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Relationship between sleep disturbance and recovery in patients with borderline personality disorder

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Abstract

Objective—Patients with borderline personality disorder (BPD) frequently experience sleep disturbance, however, the role of sleep quality in the course of BPD is unknown. The purpose of this study was to evaluate the cross-sectional association between sleep quality and recovery status (symptomatic remission plus good concurrent psychosocial functioning) in a well-characterized cohort of patients with BPD to examine the role of sleep disturbance in the course of the disorder.

Methods—223 patients with BPD participating in the McLean Study of Adult Development (MSAD) were administered the Pittsburgh Sleep Quality Index (PSQI) as part of the 16-year follow-up wave. Sleep quality was compared between recovered (n=105) and non-recovered (n=118) BPD participants, including adjustment for age, sex, depression, anxiety, and primary sleep disorders.

Results—Non-recovered BPD patients had significantly worse sleep quality than recovered BPD participants as measured by the global PSQI score (adjusted means 12.01 vs. 10.73, p=0.03). In addition, non-recovered BPD participants had longer sleep onset latency (adjusted means 39.20 vs. 28.11 minutes, p=0.04), as well as increased odds of using sleeping medication (adjusted OR 1.49, p=0.009) and experiencing daytime dysfunction as a result of their sleep disturbance (adjusted OR 1.48, p=0.008).

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CONFLICT OF INTEREST STATEMENT

All authors have completed the Unified Competing Interest form at http://www.icmje.org/coi_disclosure.pdf. No authors received support from a commercial interest for the submitted work; Dr. Plante has received royalties from Cambridge University Press, honoraria from Oakstone Medical Publishing, and formerly owned stock in Pfizer in the past three years that could be perceived to constitute a conflict of interest; No spouses, partners, or children of have financial relationships that are relevant to the submitted work; and no authors have non-financial interests that may be relevant to the submitted work.

Conclusion—These results demonstrate an association between subjective sleep disturbance and recovery status among BPD patients. Further research is indicated to evaluate the mechanisms underlying sleep disturbance in BPD, and whether treatment of sleep complaints improves the symptomatic and psychosocial course of the disorder.

Keywords

borderline personality disorder; sleep; insomnia

Introduction

Sleep disturbance is a common symptom encountered across the spectrum of psychiatric illness that affects the course of multiple disorders. In particular, prospective morbidity studies have demonstrated that untreated insomnia, a form of sleep disturbance characterized by difficulty initiating or maintaining sleep despite adequate opportunity for sleep, is associated with an increased risk of major depressive disorder (MDD) [1-8] and anxiety disorders [2,3,5,6,9,10]. Moreover, insomnia is a highly treatment-resistant symptom [11-13], increases the risk of relapse to depressive episodes [12,14,15], and increases suicidal ideation and the risk of suicide [16-18], highlighting the need for further research in the role sleep disruption plays in the course of psychiatric illness.

Borderline personality disorder (BPD) is a common psychiatric disorder characterized by interpersonal hypersensitivity, affective dysregulation, self-damaging impulsivity, and identity disturbance, that is a significant cause of both morbidity and mortality [19]. Although patients with BPD frequently present with sleep-related complaints in clinical settings, sleep disturbance is not part of the DSM-IV diagnostic criteria for the disorder [20], and compared to mood and anxiety disorders, there is relatively little research that explores the relationship between sleep disturbance and BPD. Some prior investigations that have utilized polysomnography (PSG) to examine objective sleep measures in BPD have demonstrated alterations in rapid-eye movement sleep, slow wave sleep, and/or measures of sleep latency and continuity, however results have not been consistent across studies (reviewed in [21]), and such PSG abnormalities are not specific to BPD as they can be similarly observed in other psychiatric disorders [22].

Impaired sleep quality in BPD is also frequently reported in studies that utilize subjective assessment. Using the Pittsburgh Sleep Quality Index (PSQI), a well-validated and widely used psychometric measure to assess sleep quality, Phillipsen and colleagues found non-depressed BPD subjects reported significantly worse sleep quality relative to healthy controls [23]. Similar self-reported decrements in subjective sleep quality in BPD patients have also been reported utilizing other psychometric batteries [24,25]. In addition, a cross-sectional study found that the PSQI correlated with psychometric measures of borderline personality symptomatology and self-harm inventories, suggesting that patients with more severe symptoms of the disorder may have more severe sleep disturbance [26]. However, this investigation did not control for other psychiatric comorbidities (e.g., mood and anxiety disorders) that might cause sleep disturbance, and was performed in a sample of internal medicine outpatients rather than a well-characterized cohort of clinician-diagnosed BPD patients.

