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A theoretical perspective on the role of sleep in borderline personality disorder: From causative factor to treatment target

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ABSTRACT

Sleep plays a crucial role in emotion regulation. Disturbed sleep is therefore increasingly seen as a potential causal factor for the development and maintenance of affective psychiatric disorders. This may hold especially for borderline personality disorder (BPD), a core emotion dysregulation disorder. Although BPD is strongly associated with sleep disturbances such as insomnia, nightmares and circadian dysrhythmia, research into the role of sleep in BPD remains sparse. In this narrative review, we outline a putative vicious cycle of reciprocal exacerbation of disturbed sleep and emotion dysregulation in BPD, that sheds light on BPD pathophysiology and opens up new avenues for sleep-based treatments. We discuss emotional dysregulation as the base of BPD as well as the observed sleep disturbances in BPD. Based on existing theories of sleep's role in emotion regulation and memory, we then propose several behavioral and neurobiological pathways by which inherent sleep disturbances in BPD may hamper adaptive overnight emotional processing. This likely results in sustained emotional states and associated sleep-disruptive behavior, which in turn negatively impact sleep. We end by proposing a sleep-based research agenda for BPD to further detail the causative role of disturbed sleep in BPD and test the effectiveness of novel sleep-based treatment strategies.

Abbreviations

ACC	anterior cingulate cortex
ADHD	attention deficit hyperactivity disorder
BD	bipolar disorder
BDLEs	evening blue-depleted light environments
BPD	borderline personality disorder
CBT-I	cognitive behavioral therapy for insomnia
DBT	dialectical behavior therapy
EEG	electroencephalogram
HPA	hypothalamic-pituitary-adrenal
IRT	imagery rehearsal therapy
LC	locus coeruleus
NREM	non-rapid eye movement
NSSI	nonsuicidal self-injury
PFC	prefrontal cortex
PSG	polysomnography
PTSD	post-traumatic stress disorder
REM	rapid eye movement
SWS	slow wave sleep

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1. Introduction

Borderline personality disorder (BPD) is a psychiatric disorder characterized by an enduring and pervasive pattern of severe instability in emotions, interpersonal relationships, and self-image, as well as marked impulsivity and self-harming and suicidal behavior [1]. Prevalence rates of BPD range from 0.7 to 2.7 % in Western community samples [2] to 11–35 % in clinical samples [3]. BPD poses both a personal and societal burden as it is associated with functional impairment, high suicide risk, high comorbidity rates, and extensive use of (mental) health care [2].

Currently, standard psychotherapeutic treatment options for BPD, albeit often effective, are generally lengthy, expensive and not always accessible for patients [3,4]. Medication effects in BPD are modest and limited to individual symptoms [5,6]. Novel (complementary) treatment strategies for BPD are therefore needed. At the core of BPD symptomatology lays emotion dysregulation [7,8], and a growing neuroscientific literature highlights the essential role of sleep in emotion regulation and emotional memory [9–11]. Based on these insights, disturbed sleep is increasingly recognized as a causal factor (as opposed to a mere symptom) in several affective psychiatric disorders [11–14]. In theory, this could be particularly true for clinical manifestations of emotion dysregulation, like BPD. Importantly, BPD patients often experience severe sleep disturbances that in some studies have been shown to aggravate BPD symptoms such as emotion dysregulation, nonsuicidal self-injury (NSSI), and suicidal behavior [15–17]. Yet, disturbed sleep and sleep treatment in BPD receive relatively little interest in both research and clinical settings [18,19], and sleep's mechanistic role in BPD remains unknown. In this narrative review, we discuss the possible causative contribution of sleep disturbances to BPD and explore sleep as a new treatment target in BPD patients. First, we discuss why BPD is considered a disorder of emotion regulation. Next, we review what sleep disturbances are prevalent in BPD patients. Based on a growing literature in healthy and other psychiatric populations highlighting the fundamental role of sleep in emotion regulation, we then propose several possible pathways that explore a causative role of disturbed sleep in the development and maintenance of BPD. Finally, we discuss new potential treatment targets for BPD focusing on sleep, including the experimental avenues to investigate them.

2. BPD as a disorder of emotion regulation

In this review, we define emotion dysregulation both as heightened emotional sensitivity and intensity, and difficulties in adaptively regulating emotional responses [20]. Emotion dysregulation is at the core of BPD. It manifests itself as the emotional symptoms of BPD (affective instability, anger attacks, fear of abandonment), drives the severe behavioral symptoms (impulsivity, NSSI and suicidal behavior), and fosters the cognitive (self-image disturbance, stress-related dissociation and paranoid ideation) and relational (fear of abandonment, extremes of idealization and devaluation) symptoms of the disorder [7,8]. Typical harmful behaviors, such as substance abuse, overeating, NSSI, and suicidal behavior are often maladaptive attempts to suppress emotional distress [8]. Although these strategies often have short-term benefits, they ultimately undermine long-term emotional stability and enhance other BPD symptoms such as unstable relationships, the fear of abandonment, and feelings of emptiness [7,8,20]. The dimensional approach for diagnosing BPD according to the alternative DSM-5 model for personality disorders additionally supports emotion dysregulation as the main dimension of BPD, differentiating this otherwise heterogeneous diagnostic construct from other personality disorders and healthy behavior [21].

3. Sleep disturbances in BPD

Both subjective and objective sleep disturbances are highly prevalent in BPD [16,17]. A meta-analysis of sleep profiles in BPD showed that BPD patients report longer sleep onset latency and lower sleep quality than healthy controls [15]. Some studies additionally found lower sleep quality in BPD patients than in patients with attention deficit hyperactivity disorder (ADHD) [22], depression, or anxiety disorders [23]. BPD patients report high insomnia severity [22–24]. Up to 63 % of BPD patients experience chronic insomnia symptoms, and associations with insomnia are as strong as in disorders that are more commonly linked to insomnia or, unlike BPD, have insomnia-related diagnostic criteria, such as generalized anxiety disorder, depression, and post-traumatic stress disorder (PTSD) [25]. BPD patients additionally report lower sleep efficiency, spent more time in bed, use more sleep medication, and experience more daytime sleepiness [22,24]. BPD symptom severity is associated with lower sleep quality in both community [26] and clinical samples [27].

