

What Dreams May Come: Emotional Cascades and Nightmares in Borderline Personality Disorder

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People diagnosed with borderline personality disorder (BPD) have been found to have a number of sleep problems, including frequent and distressing nightmares. The experience of nightmares is likely to worsen emotion dysregulation and decrease coping abilities the subsequent day, making it an important issue for clinicians to address. One recent theoretical model of BPD psychopathology, the Emotional Cascade Model (ECM), may shed light on this phenomenon by characterizing nightmares as the experience of emotional cascades that occur during sleep. A model is presented in which these cascades may carry over from a stressful day and lead to elevated cognitive activity during sleep, as well as nightmare-like phenomena. To test this model we used experience sampling from 47 participants exhibiting dysregulated behaviors—16 of them diagnosed with BPD. Negative emotion, rumination, and number of nightmares were assessed daily across two consecutive weeks. Analyses indicated that the BPD group experienced more frequent nightmares, that BPD diagnosis interacted with baseline trait rumination to prospectively predict number of nightmares reported during monitoring, and daily experience of emotional cascades predicted subsequent number of nightly nightmares. These findings held after controlling for key covariates, including sleep quality and diagnoses of depression and posttraumatic stress disorder. Important clinical interventions consistent with the ECM conceptualization of nightmares are proposed, including the potential for management of daily rumination and negative emotion, imagery rescripting for recurrent or anxiously anticipated nightmares, and potential prescription of prazosin (an alpha1-adrenergic antagonist) for the reduction of nightmares in this group.

Keywords: nightmares, borderline personality disorder, emotional cascades, waking nightmares, rumination

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For in that sleep of death, what dreams may come . . .
Shakespeare, *Hamlet*

Borderline personality disorder (BPD) is characterized by intense emotion reactivity and functional problems across various interpersonal and situational domains. Recently, research has indicated that these intense emotional experiences may even extend into the realm of sleep, as those with BPD and BPD-like features have been found to experience frequent and intense nightmares (Agargun et al., 2003; Asaad, Okasha, & Okasha, 2002; Claridge, Davis, Bellhouse, & Kaptein, 1998; Lancee & Schrijnemaekers, 2013; Schredl, 2003; Wood & Bootzin, 1990; Wood, Bootzin, Rosenhan, Nolen-Hoeksema, & Jourden, 1992). Despite the link between BPD and nightmares, no hypotheses have been presented for why those with BPD may have such difficulty with nightmares. The purpose of this study was to examine the potential role of emotional cascades in the nightmare experiences of those with BPD using experience sampling methodology.

NIGHTMARES AND SLEEP DISTURBANCE IN BORDERLINE PERSONALITY DISORDER

People with BPD tend to experience a number of sleep problems in addition to nightmares, ranging from insomnia (Bastien, Guimond, St-Jean, & Lemelin, 2008) to abnormal sleep architecture. Relative to healthy controls, those with BPD spend more time in REM sleep than healthy controls (De la Fuente, Bobes, Vizquete, & Mendlewicz, 2001) and have denser first-cycle REM (Battaglia, Strambi, Bertella, Bajo, & Bellodi, 1999). Furthermore, in comparison to healthy controls those with BPD have more stage 1 sleep and less stage 4 sleep (Benson, King, Gordon, Silva, & Zarcone, 1990).

Although these sleep issues are likely to aggravate both psychopathology and nightmares, nightmares themselves are often distressing and salient experiences. Nightmares are defined as vivid manifestations of escalating cognitive and emotional experiences that occur primarily during REM, and they frequently result in frequent and terrifying awakenings that can interfere with overall sleep quality (Levin & Nielsen, 2007). However, the exact definition of nightmares is still being debated, with factors such as subjective distress of the nightmare, awakening from the nightmare, and ability to recall content of the nightmare all being important factors in defining nightmares (Levin & Nielsen, 2007). The experience of nightmares has been linked to longer wake time after sleep onset (Woodward, Arsenaault, Murray, & Bliwise, 2000), and elevated anxiety and physical complaints the following day (Köthe & Pietrowsky, 2001). Importantly, frequency of nightmares has also been linked to an emotionally reactive personality style (Levin & Nielsen, 2007).

Although a number of studies have documented an association between nightmares and BPD, data are not extensive. One study found that relative to age- and gender-matched healthy controls, those with BPD reported experiencing more nightmares, bad dreams, night terror symptoms, and dream anxiety during a 3-week follow-up period (Simor, Csóka, & Bódizs, 2010). Those with BPD and nightmares tend to report a fear of sleeping because of anticipated nightmares (Agargun et al.,

2003; Semiz, Basoglu, Ebrinc, & Cetin, 2008). In addition, those with BPD have been found to have elevated rates of comorbid nightmare disorder (Semiz et al., 2008), and they also have high comorbidity rates with posttraumatic stress disorder (PTSD), a disorder for which nightmares are a key symptom (Mellman & Pigeon, 2005). On top of multiple sleep problems and nightmare-prone comorbidities, those with BPD often experience suicidality, and studies have found suicide risk to be associated with more frequent nightmares (Bernert, Joiner, Cukrowicz, Schmidt, & Krakow, 2005; Sjöström, Wærn, & Hetta, 2007; Tanskanen et al., 2001). Furthermore, it has yet to be determined whether there is a unique link between BPD and nightmare experiences, or if this link may simply be a function of comorbid disorders more commonly associated with nightmares, such as posttraumatic stress disorder (PTSD). Although much more research is needed to understand sleep problems in BPD, shedding some light on the factors that can cause or contribute to nightmares in this disorder is important for generating clinical interventions in this population.

