**0%**	**$\chi^2(1) = 34.29$, $p < .001$**	
Anorexia nervosa	5%	5%	2.5%	2.5%	$\chi^2(1) = .35$, $p = .56$	$\chi^2(1) = .35$, $p = .56$
Bulimia nervosa	5%	7.5%	0%	0%	$\chi^2(1) = .35$, $p = .56$	$\chi^2(1) = 3.12$, $p = .08$
Binge eating disorder	2.5%	0%	0%	2.5%	$\chi^2(1) = 1.01$, $p = .31$	$\chi^2(1) = 1.01$, $p = .31$
Eating disorder not otherwise specified	5%	0%	2.5%	2.5%	$\chi^2(1) = .35$, $p = .56$	$\chi^2(1) = 1.01$, $p = .31$
Insomnia	**77.5%**	**2.5%**	**27.5%**	**7.5%**	**$\chi^2(1) = 20.05$, $p < .001$**	**$\chi^2(1) = 1.05$, $p = .31$**

Note. BPD = borderline personality disorder; GAD = generalized anxiety disorder; NOS = not otherwise specified; Disorders with significant group differences are bolded.

Table 3

Means (standard deviations) of study variables across groups

	HC	BPD	GAD
Insomnia severity[a, c]	8.08 (3.40)	17.28 (4.87)	16.18 (5.44)
BPD severity[a, b, c]	.13 (.16)	1.86 (.82)	.95 (.67)
Emotion dysregulation[a, b, c]	62.33 (18.28)	122.28 (17.27)	94.45 (22.63)
Depression severity[a, b, c]	1.50 (2.35)	19.85 (10.30)	11.48 (9.19)
Time in Bed[a, c]	8.59 (1.94)	9.48 (2.65)	9.29 (2.67)
Sleep Efficiency[a, c]	.91 (.10)	.83 (.15)	.83 (.15)
Risetime Variability[b]	4.45 (2.61)	5.00 (1.89)	4.74 (3.27)
Chronotype (MEQ)[a, b]	49.33 (12.03)	41.40 (10.47)	47.20 (9.88)
Bedtime	24.29 (1.76)	24.26 (2.17)	23.80 (2.14)
Risetime	8.53 (1.80)	9.24 (2.55)	8.46 (2.00)

Note. HC = healthy control; BPD = borderline personality disorder; GAD = generalized anxiety disorder; MEQ = Morningness-Eveningness Questionnaire (Horne & Ostberg, 1976)

[a] Significant group differences between HC and BPD groups at $p < .05$.

[b] Significant group differences between BPD and GAD groups at $p < .05$.

[c] Significant group differences between HC and GAD groups at $p < .05$.

Table 4

Generalized estimating equations analyses examining group differences in insomnia behaviors

	B	*SE*	χ^2	df	*p*-value
Time in Bed (log transformed)					
Intercept	**.93**	**.01**	**17878.38**	**1**	**<.001**
Group[a]			2.93	2	.23
Number of Medications	**.06**	**.01**	**18.93**	**1**	**<.001**
Depression severity	.00	.001	.39	1	.77
Sleep efficiency (squared)					
Intercept	.80	.02	5544.35	1	<.001
Group[a]			**7.31**	**2**	**.03**
Number of Medications	.02	.02	.52	1	.47
Depression severity	**-.01**	**.001**	**15.05**	**1**	**<.001**
Bedtime					
Intercept	**24.32**	**.21**	**31674.21**	**1**	**<.001**
Group[a]			4.55	2	.10
Number of Medications	.29	.32	.84	1	.36
Depression severity	.004	.02	.07	1	.79
Risetime					
Intercept	**8.61**	**.20**	**3496.83**	**1**	**<.001**
Group[a]		**.32**	**6.02**	**2**	**.05**
Number of Medications	**1.18**	**.38**	**9.63**	**1**	**.002**
Depression severity	-.02	.02	.90	1	.34

Note. SE = standard error; df = degrees of freedom. Hypothesis-relevant significant main effects are bolded. CBT-I = Cognitive Behavioral Therapy for Insomnia (Edinger & Carney, 2015).

[a] Effect sizes and standard errors are not presented for group because group has multiple levels and therefore multiple effect sizes. Effect sizes for comparisons between each level of group and the reference category are presented in text when main effects of group are significant.

Table 5

Generalized estimating equations analyses examining whether insomnia behaviors predict emotion dysregulation

	B	*SE*	χ^2	df	*p*-value
Intercept	**88.35**	**2.86**	**2184.63**	**1**	**<.001**
Group[a]			**47.45**	**2**	**<.001**
Number of medications	**9.32**	**4.69**	**3.94**	**1**	**.05**
Depression severity	**1.08**	**.20**	**28.49**	**1**	**<.001**
Time in bed	-.01	.002	.03	1	.87
Sleep efficiency	-.07	.06	1.60	1	.21
Risetime variability	-.52	.71	.10	1	.75
Chronotype	-.17	.28	.37	1	.54
Group × time in bed[a]			**15.35**	**2**	**<.001**
Group × sleep efficiency[a]			**8.40**	**2**	**.02**
Group × risetime variability[a]			.62	2	.73
Group × chronotype[a]			1.72	2	.42

Note. BPD= borderline personality disorder; SE = standard error; df = degrees of freedom. Hypothesis-relevant significant main effects are bolded. CBT-I = Cognitive Behavioral Therapy for Insomnia (Edinger & Carney, 2015).

[a] Effect sizes and standard errors are not presented for group or interactions involving group because group has multiple levels and therefore multiple effect sizes. Effect sizes for comparisons between the relationship between insomnia behaviors and emotion dysregulation across each level of group relative to the reference category are presented in text when interactions involving group are significant.