Objectively measured sleep continuity is also disturbed in BPD, with patients having longer sleep onset latency and wakefulness after sleep onset, more awakenings, shorter total sleep time, and lower sleep efficiency than healthy controls [15,28]. Young BPD patients in contrast spend more time in bed and have longer total sleep time, but this was measured only with actigraphy [23]. There is no meta-analytic evidence for sleep continuity differences between BPD patients and clinical controls (e.g. depression or other personality disorders) [15]. BPD is also linked to polysomnography (PSG)-based sleep architecture alterations. Most consistent evidence indicates BPD patients have shorter rapid eye movement (REM) sleep latency, higher REM density, and less slow wave sleep (SWS) than healthy controls [15,28]. Again, no differences were found compared to clinical controls, but REM latency and -density differences with healthy controls were stable regardless of having comorbid, a history of, or no depression, indicating that altered sleep architecture in BPD is not explained by comorbid depression [15]. Increased REM density and shortened REM latency are thought to reflect a compensatory REM sleep rebound mechanism due to chronically fragmented REM sleep (REM sleep frequently interrupted by arousals, wakefulness or NREM sleep) [29]. BPD patients (without comorbid depression and regardless of having comorbid PTSD) also showed a higher number of arousals during the night and more fragmented REM sleep than healthy controls [30]. Some studies found higher percentages of REM [31] and N1 [32], and lower percentages of N3 sleep [33,34] in BPD patients. Only two studies assessed electroencephalogram (EEG) spectral power and tentatively indicate increased delta and theta power during non-REM (NREM) sleep in patients with BPD, although small sample sizes limit generalizability [33,34].

Circadian rhythm disturbances are also related to BPD [35]. BPD patients report a later chronotype and more varying bed and rise times than healthy and clinical controls [23,24]. Actigraphy-based studies similarly found delayed sleep phase and more varying total sleep time and bed and rise times [23,36]. Patients with BPD also have more desynchronized diurnal rhythms than patients with bipolar disorder (BD) and healthy controls, reflected by a longer time lag between physical activity and sleep onset [37]. These findings indicate circadian rhythm in BPD is phase-delayed and desynchronized, with misaligned rest-activity patterns.

Finally, BPD patients often experience altered dream activity, which impacts sleep continuity and quality. Various studies linked BPD to more negatively toned dreams, nightmares, and dream anxiety, independent of comorbid PTSD or depression [15,38,39]. Prevalence rates of nightmares in BPD specifically are estimated to be around 50 % [40]. Also in community samples, BPD symptom severity is associated with higher nightmare frequency and distress [41–43].

In sum, sleep in BPD is characterized by shortened, fragmented, and lower quality sleep, with alterations in both REM sleep and SWS, circadian (phase delayed and desynchronized) dysrhythmia and prevalent nightmares. These disturbances are often similar to but not accounted for by possible comorbid affective psychiatric disorders.

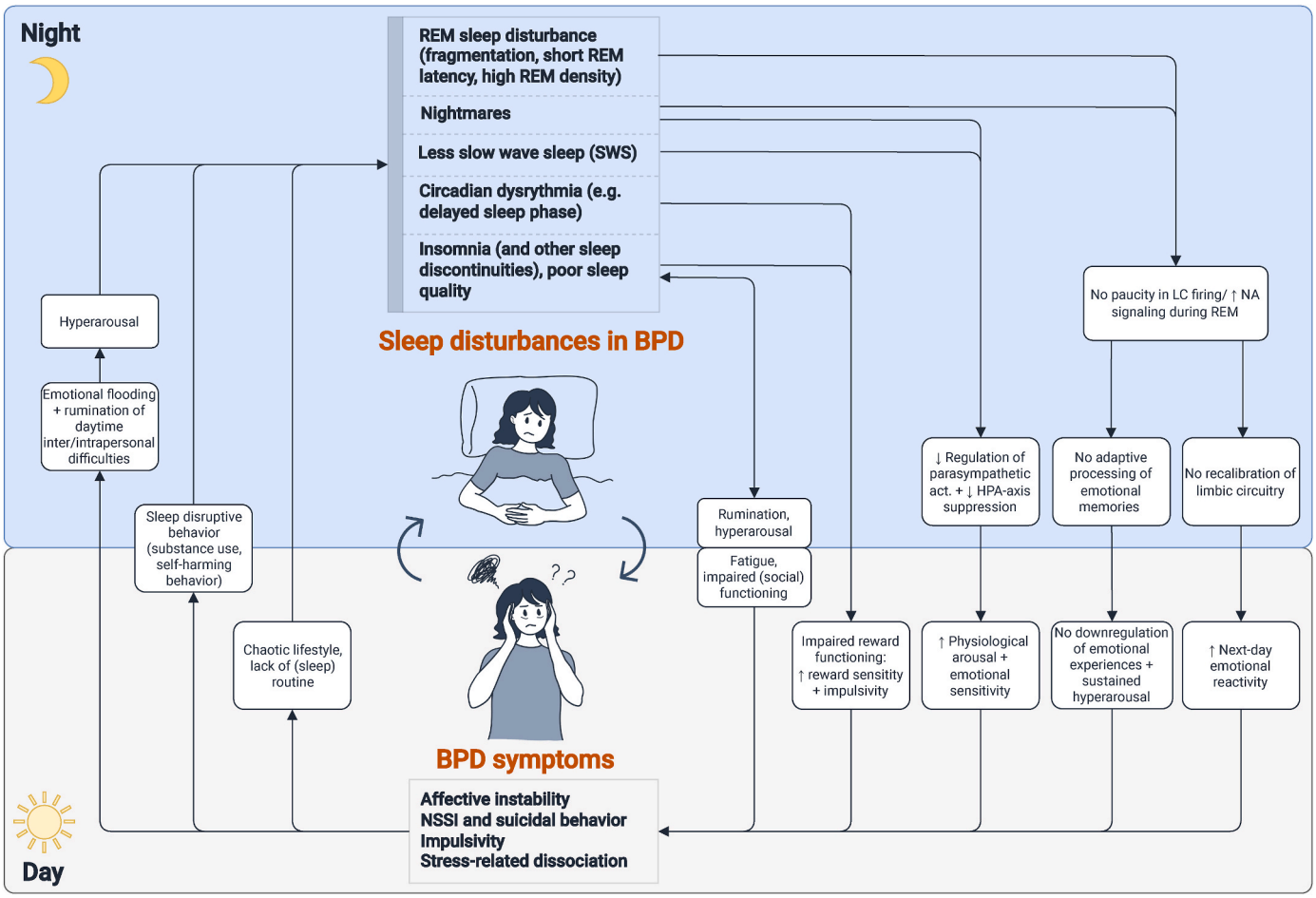

Fig. 1. Hypothesized reciprocal relation between sleep disturbances and borderline personality disorder (BPD) symptoms. HPA (hypothalamic-pituitary-adrenal); LC (locus coeruleus); NA (noradrenergic); NSSI (nonsuicidal self-injury); REM (rapid eye movement).