Despite evidence supporting the contention that BPD patients experience impaired sleep quality, to our knowledge, the role sleep disturbance may play in the course of BPD has not been previously evaluated. There are two concurrent longitudinal cohort studies that examine the course of BPD, the McLean Study of Adult Development (MSAD) and the Collaborative Longitudinal Personality Disorders Study (CLPS). Longitudinal data from

MSAD, collected over 16 years of prospective follow-up, suggests that 40-60% of BPD patients attain recovery from BPD, defined as remission of symptoms as well as good social and vocational functioning, but that 20-44% of BPD subjects have a subsequent loss of this recovery from illness [27]. Ten year data from CLPS corroborates high rates of remission of BPD symptoms, with an attenuated rate (21%) of patients attaining good psychosocial functioning [28]. Given the known effects sleep disturbance has on the course of other psychiatric disorders, and the subjective sleep disturbance experienced by patients with BPD, we sought to examine cross-sectional data on sleep complaints in recovered and non-recovered BPD subjects in MSAD. Given the prior literature in mood and anxiety disorders, we hypothesized that non-recovered BPD subjects would have worse self-reported sleep quality compared to recovered BPD participants.

Method

Subjects

The current study utilized data from the McLean Study of Adult Development (MSAD), a naturalistic, longitudinal study of the course of BPD. The details of the design and methodology of MSAD are described in detail elsewhere [29]. In brief, subjects were first identified during hospitalization at McLean Hospital in Belmont, Massachusetts. Initial inclusion criteria included age 18 to 35 years; known or estimated IQ greater than or equal to 71; no prior or incident symptoms of schizophrenia, schizoaffective disorder, bipolar I disorder, or an organic etiology that could cause psychiatric symptoms; and fluency in English. At the initiation of the study, a total of 362 subjects were enrolled: 290 meeting criteria for BPD, 72 subjects meeting criteria for other personality disorders. Participants have subsequently been re-assessed (described below) at two-year intervals. In terms of continuing participation, 87.5% (N=231/264) of surviving borderline patients (13 died by suicide and 13 died of other causes) were reinterviewed at all eight follow-up waves. The current study utilized cross-sectional data from the most recent completed follow-up wave (16-year) and available information on 223 BPD patients as we did not obtain self-report measures on eight BPD subjects.

Procedures

After study procedures were explained at baseline, written informed consent was obtained. A masters-level interviewer conducted a comprehensive diagnostic assessment blinded to the subject's clinical diagnosis. Three semi-structured diagnostic interviews were administered: the Structured Clinical Interview for DSM-III-R Axis I Disorders (SCID-I), the Revised Diagnostic Interview for Borderlines (DIB-R), and the Diagnostic Interview for DSM-III-R Personality Disorders (DIPD-R) [30-32]. Inter-rater and test-retest reliability of these three measures have been found to be good to excellent [33,34].

At each follow-up wave 24 months apart, staff members blind to baseline diagnoses reassessed Axis I and II psychopathology. Informed consent was obtained again in each case, and the aforementioned diagnostic battery was re-administered. The follow-up inter-rater and longitudinal reliabilities of these three measures have also been found to be good to excellent [33,34].

To explore the role of sleep disturbance in the course of BPD, the Pittsburgh Sleep Quality Index (PSQI) was included in the 16-year follow-up wave [35]. This questionnaire consists of directed response and likert-scale (4-point) questions regarding seven component areas: sleep quality, latency, duration, efficiency, disturbance, use of sleeping medications and daytime dysfunction over the preceding month. Responses in each category are scored to range from 0 to 3, and the global score equals the sum of the seven components (maximum

score 21), with higher scores indicating greater sleep disturbance. In addition, participants were queried regarding self-reported primary sleep disorders including obstructive sleep apnea (OSA) and restless legs syndrome (RLS), which are common sleep disorders that can disrupt sleep and diminish sleep quality [36,37].

Definition of Recovery From Borderline Personality Disorder

Recovery from BPD was defined as having remission of symptoms on both of our BPD interviews as well as attaining good social and vocational functioning during the preceding two years [27,38]. Specifically, BPD participants in symptomatic remission no longer met study criteria for BPD (DIB-R criteria and DSM-III-R). In addition, a global assessment of functioning (GAF) score of 61 or higher (which no BPD subject had at baseline) was required to define symptomatic recovery. This GAF threshold offers a reasonable description of positive overall clinical outcome with some mild symptoms or difficulties in social or occupational functioning, but generally functioning fairly well and engaging in some meaningful interpersonal relationships. As in prior studies from our laboratory, the GAF score was operationalized such that a score of 61 or higher required symptomatic remission from BPD and at least one emotionally sustaining relationship with a close friend, life-partner, or spouse, as well as consistent participation in full-time work or school [27,38].