EMOTIONAL CASCADE MODEL

One potential explanation for why those with BPD have elevated nightmares may come from the Emotional Cascade Model (Selby & Joiner, 2009; Selby, Anestis, Bender, & Joiner, 2009). This model proposes that people, especially those with BPD, can experience “emotional cascades,” which are upsetting emotional experiences potentiated via rumination and subsequent escalation of negative emotions. Rumination is a process during which people repetitively think about an upsetting situation, how it relates to past problems, and the future problems predicated by the situation (Nolen-Hoeksema, 1991). It has been found to reliably exacerbate negative emotional states (Thomsen, 2006). Elevations of negative emotion resulting from rumination have in turn been found to reciprocally increase rumination (Moberly & Watkins, 2008). Thus, the ECM suggests that a similar set of processes is at play with individuals suffering from BPD: when individuals with BPD ruminate it makes them more upset, and this interacts with increasing negative emotion, which serves to further increase rumination. As a result of this self-amplifying positive feedback loop between intense rumination and negative emotion, an extremely aversive emotional state develops, often leading to dysregulated behaviors used for affect regulation, such as substance use, binge eating, fighting, and/or self-injury. Essentially, in this model emotional cascades are thought to be responsible for the affective lability displayed by a majority of patients with BPD (Selby & Joiner, 2009). Of note, cross-sectional, experimental, and ecological-longitudinal data have provided support for this model (Selby, Anestis, & Joiner, 2008; Selby et al., 2009; Selby, Connell, & Joiner, 2010; Selby & Joiner, 2012).

EMOTIONAL CASCADE MODEL AND NIGHTMARES

Though nightmares represent a significantly impairing and distressing clinical concern, nightmare pathogenesis remains unclear. Of theoretical models proposed,

the AMPHAC network (Amygdala-Medial Prefrontal Cortex-Hippocampus-Anterior Cingulate Cortex) neurocognitive model of disturbed dreaming (Levin & Nielson, 2007; Nielsen & Levin, 2007) appears to be particularly promising, offering a comprehensive and integrative account drawing from cognitive neuroscience, sleep physiology, and fear conditioning literature. According to this model, normal dreaming is involved in fear-extinction processes and nightmares represent a failure in emotion regulation. In this model, two primary processes are implicated in nightmare pathogenesis: affect load (i.e., a transitory factor that refers to ongoing buildup of emotionally valenced negative events that can weaken an individual's capacity for emotion regulation) and affect distress (i.e., a dispositional factor that refers to a tendency to experience heightened distress in response to emotional stimuli). Thus, in short, this model would suggest that emotionally vulnerable individuals experiencing daily negative events are at heightened risk of nightmare experiences.

Emotional cascades, therefore, may be risk factors for initiating and/or exacerbating nightmares experienced during sleep. In extending the ECM to nightmare pathogenesis in BPD individuals in particular, emotional cascades experienced in response to stressful situations during the day may elevate cognitive arousal during sleep, potentially resulting in the continued experience of emotional cascades while sleeping. In line with this hypothesis, studies have found cognitive arousal continues into sleep (Burton, Harsh, & Badia, 1988; Cicogna, Cavllero, & Bosinelli, 1991). In addition, another study found that the amygdala demonstrated elevated responsiveness to dream stimuli, which may be because REM sleep produces imagery and sensations of almost-real quality (Nielsen & Stenstrom, 2005). Dream stimuli may therefore contribute to the positive feedback loop of emotional cascades experienced while sleeping. The associated cognitive arousal from emotional cascades may contribute to dream/nightmare stimuli, which may result in amplified emotional experience and contribute further to emotional cascades while sleeping. The details of this framework are displayed in Figure 1.

Based on this model, those who experience more cognitive arousal, due to higher levels of rumination (both trait and state levels), should also experience more nightmares as a result. It should be noted that increased cognitive arousal

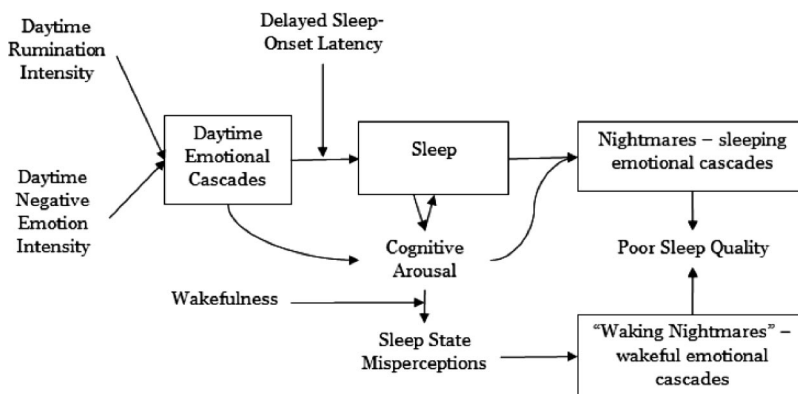


Figure 1. Model for contributions of emotional cascades to nightmares and poor sleep quality in borderline personality disorder.