4. Reciprocal relation between BPD and disturbed sleep

Sleep plays a pivotal role in emotional memory and -processing. Disturbed sleep is therefore increasingly seen as a causative factor for psychiatric disorders, rather than a mere symptom or epiphenomenon [13,44]. This may hold especially for BPD, given the centrality of emotion dysregulation in the disorder. Below, we discuss a number of hypothesized neurobiological and behavioral pathways to understand emotion dysregulation in BPD that putatively link the observed sleep disturbances in BPD to existing theories on the function of sleep in emotion regulation and memory. For each proposed pathway, due to the relative lack of research in BPD patients, we first outline sleep's role in emotion regulation drawing from studies in non-clinical or other psychiatric populations, before we explore how these mechanisms may relate to BPD symptomatology, incorporating all available evidence in BPD patients. The following pathways will be explored: Noradrenergic overdrive during (fragmented) REM sleep, lack of SWS, circadian dysrhythmia, insomnia and other sleep continuity disturbances, nightmares, and BPD symptoms impacting sleep. See Fig. 1 for a schematic overview of the pathways representing a vicious cycle of reciprocal exacerbation of disturbed sleep and BPD symptoms.

4.1. Noradrenergic overdrive during (fragmented) REM sleep

4.1.1. The role of REM sleep in emotional memory

Healthy emotion regulation depends critically on healthy sleep and specifically on sound REM sleep. One of sleep's primary functions is memory consolidation [10,45], during which initially labile memory traces, represented by hippocampal-cortical neural patterns, get reorganized as cortico-cortical associations, thereby stabilizing the memory and integrating it into pre-existing semantic memory networks [46]. The reorganization of emotional memory traces, according to the prevalent *Sleep to remember and sleep to forget* hypothesis, serves to preserve the factual core of the emotional memory, while depotentiating and ultimately ameliorating the affective charge and autonomic arousal connected to the memory [9,47]. Animal work indicates that this process depends critically on intrinsically low levels of the neurotransmitter noradrenaline during REM sleep due to silencing of the locus coeruleus (LC) [48]. Together with high cholinergic activity during REM sleep, this permits theta oscillation coupling between the amygdala, hippocampus and medial PFC [49]. Specifically, anti-phase theta coupling between the hippocampus and amygdala, with hippocampal input arriving time-locked to theta troughs, is thought to result in the depotentiation of synapses in the central nucleus of the amygdala, which regulates autonomic and stress responses [50–52]. This is thought to facilitate the preferential consolidation of emotional memory traces, albeit disconnected from their limbic representation and associated autonomic arousal [53]. In contrast, protracted noradrenergic activity, associated with fragmented REM sleep [11,53], is thought to result in in-phase (during theta peaks) instead of anti-phase theta synchrony between the hippocampus and amygdala, potentiating amygdalar synapses and preventing the adaptive regulation of autonomic and stress responses linked to the memory trace [51,52]. This may result in sustained or even increased salience of emotional memories [53].

In humans, experimental evidence for this model is still sparse but growing. Using a shame-inducing experimental paradigm in people with

varying degrees of insomnia, Wassing and colleagues showed that sound, undisturbed REM sleep is associated with reduced subjective ratings of and amygdala reactivity to shameful stimuli [54,55]. This indicates adaptive overnight alleviation of emotional distress. Fragmented REM sleep, however, was shown to negate this beneficial effect of sleep, as reflected by sustained amygdala reactivity overnight and in cases of most fragmented REM sleep, even worsening of the subjective experience of shame. Using a similar shame-inducing paradigm, REM fragmentation was additionally shown to be associated with less overnight reduction in autonomic arousal, especially in people with high shame-proneness [56]. A study in PTSD patients, trauma-exposed individuals, and controls showed that, across all groups, high REM fragmentation was associated with increased autonomic arousal, which predicted poorer overnight emotional memory consolidation [57]. REM sleep duration and specifically high theta power during REM sleep seem to facilitate overnight distress regulation as they were associated with increased emotional memory consolidation in a non-clinical population [58], less intrusive re-experiencing symptoms after watching traumatic videos in people with insomnia [59], and resilience to developing PTSD in people having experienced a traumatic event [60]. Reversely, low REM frontal theta power was associated with high levels of pre-sleep arousal and emotional distress in healthy people with bruxism [61].

Although current evidence is mostly based on small samples, these findings propagate a possible vicious cycle of fragmented REM sleep failing to host an overnight downregulation of emotional distress, leading to sustained hyperarousal and increased emotional reactivity by day. This may in turn negatively impact on REM-consistency during subsequent sleep through protracted LC-firing [11]. Chronically fragmented REM sleep, in some cases even emanating from early life adversity [62], may then lead to accumulation of long-lasting emotional distress and chronic hyperarousal [63]. This is illustrated in insomnia patients with high levels of REM fragmentation that showed high limbic activation in reaction to both shameful memories from the past as well as novel shameful experiences [64]. Ultimately, this may increase the risk for affective psychiatric disorders like depression and PTSD [11]. This notion is supported by longitudinal data showing that REM fragmentation predicts future depressive symptoms in patients with short-term insomnia [65], as well as the onset of PTSD after experiencing psychological trauma [66].

4.1.2. The role of REM sleep in limbic recalibration

REM sleep is also thought to directly facilitate a recalibration of limbic circuitry, shaping next-day reactivity to new emotional experiences. The unique paucity of LC-firing during solid REM sleep results in strongly reduced noradrenergic neurotransmission throughout limbic and other brain regions. This is thought to regulate sensitivity of noradrenergic receptors [67] and set the stage for balanced (low tonic, strong phasic) next-day LC-firing [68]. In addition, balanced LC-firing facilitates top-down prefrontal modulation of limbic circuitry [9,69]. Reversely, sustained LC-firing and high noradrenergic activity resulting from REM fragmentation likely hinder the recalibration of limbic circuitry which may lead to impaired adequate next-day emotional salience detection, discrimination and responsivity [9]. This is evidenced by studies in healthy subjects showing that REM sleep facilitates adaptive and selective reactivity to salient stimuli, such as negative and positive faces [70], while REM sleep deprivation leads to less differentiation in fear response between threatening and non-threatening stimuli [71].