Statistical Analyses

Chi-squared tests and t-tests were used to compare demographic and clinical characteristics between recovered and non-recovered BPD groups. Simple linear regression was used to compare overall PSQI scores between groups, which was the primary outcome measure of interest, as well as the continuous variables that could be derived from the PSQI including self-reported sleep onset latency (SOL), total sleep time (TST), and sleep efficiency (SE). The Cochran Armitage trend test was used to compare ordinal PSQI component scores (component 1: subjective sleep quality, component 5: sleep disturbance, component 6: use of sleeping medication, and component 7: daytime dysfunction) between groups. Ordinal logistic regression (proportional odds model) was used to compare ordinal PSQI component scores between recovered and non-recovered BPD adjusted for potential confounding variables. Both linear and ordinal logistic regression models included age, sex, current depressive episode, anxiety disorders (panic disorder, post-traumatic stress disorder (PTSD), and generalized anxiety disorder (GAD)), obstructive sleep apnea (OSA), and restless legs syndrome (RLS) as covariates in these adjusted analysis. Similar to prior studies, the anxiety disorders included in the adjusted analyses were selected because they are the three anxiety disorders most frequently associated with sleep disturbance [39,40]. Statistical significance was fixed at $\alpha=0.05$. Statistical analyses were performed using JMP®Pro 10.0 (SAS Institute, Inc., Cary, NC).

Results

Table 1 presents clinical variables of interest for recovered and non-recovered BPD subjects. Recovered BPD subjects were significantly younger than non-recovered subjects. Additionally, non-recovered subjects had higher prevalence of current panic disorder, PTSD, current major depressive episode, and obstructive sleep apnea. The proportion of men and women were not significantly different between groups.

The primary outcome variable of interest, total PSQI score, was significantly different between non-recovered and recovered BPD subjects in the unadjusted analysis (11.06 ± 4.43 vs. 8.28 ± 3.78 ; $p<0.0001$) (Table 2). Similarly, a significantly higher proportion of non-recovered BPD subjects had a total PSQI score greater than 5, which is generally considered the cutoff for clinically significant sleep disturbance [33], compared to recovered BPD

subjects (89.0% vs. 72.4%, $p<0.002$). Significantly higher PSQI scores (which reflect greater sleep disturbance) remained in the non-recovered compared to recovered BPD groups after adjustment for age, sex, comorbid depression, anxiety, and sleep disorders (12.01 vs. 10.73; $p=0.03$) (Table 2).

Among self-reported sleep continuity variables, only SOL was significantly different between groups in the unadjusted analysis with non-recovered subjects reporting greater SOL than recovered subjects (40.23 ± 43.35 vs. 28.68 ± 23.52 minutes; $p=0.02$). SOL remained significantly elevated in non-recovered subjects after adjustment for all covariates (39.30 vs. 28.11 minutes; $p=0.04$) (Table 2). There were no significant differences between groups for total sleep time or sleep efficiency in either unadjusted or adjusted analyses.

Unadjusted analyses of ordinal PSQI component items demonstrated significant differences between recovered and non-recovered BPD groups on all items (Table 3). After adjustment for covariates, there was no significant difference between groups for component 1 (subjective sleep quality). However, the odds of non-recovered BPD subjects scoring higher on ratings of subjective sleep disturbance (component 5: OR 1.36, $p=0.04$), use of sleeping medications (component 6: OR 1.49, $p=0.009$), and daytime dysfunction related to sleep (component 7: OR 1.48, $p=0.008$) remained significantly elevated in the adjusted analysis. That is, non-recovered BPD subjects had approximately one and half times the odds of scoring higher on these PSQI components.

Discussion

These results confirm our hypotheses that non-recovered BPD subjects have worse subjective sleep quality than recovered BPD subjects, and that these differences remain after controlling for several covariates that can disrupt sleep including age, sex, co-morbid depressive episode, anxiety disorder, obstructive sleep apnea, and restless legs syndrome. Similar to the role insomnia as a specific form of sleep disturbance plays in the course of affective and anxiety disorders [1-10], our findings suggest that alterations in sleep quality may also be related to the course of BPD. Strengths of this investigation include a relatively large and well-characterized sample of BPD subjects that has been followed over time, which allowed for statistical adjustment for potential confounders and assessment of recovery status. These results are congruent with several prior reports of subjective sleep disturbance in BPD [23-26], and extend this area of inquiry by demonstrating that impaired sleep quality is associated with an absence of recovery from the disorder.