(i.e., cognitive activity¹) has consistently been found to negatively impact quality of sleep (Tang & Harvey, 2004; Harvey, 2001). The link between worry in particular and sleep disturbance is strong and well documented. Evidence from both cross-sectional and experimental studies consistently support the negative impact of worry on both self-report and objective measures of a host of sleep outcomes (see Harvey, 2001 for a review). It has also been suggested that the negative effects of cognitive arousal may not be limited to worry but also extend to other phenomena that increase cognitive activity—rumination, in particular, as it represents an overlapping (though somewhat distinct²) construct with respect to worry. Self-reported trait rumination as well as experimentally induced rumination has been shown to be associated with poorer subjective sleep quality (Thomsen, Mehlsion, Christensen, & Zacharaie, 2003; Guastella & Molds, 2007). In addition, longer sleep onset latency as measured by objective sleep instruments has been associated with both trait rumination as well as stressor-specific rumination (Zoccola, Dickerson, & Lam, 2009). Although very limited literature exists on the effects of rumination and nightmares in particular, a link has been documented in case studies (e.g., Willner, 2004).

The potential link between BPD, emotional cascades, and nightmares may be further complicated by periods of actual awakening or light sleep, however. Due to cognitive arousal, people may actually rise from deep sleep to experience periods of actual or semiawakening, which they may mistake for sleeping or dreaming, a phenomenon known as positive sleep-state misperception (Trajanovic, Radivojevic, Kaushansky, & Shapiro, 2007). The experience of sleep state misperceptions may also lead to actual experiences of cognitive processes such as worry (Lundh & Broman, 2000). Given these findings, those with BPD may experience sleep state misperceptions during which actual emotional cascades occur, which are then incorrectly interpreted as occurring during sleep and labeled as nightmares. This process of awakening and experiencing a wakeful emotional cascade, but misinterpreting oneself as sleeping, could be considered a nightmare-like experience called *waking nightmares*. Similarly, these experiences may lead to hypnopompic/hypnogogic hallucinations (Cheyne, 2003), which are dream-like phenomena that occur in the transition to and from sleep. Importantly, preliminary evidence suggests that those with BPD may have more problems with sleep state misperceptions than healthy controls (Philipsen et al., 2005), making these waking nightmares a likely possibility. Thus, emotional cascades may contribute to nightmares in those with BPD via two routes: actual initiation and/or aggravation of true nightmares, and experience of nightmare-like experiences that are mistakenly labeled as nightmares. Finally, the experience of nightmares may subsequently result in more emotional cascades the following day, as nightmares may increase negative emotion and vulnerability to daily stressors.

¹ For the current discussion, we refer to the cognitive activity that takes place during sleep as *cognitive arousal*, although it should be noted that this concept is frequently used to describe cognition/rumination that interferes with sleep onset (Harvey, 2002).

² Primarily they are differentiable on the basis of temporal focus—worry is focused on events that have not yet taken place whereas rumination is focused on past events (Watkins, 2008). Yet rumination and worry may be essentially the same process, with different temporal focus.

CURRENT STUDY

In the current study we examined the potential extension of the ECM to nightmares in BPD with the following hypotheses: Hypothesis 1) to examine if the findings of elevated nightmares in BPD relative to a comparison group could be replicated longitudinally with baseline and subsequent daily monitoring data, Hypothesis 2) to determine if trait rumination interacted with BPD diagnosis to prospectively predict frequency of nightmares, as would be indicated by the ECM, and Hypothesis 3) to determine if emotional cascades, defined as the three-way interaction between BPD diagnosis, day-of rumination intensity, and day-of intensity of negative emotion, predicted the frequency of experiencing nightmares that same night. In addition, for Hypothesis 4 we also examined the impact of number of nightly nightmares on next day level of negative emotion and rumination, with the prediction that more nightmares would increase subsequent daily intensity of negative emotion rumination, particularly for those with BPD. To test these hypotheses we utilized experience sampling methodology, where participants completed daily assessments of their experience of nightmares, as well as multiple random recordings each day for assessing their emotional and cognitive states. Those with BPD were compared to those who endorsed dysregulated behaviors in order to demonstrate that simply due to severity of emotional and behavioral problems. Of note, the purpose of the study was not to test the whole model displayed in [Figure 1](#), in part because teasing apart actual nightmares and nightmare-like experiences is particularly difficult and would require multiple nightly assessments in a sleep lab paired with self-reported nightmare documentation—and an assessment in a sleep lab may itself interfere with assessment of nightmares that occur in daily life. Rather, the goal of this study was to examine emotional cascades as potential contributors to subjectively reported nightmare experiences of any kind, which may also include nightmare-like experiences.

METHOD

Participants

This sample consisted of 47 participants, all of whom were recruited for having some self-reported difficulties with dysregulated behaviors. The sample was 66% female. Ethnic breakdown was as follows: 73% Caucasian, 19% African American, 6% Asian American, 2% Native American. Approximately 9% identified as Hispanic and 27% of the sample reported being from low socioeconomic status. For entry in the study, all participants had to report having engaged in at least four dysregulated behaviors, which were “difficult to control,” over the previous 2 weeks, including: binge eating, self-injury, impulsive shopping, reckless driving, alcohol use, marijuana use, physical fighting, and verbal aggression.

Participants were recruited from both community and undergraduate settings, and in the current study both samples were combined as previous research with these data have found no significant group differences on diagnostic presentation, number of BPD symptoms endorsed, or levels of rumination and negative emotion

during the study (Selby & Joiner, 2012). For the student participants, over 2,500 students were first screened through the general psychology department mass screening process. Symptoms of BPD were screened using modified forced yes–no items from the SCID-II (described below). Only those students reporting at least four dysregulated behaviors in the last 2 weeks (at the time of this screening) or endorsing at least five BPD symptoms were invited by email to participate in the study. Overall 20 student participants, out of 87 who were invited (23%), participated in the study. It should be noted that students were compensated with course credit for their participation, so this low rate may have been due to credit not being enough incentive to make up for the time commitment of the study for some of those contacted. There were no significant differences on number of dysregulated behaviors or number of BPD symptoms reported between those who participated and those who were invited but did not respond. Student participants were offered course credit for their participation in the study. In order to enhance compliance with the daily monitoring, students were offered the opportunity to receive additional course credit for completing at least 80% of the random daily assessments.