4.1.3. Noradrenergic overdrive during (fragmented) REM sleep as possible mechanism in BPD

BPD sleep is characterized by REM sleep disturbances, such as REM fragmentation, as well as increased REM density and shortened REM latency. In addition, several common sleep disturbances in BPD, such as insomnia and nightmares, are characterized by fragmented REM sleep [29,72,73]. Although direct evidence is currently lacking, these REM sleep disturbances in BPD, and especially fragmented REM sleep associated with sustained LC-firing, heightened noradrenergic signaling and likely desynchronized theta coupling, may theoretically prevent the downregulation of autonomic and stress responses linked to emotionally distressing memories in BPD. This leaves the patient in a state of continued hyperarousal, unable to disengage from the emotionally charged experiences of the previous day, potentially underlying typical BPD symptoms like affective instability and unremitting interpersonal problems and increasing the risk for maladaptive emotion regulatory strategies such as NSSI and substance abuse. Sustained emotional distress and hyperarousal may in turn negatively impact power and synchronization of theta oscillations [74] and REM-consistency during subsequent sleep through protracted LC-firing [11], closing the vicious cycle. Noradrenergic overdrive during (fragmented) REM sleep may additionally hamper adequate recalibration and prefrontal top-down modulation of limbic circuitry [9,69]. As a result, next-day reactivity to negative experiences is increased and the threshold for reactivity to even non-threatening stimuli is lowered, potentially aggravating the experience of overwhelming and unmanageable emotions throughout the day and increasing the risk of interpersonal conflicts and dysfunctional emotion regulation strategies seen in BPD.

As said, despite the strong theoretical rationale of these pathways, no studies so far investigated nighttime noradrenergic overdrive during (fragmented) REM sleep in BPD. BPD is linked to daytime autonomic dysfunction evidenced as increased, prolonged and more stable EEG-vigilance, indicative of LC-driven noradrenergic hyperreactivity [75], and lower resting state vagal tone [76,77]. Neuroimaging results in BPD also showed stronger LC-anterior cingulate cortex (ACC) connectivity, which may indicate hampered prefrontal control and higher tonic LC-firing [78]. In addition, brain activation patterns in BPD are characterized by amygdala hyperreactivity and impaired amygdala habituation to emotionally negative stimuli, associated with affective instability, impulsivity, and aggression [79–81], impaired improvement in BPD symptoms over time [82,83], as well as a deficient prefrontal control over limbic-driven emotional reactivity and impulsivity [84,85]. BPD patients moreover show a more negative bias towards and increased autonomic and limbic reactivity to neutral stimuli, which is associated with the tendency to anticipate interpersonal threat and rejection and may lead to avoidance, increased fear of abandonment, and anger attacks [86–88]. Additional support for a causal role of noradrenergic overdrive on BPD symptoms, comes from a pharmacological study showing reduced aversive inner tension, dissociation, NSSI and suicidal ideation after blocking noradrenergic activity with clonidine, an alpha-2-adrenergic-receptor agonist [89]. None of the above-mentioned studies in BPD however, investigated a direct link with sleep. A recent BPD treatment study, measuring sleep but not noradrenergic activity, showed that shorter REM sleep duration and lower REM percentage in BPD patients were associated with higher BPD symptom severity [90]. In addition, a treatment study in BPD patients with co-morbid PTSD showed that longer REM sleep duration pre-treatment predicted greater improvement after trauma treatment [91]. In paragraphs 5 and 6, we suggest several routes to investigate the role of REM sleep disturbances and associated noradrenergic overdrive in BPD and further elucidate this pathway.

4.2. Lack of SWS

4.2.1. The role of SWS in emotion regulation

Next to REM sleep, NREM slow-wave sleep (SWS) is additionally implicated in the consolidation of emotional memories [92] and most likely, NREM and REM sleep act interactively and complementarily in the processing and long-term storage of emotional memories [93]. Experimentally, more slow wave activity and associated SWS is shown to predict greater overnight anxiety resolution in a non-clinical population [94]. Mechanistically, SWS may foster emotion regulation through its restorative effect on parasympathetic vagal activity) [94,95],

and intrinsically low hypothalamic-pituitary-adrenal (HPA)-axis activity during SWS [96,97]. The HPA-axis follows a 24-h pattern, with circadian-dependent cortisol secretion. During the first part of the night, dominated by SWS, a very low level of cortisol is maintained mainly through mineralocorticoid receptor-mediated negative feedback from the hippocampus at the level of the hypothalamus and pituitary [98]. When SWS is disturbed, this inhibitory control of the HPA-axis is impaired [99]. Although the exact ways in which disturbed SWS impacts emotion regulation remain unclear, the resulting high levels of cortisol together with autonomic hyperactivity may hamper emotion regulation by impairing prefrontal control over limbic neurocircuitry [100,94] and by interfering with adaptive SWS-dependent memory consolidation [101]. High cortisol levels in addition reduce hippocampal neurogenesis and functioning, which in turn negatively impacts inhibitory control on the HPA-axis [100]. HPA-axis dysregulation may in turn also disrupt SWS [99], leading to a cascade of dysregulated stress and autonomic functioning and chronically disrupted SWS, which is commonly thought to underlie insomnia [102].

4.2.2. Disturbed SWS as possible mechanism in BPD

Disturbed or reduced SWS, as often observed in BPD [15,28], may impair overnight regulation of sympathetic activity and adequate curtailing of HPA-axis functioning, resulting in heightened basal physiological arousal, increased levels of cortisol, a failing prefrontal control over hyperactive limbic neurocircuitry, and hampered memory consolidation. This could attribute to typical BPD symptoms such as affective instability, impulsivity and sustained emotional states. As described in 4.1, BPD is characterized by impaired vagal activity and sympathetic predominance, which is associated with emotion dysregulation and impulsivity [77]. Reversely, longitudinal studies in adolescents with BPD additionally show that higher vagally mediated heart rate variability was associated with reduced NSSI [103] and better DBT treatment outcomes over time [104]. BPD is also associated with aberrant HPA-axis functioning, reflected by increased background cortisol levels and a blunted cortisol response, which may respectively indicate a perpetual state of arousal and habituation resulting from sustained periods of HPA-axis hyperactivity [105]. Again, a direct link with slow wave (or other) sleep remains uninvestigated in these BPD studies.

4.3. Circadian dysrhythmia

4.3.1. The role of circadian rhythm in emotion regulation

In non-clinical and other (non-BPD) psychiatric populations, like mood- and substance use disorders, circadian irregularities contribute to emotion dysregulation through several pathways. Circadian dysrhythmias, like high variability in sleep times, delayed sleep phase, and evening chronotype, are associated with impaired functioning of reward circuitry, leading to enhanced reward sensitivity, impulsivity, and substance use [106,107]. In addition, the suprachiasmatic nucleus (SCN), which governs circadian rhythms, also controls hypothalamic regions involved in aggression regulation and autonomic functioning [108]. Disrupted circadian rhythms may then impair these systems, resulting in sympathetic hyperarousal and promoting a fight rather than flight response during stress. Circadian dysrhythmia has been associated with more anger expression and maladaptive anger coping strategies in healthy people [109] and patients with depression and BD [110]. Circadian rhythm disturbances are furthermore thought to induce dissociation as they contribute to more labile sleep-wake cycles, which may facilitate transitions between states of consciousness [111,112].

4.3.2. Circadian dysrhythmia as possible mechanism in BPD

Circadian rhythm in BPD is often delayed and desynchronized, which may importantly contribute to BPD symptomatology, as evidenced by multiple correlational studies. Greater rhythm fragmentation, due to nightly awakenings or daytime naps, and more varying sleep-wake and rest-activity patterns, were shown to predict more impulsivity and emotional instability in BPD patients, while these associations were not found in healthy controls or BD patients [113,114]. BPD patients with a late chronotype reported more suicide attempts, suicidal ideation, and feelings related to increased suicide probability (i.e., hopelessness and negative self-image) than patients without a late chronotype [115]. Furthermore, misalignment to time-of-day societal and social demands, as seen in BPD, likely exacerbates BPD symptoms, similar to a jetlag-like syndrome increasing vulnerability to emotion dysregulation, fatigue, irritability, and impulsiveness [114]. Consequences of circadian irregularities on sleep quality and continuity, such as daytime sleepiness or increased sleep onset latency due to delayed sleep phase, may further exacerbate BPD symptoms [35]. Related to the mechanistic pathways described in 4.3.1, neuroimaging studies in BPD show impaired functioning of reward circuitry, as indicated by impaired activity in impulse control frontal areas (e.g., ACC, and dorsolateral PFC) during gambling and reward anticipation tasks [80,116]. This may contribute to the impulsive and pleasure-seeking behavior seen in BPD. Anger attacks and dissociation are common in BPD, but have so far not been linked to dysrhythmia-induced hypothalamic dysregulation of aggression or altering states of consciousness respectively.

4.4. Insomnia and other sleep continuity disturbances

4.4.1. The role of insomnia and sleep continuity disturbances in emotion regulation

Insomnia is widely considered a disorder characterized by hyperarousal, manifested across cognitive, emotional, neurological, and physiological domains, and is inherently linked to REM fragmentation, disturbed SWS, and circadian dysrhythmia [11,117]. The hyperarousal theory of insomnia offers a comprehensive framework for understanding the role of insomnia in emotion dysregulation [14,100] and the involved mechanisms are partly already discussed in paragraphs 4.1 to 4.3.

Disruptions in sleep continuity, such as those seen in insomnia, also have profound effects on emotion regulation [11,100,118]. On a behavioral level, experimental studies in non-clinical populations, e.g. using (partial) sleep deprivation schemes, show that sleep loss results in less adaptive emotion regulating behavior and more negative and less positive moods [119], and higher emotional (threat) sensitivity [118], although findings are not always consistent [120]. In different populations varying from non-clinical to non-BPD psychiatric patients, sleep disturbances, like insomnia, or poor (subjective) sleep quality are similarly associated with less adaptive emotion regulation, more negative and less positive moods [121,122], continued hyperarousal [123], as well as with increased impulsive and aggressive behavior [124,106], more dissociative symptoms [111], and more NSSI and suicidal behavior [125,126].

Both clinical and experimentally-induced forms of sleep discontinuity also impact the neural circuitry underlying emotion regulation, including impaired prefrontal-limbic functioning (as discussed in 4.1.1, 4.1.2. and 4.2.1.), autonomic functioning (as discussed in 4.1.1., 4.2.1. and 4.3.1.), HPA-axis reactivity (as discussed in 4.2.1.) and reward circuit functioning (as discussed in 4.3.1.).

4.4.2. Insomnia and sleep discontinuity as possible mechanism in BPD

Insomnia and other sleep discontinuities as well as poor sleep quality are mostly linked to BPD symptoms through pre-sleep hyperarousal and nightly maladaptive behavioral regulation strategies to avoid or suppress emotional distress. People with BPD who have higher insomnia severity experience more difficulties in self-care and in emotional, social, and cognitive (memory) functioning [25]. Poor sleep quality mediates the association between BPD symptom severity and NSSI and suicide risk, with heightened emotion dysregulation due to poor sleep quality additionally explaining more NSSI (but not suicide risk) [27]. Longer sleep onset latency predicts more negative affect and suicidal ideation in BPD patients and poor sleep quality also predicts within-patient next-day negative affect [127]. Similarly, longer sleep

onset latency, shorter sleep duration, and spending more time in bed, which is often an attempt to (ineffectively) escape from complex BPD-related daytime difficulties [128], predict more NSSI and suicidal behavior in BPD patients [127,129]. These sleep disturbances have both nighttime (e.g., loneliness while awake, rumination) and daytime (e.g., fatigue, impaired social and occupational functioning) consequences that may worsen symptoms key to NSSI and suicidal behavior, such as feeling powerless, unconnected or a burden to others [129]. Poor sleep quality and insomnia in BPD are additionally associated with more impulsivity, particularly increased restlessness and difficulty controlling thoughts [130]. BPD patients tend to engage in maladaptive cognitions about their sleep more often [15], which enhances (pre-sleep) hyperarousal and emotion dysregulation [121] and is considered a key mechanism linking insomnia symptoms and suicide risk [131]. With respect to developing BPD, a longitudinal cohort study showed that shorter sleep duration and later bedtimes in childhood are predictive of BPD onset later in life, also when accounting for other risk factors (e.g., family adversity, childhood abuse) [132]. Good sleep quality may enhance treatment effectiveness in BPD and reduce risk of relapse. While research showed dialectical behavior therapy (DBT) initially improved BPD symptoms and sleep quality, these effects were not sustained, whereas better subjective sleep quality at the start of and directly after DBT were associated with greater symptom improvement and lower relapse rates [90]. In contrast, diverse subjective sleep problems in BPD patients have been shown to negatively impact BPD treatment. Specifically, disturbed sleep/wake behavior (e.g. long sleep onset latency, oversleeping) and shorter sleep duration at admission to a psychiatric inpatient treatment program for adolescents, predicted more BPD symptoms and emotion dysregulation at discharge, as well as at 6 months post-discharge, even after controlling for (post-)discharge sleep disturbance levels, depression, and anxiety [133]. In another study, non-recovered BPD patients had lower sleep quality and longer sleep onset latency, used more sleep medication, and experienced more daytime difficulties due to disturbed sleep compared to recovered BPD patients [134].