Community participants ($N = 27$) were recruited through local advertisements and flyers placed in community mental health centers, as well as through advertisements in the volunteer section of online classified websites. Flyers and advertisements listed examples of dysregulated behaviors and symptoms of BPD, indicating that anyone who experiences difficulties with any of these problems was eligible to undergo screening to participate in the study. The same dysregulated behavior criterion described above was applied to community participants for entry into the study, and all community participants who came to the lab for participation in the study met criteria for entry into the study. Community participants were compensated \$50 for completing the study. In order to enhance compliance with the daily monitoring, however, community participants were offered an additional \$50 if they completed at least 80% of the random daily assessments. Both participant samples completed over 80% of the daily assessments and were provided additional credit and monetary compensation for participating in the study.

Procedures

At the start of the study all participants were assessed to ensure that they met the necessary criteria for entry into the study (i.e., at least four dysregulated behaviors reported in the last 2 weeks). They then completed structured clinical interviews for Axis I diagnoses and BPD, and they were also trained on using the portable digital devices (Palm Zire 31 model). All participants who participated in the experience sampling portion of the study carried the device for two practice days, followed by two consecutive weeks of actual monitoring. The devices were programmed to “beep” or alert the participant five times at random between 9:00 a.m. and 9:00 p.m. every day, approximately every 2–3 hours. At each signal, the participant was requested to complete the momentary assessment questionnaire on the device. The participants had the opportunity to “snooze” the device if they were in a situation where completing the form was problematic (e.g., driving); doing so silenced the beeping for 5 minutes. Participants were informed that they needed to

respond to the signal within 1 hr for the record to count toward the compliance incentive previously described.

Baseline Assessment Measures

Cognitive Emotion Regulation Questionnaire (CERQ; Garnefski, Kraaij, & Spinhoven, 2001)

The CERQ is a measure of trait cognitive emotion regulation strategies used, positive and negative, which includes information on ruminative processes. For this study, both the rumination subscale and catastrophizing subscales were combined, consistent with previous studies, due to their similar ruminative qualities and evidence that catastrophizing is a future oriented form of rumination (Selby et al., 2008, 2009). The combination of these two subscales yielded an alpha of .88, which further supported combining the items into one scale. Importantly, the CERQ has consistent correlations with other measures of rumination (Selby et al., 2008), further supporting its use in the current study.

Beck Scale for Suicide Ideation (BSS; Beck & Steer, 1993)

This is a self-report measure of suicidal ideation and intent over the past week. The measure consists of 21 items and items are rated from 0–2; the item scores were summed so that higher scores indicated worse suicidality. In the current study BSS total score demonstrated strong internal consistency ($\alpha = .94$).

Assessment of Axis I diagnoses: Mini International Neuropsychiatric Interview (MINI; Sheehan, Lecrubier, & Sheehan, 1998)

All participants were assessed with the MINI to diagnose potential Axis I psychopathology. All clinical assessments were completed by the first author, who was a masters-level trained clinician at the time of data collection. A second masters-level graduate student in clinical psychology, who was not involved in data collection, independently reevaluated all participant diagnostic interviews and established comparison Axis I and BPD diagnoses (assessment for which is described below) for each participant. Interrater reliability indices indicated that all Axis I diagnoses had adequate interrater reliability of over $\kappa = .80$, and in this study only PTSD and MDD diagnoses were used in analyses.

Assessment of Borderline Personality Disorder

All participants were administered the Structured Clinical Interview for DSM–IV Axis II personality disorders (SCID-II; First, Gibbon, Spitzer, Williams, & Benjamin, 1997) module for BPD. For a diagnosis of BPD, a participant had to have endorsed at least five out of nine diagnostic criteria. The interrater reliability

estimate for BPD diagnosis was $\kappa = .81$, which indicated a high level of diagnostic agreement (Landis & Koch, 1977). In the current study, 16 participants (34%) met criteria for BPD.

Daily Assessments

The following assessments were completed at least once per day and were answered on portable digital devices.

Sleep Assessment

All participants completed information about how they slept the previous night during the first signal they completed each day. They were asked to report their total number of nightmares experienced during the previous night's sleep. Because participants were asked during this protocol to rate their nightmares experienced, and additional information on awakening and distress caused by the nightmare were not collected, the nightmares reported might have included nightmare-like experiences such as bad dreams, hypnopompic/hypnogogic hallucinations, or other experiences, which is a limitation to these data. Participants were also asked during this assessment to rate the number of hours they slept during the previous night ranging from 0 to 10, and their subjective sleep quality ranging from (1) *terrible* to (10) *excellent*. The palm pilot was programmed in such a way that all participants completed these questions for their first daily assessment, regardless of whether or not they missed the first random assessment of the day.

Daily Negative Emotion Assessment

At each signal, participants rated their experience of negative emotions "RIGHT NOW," from 1 (*low*) to 10 (*high*). The specific negative emotions assessed were: sad, angry, worried, ashamed, and numb; all were summed into a single scale and averaged for each day's intensity of negative emotion. These items demonstrated good internal consistency across monitoring for all participants ($\alpha = .75$).