4.5. Nightmares

4.5.1. The role of nightmares in emotion regulation

Nightmares may contribute to emotion dysregulation mostly through an association with increased suicidality and REM sleep disturbances. In both community and non-BPD psychiatric populations, longitudinal and meta-analytic evidence points towards a strong link between nightmares and increased suicide ideation and risk [126,135], through increased feelings of hopelessness and entrapment and a disruption of overnight emotional regulation processes, which may increase the risk for maladaptive coping strategies such as NSSI and suicidal behavior [136,137]. Nightmares, occurring predominantly but not exclusively during REM sleep, may cause REM sleep fragmentation and consequently disturb emotional memory consolidation (see 4.2.1.). Patients with nightmare disorder showed increased emotional arousal during REM sleep [138]. An experimental nap study additionally showed that people with frequent nightmares, who had more fragmented REM sleep than controls, had sustained priming effects in response to emotional stimuli over time, whereas this effect dissipated in controls [139]. Nightmares are also accompanied by increased sympathetic and decreased parasympathetic tone [140,141], fragmented NREM sleep, and less SWS [142].

4.5.2. Nightmares as possible mechanism in BPD

Nightmares are highly frequent in BPD and may play a pivotal role in aggravating BPD symptoms [16,40]. Correlational studies support this notion by linking more nightmares to higher BPD symptom severity and suicide risk [42]. BPD patients with comorbid nightmare disorder showed more NSSI, suicidal behavior, substance abuse, and dissociative symptoms than patients without nightmare disorder [38]. Nightmares are also implicated in the development of BPD symptoms over time, as the experience of frequent nightmares in childhood increased the risk of BPD symptom development in adolescence [43]. BPD patients with higher symptom severity in addition reported more anxiety-, fear-, and frustration-themed dreams, which was partly due to higher rejection sensitivity [143]. On a behavioral level, nightmares may enhance emotion dysregulation by increasing severe day- and nighttime rumination and negative affect, which then leads to more nighttime arousal and subsequent nightmares, as shown in an experience sampling study in BPD patients [39]. These effects were significantly stronger in BPD patients compared to non-clinical controls. Nightmares may in addition hamper nighttime emotional processing by disrupting sleep continuity, with BPD patients reporting that the nightmare-related fear of sleep often delayed sleep onset, and resulted in difficulty getting back to sleep after waking due to nightmares [128]. The association between nightmares and suicidal behavior in BPD may be partly explained through (nightmare-related) insomnia, as insomnia severity, rather than nightmare frequency or distress, is reported to mediate the relation between BPD symptoms and suicide risk [41].

4.6. BPD symptoms negatively impact sleep

4.6.1. The role of emotion dysregulation in sleep disturbance

Thus far we outlined several pathways through which disturbed sleep impacts emotion regulation. Reversely, poor emotion regulation also predicts poor sleep. Daily emotional and stressful experiences may disrupt subsequent sleep, and require adaptive emotion regulation to modulate their detrimental effect on sleep [122]. Difficulties with emotion regulation, such as proneness to rumination and resulting (pre-sleep) hyperarousal, may lead to sleep disturbance and insomnia [121]. Experimental studies in non-clinical populations consistently indicate that pre-sleep induction of stress and negative emotions leads to increased PSG-assessed sleep disturbances such as lower sleep efficiency, shorter sleep duration, less REM sleep, more awakenings from REM sleep, higher REM density, less SWS, and longer latency to SWS [122].

4.6.2. BPD as possible mechanism in sleep disturbance

Also in BPD, emotion dysregulation may aggravate sleep disturbances. Aspects inherent to BPD, such as overwhelming and uncontrollable emotions and a lack of adaptive emotion regulation strategies, are thought to increase sleep disturbance due to sleep disrupting behavior, nighttime rumination, and pre-sleep hyperarousal [26,144]. Impulse control difficulties may additionally foster sleep disturbance in BPD by worsening sleep-disruptive behavior [144]. Because of the cross-sectional nature of these studies, further longitudinal and experimental studies are needed to determine true causal directionality. BPD patients additionally report that a lack of routine, often due to chaotic and erratic lifestyles and too little or irregular occupational and social activity, prevents them from establishing healthy and consistent sleeping patterns [128].

4.7. Concluding remarks on the reciprocal relation between sleep and BPD

In sum, despite only sparse experimental evidence in BPD samples, we propose a heuristic framework in which the described neurobiological and behavioral pathways maintain a vicious cycle of disturbed sleep and emotion dysregulation symptoms underlying BPD. Importantly, multiple other mediating and moderating factors, beyond the scope of this narrative review, may affect this reciprocal relation. Sex differences may influence the dynamics of the discussed pathways, for instance concerning the effect of fluctuating sex hormone levels on sleep quality and circadian rhythm [145,146]. Likewise, there may be a moderating role of early life trauma with pronounced and sustained (epigenetic) effects on both HPA-axis functioning, emotion-related neurocircuitry and sleep [11,147,148]. Finally, highly prevalent psychiatric

Box 2
Practice Points

- In this review, we propose neurobiological and behavioral pathways that may underlie the reciprocal relation between disturbed sleep and emotion dysregulation in BPD.
- REM sleep disturbances, associated with sustained LC-firing, heightened noradrenergic signaling and possibly desynchronized theta coupling, may hamper adaptive regulation of emotional memories and experiences, leading to sustained next-day emotional distress, hyperarousal, emotional reactivity and affective instability. The associated sleep-disruptive behavior may in turn negatively impact sleep.
- SWS disturbances may impair overnight regulation of parasympathetic activity and suppression of HPA-axis functioning, leading to failing prefrontal control over limbic hyperreactivity. This likely results in increased basal arousal and emotional reactivity, which may contribute to affective instability, impulsivity, and maladaptive emotion regulatory strategies.
- Circadian irregularities (e.g. delayed or desynchronized sleep phase), associated with impaired functioning of reward circuitry and misalignment to time-of-day demands, may increase reward sensitivity, impulsivity, emotional dysregulation, dissociation, and suicidal behavior. The resulting sleep-disruptive behavior and inconsistent sleeping patterns may in turn negatively impact sleep.
- Insomnia and sleep discontinuity likely increase emotion dysregulation, impulsivity and suicidal behavior in BPD, mainly through pre-sleep hyperarousal and maladaptive nighttime emotion regulation strategies, and may additionally hamper BPD treatment effectiveness.
- Nightmares may, next to causing REM fragmentation, lead to severe emotional distress and (nightmare-related) insomnia, which may increase emotion dysregulation, suicidal behavior, substance abuse and dissociation.
- Sleep (disturbance) receives too little attention in clinical guidelines for BPD, whereas the described hypothesized pathways may open up new avenues for sleep-based treatments (see Box 3: Research agenda).

comorbidities such as PTSD and depression may influence the relation between sleep and emotion dysregulation in BPD, as they are also strongly related to (REM) sleep disturbances [149]. Importantly, while the proposed reciprocal relation shares similarities with these other disorders [13,44], it is not fully accounted for by comorbid complaints such as depression [27,127,83,129,130], substance use [25,27] or PTSD [38], and is sometimes even stronger than in psychiatric disorders traditionally linked to sleep disturbances such as BD [113,114]. This is further supported by longitudinal evidence in a group of personality disorder patients, showing that decreased severity of the personality disorder predicted improvement in sleep disturbance, regardless of changes in anxiety and depression [150]. Moreover, controlling for changes in personality disorder severity diminished the previously observed positive effects of recovery from anxiety or depression on improved sleep. Finally, a recent network analysis exploring associations between insomnia and several psychiatric disorders additionally revealed that insomnia was directly linked to BPD symptoms, next to symptoms of depression and generalized anxiety [151]. This emphasizes the importance of disturbed sleep in BPD patients.

5. Future treatment options

The presumed reciprocal relation between sleep disturbances and BPD symptoms supports the potential for treating sleep problems in BPD patients (see Box 2). So far, however, despite a growing interest in disturbed sleep in personality disorders [15–17], including as outcome measure in treatment studies [18], there is little to no consideration of sleep disturbances and sleep treatment in clinical guidelines for BPD [2,3,19]. Below we shortly outline four promising sleep-based strategies to treat BPD (also see Box 3).

5.1. Cognitive behavioral therapy for insomnia

Cognitive behavioral therapy for insomnia (CBT-I) is considered the first-choice insomnia treatment with proven effectiveness also in psychiatric patients with co-morbid insomnia complaints (i.e., depression, PTSD, alcohol dependency, schizophrenia and BD) [152,153]. CBT-I in BPD may help interrupt the detrimental cycle between disturbed sleep and BPD symptoms. Treatment targets such as lowering nighttime rumination, which reduces pre-sleep arousal, may improve sleep quality and insomnia [121,154], boost BPD treatment effectiveness [155], and reduce rumination-related suicide risk in BPD [131], while improving sleep efficiency through bedtime restriction possibly targets the observed relation between sleep discontinuity in BPD and NSSI and suicidal behavior [127,129]. Given sleep's role in memory consolidation, CBT-I prior to psychotherapeutic BPD treatment may additionally facilitate long-term storage of adjusted maladaptive memories or beliefs and newly acquired cognitive and behavioral skills [156]. Together, this is the rationale for a currently running clinical trial assessing the effectiveness of guided, online CBT-I prior to standard psychotherapeutic treatment in patients with BPD and comorbid insomnia complaints [157]. Future studies could additionally assess effectiveness of CBT-I for BPD using other modes of delivery, such as regular (face-to-face) CBT-I or group-delivered CBT-I.

5.2. Targeting noradrenergic overdrive and fragmented REM sleep

Fragmented REM sleep and the associated heightened noradrenergic signaling, can be targeted behaviorally through interventions such as (early morning) sleep restriction [158,159] or implementation of evening blue-depleted light environments (BDLEs) [160]. Although these approaches have not yet been tested in BPD patients, they could be integrated into clinical studies to evaluate their effects on both REM fragmentation and BPD-related outcomes, such as symptom severity, overnight regulation of emotional distress, and potential adverse effects (e.g., sleep deprivation-related affective instability). Psychiatric inpatient settings may offer a promising starting point for structured evaluation, with nurse supervision supporting consistent adherence to sleep restriction and scalable implementation of BDLEs through ward-wide lighting adjustments during evening hours. Future studies should also explore their applicability in outpatient settings, where implementation may be more variable.

Nighttime noradrenergic overdrive can also be targeted pharmacologically. So far, only a few studies in BPD patients (with and without comorbid PTSD) indicated effectiveness of clonidine, an alpha-2-adrenergic-receptor agonist lowering noradrenaline levels, and doxazosin, an alpha-1-adrenergic receptor antagonist blocking postsynaptic noradrenergic action. Clonidine improved sleep quality, hyperarousal, and BPD symptom severity [161], and doxazosin reduced nightmare severity and frequency, improved sleep quality, and reduced wakefulness after sleep onset [162]. Since these studies did not objectively measure sleep, it cannot be determined whether reducing noradrenergic tone through these drugs also resulted in consolidated REM sleep. In this context, suvorexant, a dual orexin receptor antagonist suppressing LC activity, seems promising by simultaneously increasing REM duration while reducing REM fragmentation in patients with

Box 3
Research Agenda

Clinical trials testing sleep-based interventions for BPD.

- Cognitive behavioral therapy for insomnia (CBT-I), stand-alone or prior to standard psychotherapeutic BPD treatment.
- Behavioral interventions aimed at increasing consolidated REM sleep (e.g. sleep restriction, evening blue-depleted light environments (BDLEs)).
- Drugs aimed at reducing nocturnal noradrenergic signaling and possibly increasing consolidated REM sleep (e.g. clonidine, doxazosin, suvorexant), possibly as adjuvant to psychotherapeutic treatment.
- Behavioral and pharmacological interventions aimed at stabilizing circadian rhythm disturbance (e.g. chronotherapy (possibly including BDLEs), CBT-I, melatonin, or interpersonal and social rhythm therapy).
- Psychotherapeutic and pharmacological interventions aimed at nightmare distress (e.g. imagery rehearsal therapy (IRT), prazosin).

Observational studies investigating sleep-related mechanisms underlying BPD.