Daily Rumination Assessment

At each signal, participants answered questions about their current thought patterns, a number of which were about "ruminative" thinking. Each question answered was prefaced with the statement, "Please rate how much you are CURRENTLY thinking about the following from 1 (*not at all*) to 10 (*very much so*)." The rumination-specific questions were: "a currently upsetting problem," "upsetting memories," "the emotions that I am feeling," and "negative future situations." The items demonstrated adequate internal consistency across recordings for all participants ($\alpha = .79$). Previous analyses of these data have found this scale to be moderately correlated with the CERQ as a baseline measure of trait

rumination ($r = .45$; Selby & Joiner, 2012), supporting it as a momentary measure of rumination. It should be noted, however, that baseline self-report measures are often biased with retrospective recall, potentially lowering the correlation with actual reported rumination. For this study, rumination level was summed and averaged for each day of monitoring for each participant.

Data Analytic Strategy

Data analyses occurred in three stages. First we examined the total frequency and distribution of nightmares, as well as sleep quality and sleep total, during monitoring as a function of BPD diagnosis (Hypothesis 1). Next, using generalized hierarchical linear regression models, we examined the prospective predictive value of the interaction between BPD and baseline rumination in predicting total number of nightmares reported during monitoring, while controlling for key variables (Hypothesis 2). We accounted for the count nature of the nightmares outcome variable, which was not normally distributed, using a Poisson distribution and log link function.

Next, we used hierarchical generalized linear modeling to examine the intensity of daily emotional cascades in BPD, defined as the three-way interaction between BPD diagnosis, day-of rumination intensity, and day-of negative emotion intensity, in predicting the number of nightmares experienced the subsequent night (Hypothesis 3). The day-of variables consisted of the daily average of rumination or negative emotion across assessments. This model required a hierarchical approach because the emotional, ruminative, and nightmares variables were all nested within days for each individual participant. Again, because number of nightly nightmares was a count variable, a Poisson distribution with log link function was used. The model was specified to have a random intercept, but all other effects were fixed. The detailed model equations are displayed below:

$$\text{Response distribution: } Bx_{ij} \mid \mu_{ij} \sim \text{BER}(\mu_{ij})$$

$$\text{Link function: } \eta_{ij} = \log(\mu_{ij})$$

$$\text{Level 1 (day). } \eta_{ij}(\text{Nightmares}) = \beta_{0j} + \beta_{1j}(\text{Rumination}_{\text{DayIntensity}})_i + \beta_{2j}(\text{Negative Emotion}_{\text{DayIntensity}})_i + \beta_{3j}(\text{Rumination} * \text{Negative Emotion})$$

$$\text{Level 2 (individual). } \beta_{0j} = \beta_{00} + \beta_{01}(\text{Group})_i + u_{0i}$$

$$\beta_{1j} = \beta_{10} + \beta_{11}(\text{Group})_i$$

$$\beta_{2j} = \beta_{20} + \beta_{21}(\text{Group})_i$$

$$\beta_{3j} = \beta_{30} + \beta_{31}(\text{Group})_i$$

$$\text{Reduced Equation: } \eta_{ij}(\text{Nightmares}) = \log(\mu_{ij}) = \beta_{00} + \beta_{10}(\text{Rumination}_{\text{DayIntensity}})_{ij} + \beta_{20}(\text{Negative Emotion}_{\text{DayIntensity}})_{ij} + \beta_{30}(\text{Rumination} * \text{Negative Emotion})_{ij} + \beta_{01}(\text{Group})_i + \beta_{11}(\text{Group} * \text{Rumination})_i + \beta_{21}(\text{Group} * \text{Negative Emotion})_i + \beta_{31}(\text{BPD} * \text{Rumination} * \text{Negative Emotion})_i + u_{0i}$$

As can be seen in the reduced model equation, nightly number of nightmares reported (η_{ij}) each day (j) for each person (i) was log transformed to fit the Poisson distribution (μ_{ijk}), and the term is identified by an intercept (β_{00}) with individual-level random error (u_{0i}). Also included are the main effects for BPD diagnosis (β_{01}), day-of

rumination (β_{10}), and day-of negative emotion (β_{20}); effects are also present for the same and cross level two-way interactions and the cross-level three-way interaction (β_{31}).

Finally, we used hierarchical linear models to examine the effect of number of nightly nightmares on the subsequent day's levels of negative emotion and rumination (Hypothesis 4). Throughout analyses, the following variables were used as covariates because of potential influence on nightmare experiences: PTSD diagnosis, MDD diagnosis, suicidality (as indexed by BSS scores), age, sex, community versus student status, and if the participant was taking psychotropic medication. The analysis of daily nightmares also included previous night's number of nightmares, same-night sleep quality and sleep total to ensure that these factors did not account for any detected effects. All analyses were conducted with SPSS Version 20.

RESULTS

Preliminary Analyses

During the 2-week monitoring period, a total of 219 nightmares were reported across the sample, with a range of 0 to 24, averaging a total of .33 per person per day. The average number of total nightmares for the BPD group was 5.44 ($SD = 7.12$), and the average for the non-BPD group was 4.26 ($SD = 5.26$), and both groups are relatively higher than the general population average of one nightmare per year (Hartmann, 1987). The average hours of sleep per night for the BPD group was 6.98 ($SD = 1.73$) while the non-BPD group was 7.33 ($SD = 1.41$). Regarding average sleep quality, the BPD group had an average of 5.63 ($SD = 1.69$) while the non-BPD group had 6.18 ($SD = 1.17$). The two groups were compared on their number of nightmares reported during monitoring using a Poisson regression analysis, given the count nature of the number of nightmares reported. The analysis indicated that, consistent with Hypothesis 1, the BPD group reported significantly more nightmares during monitoring ($B = .261$, $SE = .074$, $wald = 12.34$, $p < .001$, $RR = 1.30$). Using standard regression modeling, BPD was also a significant predictor of less average sleep hours over monitoring ($B = -.25$, $SE = .12$, $t = 4.40$, $p < .05$). However, those with BPD did not report worse average sleep quality ($B = -.16$, $SE = .36$, $t = .56$, $p = .45$). Relevant covariates were also added to the models, including age, sex, PTSD diagnosis, MDD diagnosis, level of suicidality, community versus student participant, and currently taking medications, to determine if these factors were driving the group differences. The results of these analyses remained unchanged after including these covariates.

Nightmares, Borderline Personality Disorder, and Baseline Trait Rumination

For the generalized linear regression model examining total nightmares reported across monitoring with BPD and trait rumination as predictors (displayed in Table 1), there was a significant intercept ($B = 2.3$, $SE = .59$, $wald = 14.73$, $p < .001$). When entered simultaneously, there was a significant main effect for BPD ($B = .23$, $SE = .08$, $wald = 8.34$, $p < .01$, $RR = 1.25$), but not for trait rumination

Table 1. Interaction Between BPD and Trait Rumination Predicting Nightmare Frequency

Variable	<i>B</i>	<i>SE</i>	<i>Wald</i>	<i>p</i>	<i>RR</i>
Main effects					
Intercept	2.25	.59	14.73	<.001	—
BPD	.225	.078	8.34	.004	1.25
Baseline rumination	-.010	.014	.038	.471	—
Two-way interaction					
BPD × rumination	.043	.015	8.26	.004	1.04

Note. Not shown but included in the model as covariates were age, sex, depressive disorder diagnosis, posttraumatic stress disorder diagnosis, BSS score, that night's sleep quality, and that night's amount of sleep.

($B = -.01$, $SE = .14$, $wald = -.04$, $p = .47$). As expected (Hypothesis 2), the BPD by trait rumination interaction was a significant prospective predictor of number of nightmares reported during monitoring ($B = .043$, $SE = .015$, $wald = 8.26$, $p < .01$, $RR = 1.04$). Figure 2 displays the graphed interaction, which demonstrated that the results were consistent with our hypothesis that those with a BPD diagnosis and high trait rumination at baseline would report the highest number of nightmares experienced during monitoring. Importantly, this interaction maintained significance when controlling for sex ($B = .18$, $SE = .19$, $wald = .92$, $p = .34$), age ($B = -.01$, $SE = .01$, $wald = .511$, $p = .48$), PTSD ($B = .98$, $SE = .28$, $wald = 12.48$, $p < .001$, $RR = 2.66$), MDD ($B = .54$, $SE = .21$, $wald = 6.75$, $p < .01$, $RR = 1.72$), BSS score ($B = .009$, $SE = .14$, $wald = .45$, $p = .50$), taking psychotropic medication ($B = -.81$, $SE = .20$, $wald = 15.98$, $p < .001$, $RR = .44$), and community versus student status ($B = -.13$, $SE = .19$, $wald = .48$, $p = .49$).

Daily Intensity of Emotional Cascades and Subsequent Nightmares

The purpose of this model was to examine if emotional cascades, defined as the interaction between day-of negative emotion intensity and day-of rumination intensity for those with BPD, would predict subsequent nightly nightmares reported. The results of this model are displayed in Table 2. For this hierarchical

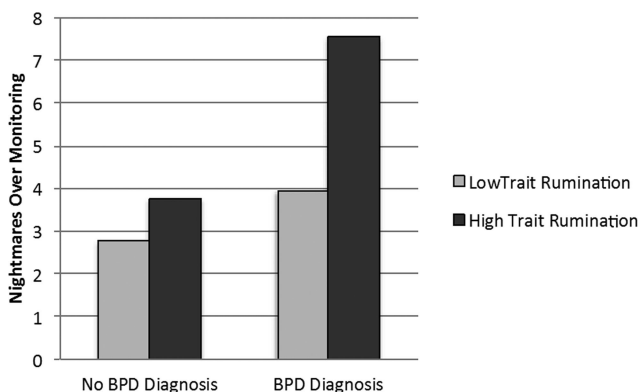


Figure 2. Two-way interaction of BPD diagnosis and baseline trait rumination predicting number of nightmares reported during the 2-week monitoring period.

Table 2. Three-Way Interaction for Emotional Cascades Predicting Subsequent Nightmares

Variable	β	<i>SE</i>	<i>Wald</i>	<i>p</i>	<i>RR</i>
Main effects					
Day	.063	.009	7.34	<.001	1.07
BPD	.110	.052	7.34	.036	1.12
Day of rumination	.046	.006	7.59	<.001	1.05
Day of NE	-.002	.016	.148	.882	—
Two-way interactions					
BPD \times DoR	-.014	.007	1.99	.047	.98
BPD \times DoNE	.006	.0017	3.98	<.001	1.01
DPR \times DoNE	-.002	.001	2.04	.041	.99
Three-way interaction					
BPD \times DoR \times DoNE	.003	.001	2.25	.025	1.01