- In-depth EEG-analyses on polysomnographic studies to elucidate the micro architecture of BPD sleep (e.g. concerning REM fragmentation, spindles analysis, spectral analysis of theta and other frequency bands).
- Sleep studies assessing the day-to-day inter-relatedness of (disturbed) sleep parameters and BPD symptoms (possibly using ambulatory EEG-devices).
- Including sleep-related outcome measures in BPD treatment studies.

trauma related insomnia [163]. The effects of these drugs can be tested stand-alone or as adjuvant to psychotherapeutic treatment in BPD.

5.3. Circadian dysrhythmia treatment

Targeting the observed circadian rhythm disturbances in BPD may improve BPD symptoms such as impulsivity and emotion dysregulation. One small study in BPD patients provided preliminary evidence that light therapy (i.e., bright light in the morning) advanced sleep phase, and decreased rise-time variability, nighttime activity/arousal, and daytime sleepiness [164]. Alternative interventions aimed at stabilizing circadian dysrhythmia (e.g. chronotherapy or melatonin) or optimizing sleep/wake and social patterns (e.g. through interpersonal and social rhythm therapy [35]) may additionally hold promise for BPD. In BPD, circadian rhythm disturbance may result from sleep disruptive behavior and other daytime BPD symptoms and often coincides with insomnia [114]. In this case, CBT-I may offer a more comprehensive treatment approach to treat (circadian) sleep problems in BPD patients with co-morbid insomnia. A future trial could test the effectiveness of CBT-I with and without circadian rhythm support in BPD.

5.4. Nightmare treatment

The treatment of nightmares may consolidate REM sleep, foster emotional memory consolidation, reduce autonomic arousal, and improve NREM sleep [138–142]. In BPD patients specifically, this may reduce daytime emotion dysregulation, suicide risk, dissociation, and (pre-sleep) hyperarousal [38,39,42]. Imagery rehearsal therapy (IRT) is recommended for treatment of nightmares and reduces nightmare frequency and -distress, next to disorder-specific psychiatric symptoms in patients with affective and personality disorders [165]. A recent pilot study assessed effectiveness of IRT in BPD patients and showed reduced levels of subjectively reported anxiety, trauma-related intrusions, and hyperarousal [166]. Sleep quality, emotion dysregulation, and depressive symptoms also significantly decreased after IRT, but these improvements did not differ between the treatment and control groups. Objective sleep macro- and microstructure parameters assessed using PSG did not improve after IRT [166]. Pharmacological studies investigating noradrenaline-antagonizing drugs, like prazosin, are mostly restricted to PTSD patients and show mixed, but overall small favorable results [167]. In BPD patients, only the effect of doxazosin has been assessed, which reduced nightmare severity and -frequency, although controlled studies are needed to replicate these findings [162]. Future trials could explore whether a combination of IRT and pharmacological interventions targeting noradrenergically-driven hyperarousal and REM fragmentation more directly, could offer relieve for BPD patients with severe nightmare disturbances.

6. Further detailing the (causative) role of sleep in BPD

We suggest several lines of research to detail the hypothesized causative role of sleep disturbances in BPD. First, PSG-based studies in BPD patients may benefit from in-depth EEG-analyses to elucidate the micro-architecture of BPD sleep. Analyses of sleep parameters like REM fragmentation, spindles count/power and spectral analyses of theta activity and other frequency bands during sleep may provide further insight into the neurobiological mechanisms linking sleep disturbance and BPD pathology. Currently, an ongoing PSG-study assesses the role of (fragmented) REM and NREM sleep in overnight alleviation of experimentally-induced emotional distress in BPD patients, and its correlation to DBT effectiveness in a longitudinal set-up [168]. Second, studies assessing the inter-relatedness of sleep and BPD symptoms on a day-to-day basis may shed light on the presumed bidirectional relation between sleep disturbances and BPD, as hypothetically outlined in Fig. 1. One such study already showed that worse subjective sleep quality preceded next-day exacerbation of negative affect in BPD patients [127]. Objectively measuring sleep in such studies becomes more feasible with ambulatory EEG-devices that permit daily recordings for extended periods of time in home-settings [169]. Integrating such sleep measurements into ongoing BPD treatment may reveal the temporal interplay between certain sleep parameters, such as REM fragmentation, and (changes in) BPD symptoms during active treatment. Existing DBT treatments that already make use of daily diary assessments to track behaviors, moods and use of therapeutic skills [170] seem especially suited for this. Third, sleep outcome parameters may be included in BPD treatment studies. Apart from the already discussed studies testing sleep-focused interventions in BPD (e.g. IRT [166], doxazosin [162] and bright light treatment [164]), there is currently only one BPD-treatment study reporting on sleep. Here preliminary evidence suggests that DBT may temporarily improve sleep quality in BPD patients [90]. Additionally, the effect of trauma therapy on sleep has been assessed in BPD patients with co-morbid PTSD, resulting in reduced sleep latency and

less EEG arousals (i.e., abrupt shifts in EEG frequency) [91], and lower insomnia severity [171]. A current RCT comparing DBT and schema therapy in patients with BPD will report on the effect of these interventions on insomnia severity and nightmare frequency [172]. In all of the above suggested studies, researchers could include information on factors potentially moderating the relation between sleep (disturbances) and BPD symptoms (e.g., sex, psychiatric comorbidities, early life trauma)).

7. Conclusion

This narrative review discussed the reciprocal relation between inherent sleep problems in BPD, such as insomnia, nightmares and circadian dysrhythmia and emotion dysregulation symptoms of the disorder. Based on sleep's role in emotional memory and regulation, we described a vicious cycle of disturbed emotional processing and sustained hyperarousal during the night which potentially aggravate daytime BPD symptoms and associated sleep-disturbing behavior, which in turn may negatively impacts sleep. Currently however, sleep disturbance receives little attention in clinical guidelines and psychotherapies for BPD. Offering more sleep-focused treatment to patients with BPD seems promising to improve sleep and reduce BPD symptoms, but so far studies are few and small. In the review, we included promising potential sleep treatment approaches in BPD, that are relatively short and can be administered prior or in parallel to standard BPD treatment, such as CBT-I and pharmacological modulation of nightly noradrenergic (over)activity. With current treatment options for BPD being only partly effective, generally lengthy and not always accessible for patients, these new sleep-focused treatment strategies are promising and much needed. We hope this review provides the necessary background for these studies to succeed and introduce sleep as a new treatment target to alleviate the tremendous disease burden associated with BPD.

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