Note. Not shown but included in the model as covariates were age, sex, depressive disorder diagnosis, posttraumatic stress disorder diagnosis, BSS score, that night's sleep quality, that night's amount of sleep, and previous night's number of nightmares; DoR = Day-of rumination intensity; DoNE = Day-of negative emotion intensity.

generalized linear model, there was a significant random intercept ($\beta = .262, p < .001$). Regarding the main effects, in this model both BPD ($\beta = .11, SE = .05, wald = 7.34, p < .05, RR = 1.12$) and day-of rumination ($\beta = .05, SE = .01, wald = 7.59, p < .001, RR = 1.05$) were significant predictors of nightly nightmares; day-of negative emotion intensity was not. All three of the two-way interactions were significant including BPD with day-of rumination ($\beta = -.014, SE = .007, wald = 1.99, p < .05, RR = .98$), BPD with day-of negative emotion ($\beta = .006, SE = .002, wald = 3.98, p < .001, RR = 1.01$), and day-of rumination with day-of negative emotion ($\beta = -.002, SE = .001, wald = 2.04, p < .05, RR = .99$).

Consistent with our original hypothesis, the three-way interaction for day-of rumination, day-of negative emotion, and BPD diagnosis ($\beta = .003, SE = .001, wald = 2.25, p < .05, RR = 1.01$) significantly predicted nightly nightmares (Hypothesis 3). The results of this interaction are displayed in Figure 3, which demonstrated that the most nightmares were reported when all three variables were high. Importantly, the interaction was significant even with the covariates in

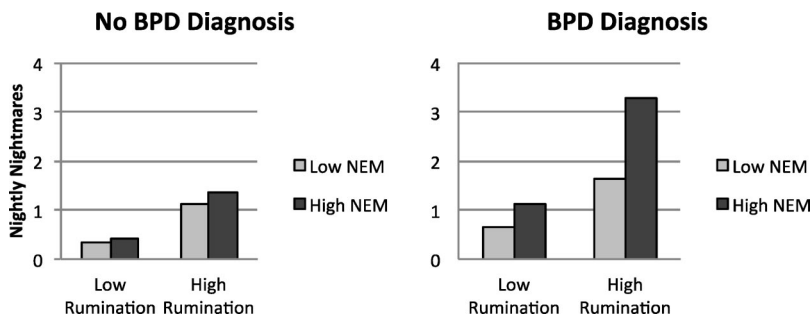


Figure 3. Three-way interaction for daily level of Emotional Cascades, defined as the interaction between day of intensity of rumination and day of intensity of negative emotion, interacting with BPD diagnosis to prospectively predict subsequent number of nightmares experience that night. *Note:* NEM = day-of intensity of negative emotion; Rumination = day-of intensity of rumination, BPD = borderline personality disorder.

this model, which included a number of significant predictors of nightmares, including: more sleep predicted more nightmares ($\beta = .78, SE = .10, wald = 7.67, p < .01, RR = 2.18$), MDD diagnosis ($\beta = .33, SE = .10, wald = 3.17, p = .002, RR = 1.39$), PTSD diagnosis ($B = .60, SE = .14, wald = 4.43, p < .001, RR = 1.82$), younger age ($\beta = -.03, SE = .001, wald = -5.95, p < .001, RR = .97$), and number of nightmares experienced the previous night ($\beta = .32, SE = .03, wald = 10.67, p < .001, RR = 1.38$). On the other hand, BSS ($\beta = .01, SE = .01, wald = 1.06, p = .29$), and sleep quality ($\beta = .02, SE = .03, wald = .55, p = .59$) did not predict number of nightmares with all other variables entered into the model.

Nightmares and Subsequent Daily Functioning

In these mixed models we examined the effects that number of nightly nightmares had on subsequent daily functioning, as indexed by daily level of negative emotion and rumination. The first model indicated that number of nightmares was a significant predictor of next day intensity of negative emotion ($\beta = .937, SE = .177, t = 5.287, p < .001$). Furthermore, number of nightmares interacted with BPD diagnosis (Hypothesis 4) to predict even higher levels of next day negative emotion intensity for those with BPD ($\beta = .902, SE = .340, t = 2.652, p = .008$). These findings held after controlling for number of hours slept ($\beta = -.001, SE = .055, t = -.001, p = .99$) and sleep quality ($\beta = -.018, SE = .182, t = -.098, p = .922$). Regarding daily rumination intensity, again number of previous nightly nightmares predicted increased subsequently rumination intensity ($\beta = 2.869, SE = .445, t = 6.443, p < .001$). This finding held after controlling for number of hours slept ($\beta = -.207, SE = .456, t = -.454, p = .65$) and sleep quality ($\beta = .039, SE = .139, t = .282, p = .778$). However, there was not a significant interaction between number of nightly nightmares and BPD diagnosis indicating higher levels of rumination in those with BPD ($\beta = .936, SE = .877, t = 1.068, p = .286$), suggesting that the effects of nightmares may impact subsequent affective functioning in those with BPD more than subsequent cognitive functioning.

DISCUSSION

Previous research has indicated that those with BPD may experience more nightmares than healthy controls, yet no hypotheses have been proposed for why this may occur. The ECM may provide some insight into this phenomenon by extending the possible experience of emotional cascades to sleep. We presented a potential model through which emotional cascades may contribute to true nightmare experiences, as well as potential nightmare-like experiences. Using experience sampling data from a sample of dysregulated participants, many of which met diagnostic criteria for BPD, we tested hypotheses derived from the ECM model. First, we replicated the finding that those with BPD reported more subjective nightmares than those without BPD diagnoses, and both were much more frequent than the general population average of one nightmare per year (Hartman, 1987). Second, BPD interacted with baseline trait rumination to prospectively predict number of nightmares reported over 2 weeks, even when controlling for crucial

covariates. Third, a three-way interaction for emotional cascades and BPD predicted subsequent nightly nightmares, again after controlling for important covariates. Finally, number of nightmares predicted elevated negative emotion and rumination the next day, and BPD interacted with nightmares to predict higher levels of subsequent negative emotion, but not rumination.

The findings of this study are consistent with a general model of nightmares proposed by [Levin and Nielsen \(2007\)](#), which suggests that nightmares are the result of daily variations in emotional pressure and high emotional reactivity to daily events. Importantly, this general theory of nightmares dovetails nicely with the extension of the ECM to nightmares in BPD. In the case of those with BPD, the emotional pressure and reactivity experienced in nightmares may potentially be caused by, or at least aggravated by emotional cascades. Although the nightmares reported in this study were not qualitatively analyzed, if these nightmares are related to daytime emotional cascades then such future analyses should indicate that many of these nightmares are related to daily and future concerns, rather than bizarre and unrelated to their daily lives.

One important finding in the current study was that the results held even after controlling for comorbid PTSD. Given that nightmares are a major factor in this disorder, which is also commonly comorbid with BPD, the findings of the current study suggest that nightmares in BPD may be different in some ways that those experience in PTSD. Following the ECM, in contrast to nightmares about reexperiencing trauma (although that may certainly be relevant in some BPD cases), nightmares in BPD may be more about upsetting current events or future problems, such as identity issues and/or anticipated abandonment, which are typical concerns for many individuals with BPD. Also important to note was that level of suicidality was controlled for in this study, which has been linked to nightmares ([Bernert et al., 2005](#)). However, we did not find a significant association between suicidal ideation as measured by the BSS and nightmares. This finding may have been due to a floor effect in this study, as the average BSS score in this study was fairly low.

Limitations

Despite the novel findings of this study, there are some limitations worth noting. The primary limitation with this study is that nightmares were self-reported. We did not assess the duration nor the content of the nightmares, subjective distress arising from the nightmares, or ability to recall the content of the nightmares, which leaves the report of nightmares open to the subjective interpretation of the participants. Some of these nightmares may actually have been bad dreams, hypnagogic, and/or hypnopompic hallucinations, which are similar but distinct phenomena from nightmares ([Zadra & Donderi, 2000](#)). Alternatively, one nightmare broken up through the night may have been reported as multiple dreams, and unpleasant dreams may have been considered a nightmare. Similarly, some of these nightmares may have actually been “waking nightmares,” which were part of the model presented but we were unable to test this component with these data. In this case participants may have actually awoken or been in the lower stages of sleep and experience a wakeful emotional cascade, rather than a nightmare. Future studies

should tease apart these potentially different experiences in BPD with both qualitative analyses of dreams and through laboratory sleep studies. Another limitation was that other sleep disturbances, such as insomnia or obstructive sleep apnea, were not assessed in this study, and this may be important because experience of other sleep problems may influence the experience of nightmares. Other limitations with the current study include a somewhat small sample size, all participants exhibited behavioral dysregulation to some extent and results may not generalize to those without, and that the results can only demonstrate temporal precedence of emotional cascades in nightmares and not causality. Furthermore, due to the complexity of our model and issues with assessing nightmares in general, we were unable to test our full model.

Clinical Implications

The experience of nightmares in those with BPD is an important phenomenon for both clinicians and researchers to understand, as many of the patients have difficult waking lives and nightmares may make it difficult for them to find peace even in sleep. If the current model is replicated in future studies, then clinicians may have a helpful explanation for participants plagued with nightmares. Rather than being mysterious and pernicious experiences, patients can learn to recognize nightmares as a sleeping extension of their waking psychopathology. Such explanations could reduce nightmare anxiety and overanalysis of the nightmares. Furthermore, if nightmares are extensions of emotional cascades, then treatment that reduces emotional cascades may also reduce nightmares.

In those with BPD, nightmare imagery rescripting (Long & Quevillon, 2009) may be helpful, but that may only be in cases where people experience a great deal of nightmare anxiety. However, if nightmares are sleeping extensions of emotional cascades, then addressing the rumination and negative emotion experienced during the day may be important for ameliorating nightmares experienced that night. Qualitatively assessing the content of nightmares reported may help clinicians identify which approach should be taken, with imagery rescripting being implicated for trauma-related dreams or situations where nightmares are anxiously anticipated, while the alternate approach may be better for other nightmares. There may also be a role for medication in the treatment of nightmares in BPD, as recent work has shown that prazosin, an alpha-1-adrenergic antagonist and antihypertensive medication, has been found to reduce nightmares (Raskind et al., 2003). Of note, the positive effects of prazosin on nightmares is consistent with the model presented in this study—by reducing blood pressure, overall arousal may be decreased, including cognitive arousal that may potentiate nightmares.

Conclusion

The findings of this study further support the notion that those with BPD have elevated problems with nightmares. The experience of these nightmares may also be linked to daily experience of emotional cascades, indicating that upsetting daily events may follow these patients even into sleep. More research is needed,

however, to examine both nightmares and nightmare-like experiences in BPD, both of which may be influenced by emotional cascades. A better understanding of how daily cognitive and emotional factors may influence nighttime experiences may help clinicians improve their treatment for nightmares in BPD, and reducing nightmares may improve the daily experiences of those patients and help them on their path to recovery.

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