Although the primary outcome measure, the total PSQI score, was significantly different between non-recovered and recovered BPD subjects, secondary evaluation of individual components of the PSQI suggest potential domains in which sleep may be particularly impaired in non-recovered BPD patients. Specifically, even after controlling for covariates, non-recovered BPD participants experienced greater difficulty with sleep initiation of approximately 11 minutes, which was 40% higher compared to recovered subjects. Although this is a modest difference in sleep onset latency, it is of comparable magnitude to the clinical improvements in sleep latency observed in studies of sedative-hypnotics and cognitive behavioral treatments of insomnia [41,42,43]. In addition, non-recovered BPD subjects exhibited more severe daytime dysfunction resulting from sleep disturbance, as well as more frequent use of soporific medications to manage sleep difficulties than recovered subjects. These data do not allow for ascertainment of the causative factor(s) involved in these particular sleep difficulties. However, they may be related to the interplay of physiological and psychological phenomena observed in BPD.

One model of chronic insomnia is the hyperarousal hypothesis, which posits sleep difficulties result from increased arousal across a range of cognitive and physiological domains during both sleep and wakefulness [44,45]. There are few studies that have evaluated physiological hyperarousal in BPD. However, ambulatory monitoring has demonstrated BPD patients experience elevations in heart rate in response to emotional distress that is comparable to controls [46]. In such a context, the sleep period, which is frequently an isolative experience, may be difficult for some individuals with BPD, given the intolerance of aloneness experienced by many BPD patients [47]. Moreover, prior investigations have reported that some with BPD may have highly irregular sleep-wake patterns when assessed with actigraphy [48,49], which could either contribute to the perpetuation of sleep initiation difficulties due to inadequate sleep promoting behaviors, or could be the result of some other factor such as affective intolerance of the distress associated with the nocturnal sleep period.

It is in some ways not surprising that non-recovered BPD subjects would also have greater daytime dysfunction associated with sleep disturbance than their recovered counterparts. First, by definition, non-recovered BPD subjects have lower social and occupational functioning, and thus higher (i.e., worse) scores on this PSQI component item may simply reflect the definitions used to delineate BPD groups in this study. Second, as proposed by cognitive models of insomnia [50], non-recovered BPD patients may be overly worried about their sleep and the resulting daytime consequences of diminished sleep quality, resulting in heightened arousal and emotional distress. Although speculative, given the chronic dysphoria experienced by many with BPD [51], non-recovered BPD subjects may both overestimate the severity of their daytime dysfunction resulting from their sleep disturbance, and/or fail to recognize that their negative sleep-related cognitions may affect the quality of their sleep. Given the potential for such dysfunctional beliefs and attitudes about sleep in BPD, further research that explores sleep-related cognitions in the disorder may prove a fruitful area of research.

A particular area in which enhanced understanding of sleep-related cognitions in BPD may affect management of these patients is the use of sleeping medications. Our results in this study are consistent with prior work from our laboratory, which has demonstrated that BPD patients have a roughly three-fold increased odds of using sleeping medications than other personality disorder comparison subjects, even when controlling for other common psychiatric comorbidities [40]. That non-recovered BPD patients report more frequent use of soporifics than recovered BPD patients fits with the hypothesis that symptomatic BPD patients may seek to be unconscious at times of intolerable emotion and/or stress. Thus, the use of sedative-hypnotics by the patient and prescribing physician may be a form of collusion to treat a more conveniently manageable sleep complaint, rather than directly address the underlying emotional distress that is causing the sleep disturbance [52]. Alternatively, considered from a biological standpoint, increased use of sleeping medications in non-recovered BPD patients may reflect an attempt to suppress the physiological hyperarousal that can occur in BPD, a notion that is supported by recent positive data for the alpha-antagonist clonidine in treating sleep disturbance in the disorder [53]. However, there is limited evidence to support the use of any specific sleeping medication in the treatment of insomnia associated with BPD, and in fact, our data suggest sleep disturbance in other domains does not remit despite more frequent use of soporifics by non-recovered BPD patients, suggesting medications may not be an effective approach to managing sleep disturbance in BPD. Furthermore, because non-medication based approaches to chronic insomnia, particularly cognitive-behavioral approaches, have demonstrated efficacy that is equal, and in some instances superior, to pharmacologic approaches [54], further research on the efficacy of psychotherapeutic approaches to treat

insomnia in BPD are clearly indicated, as these may have a better risk-to-benefit profile than sedative-hypnotics in this population.

There are limitations of this study that merit discussion. First, there may have been unknown and thus uncontrolled covariates that were not included in the adjusted analyses that may have affected findings. However, by controlling for comorbid mood and anxiety disorders, as well as common primary sleep disorders associated with sleep disturbance, this risk is minimized as these are the most common disorders associated with difficulty initiating or maintaining sleep [55,56]. Conversely, the inclusion of self-reported sleep disorders (obstructive sleep apnea and restless legs syndrome) that were not confirmed with clinical evaluation and/or polysomnography, nor established through standardized psychometric batteries, could have resulted in over-correction of the data, however, this is unlikely as results of a secondary analysis were very similar when these factors were not included as covariates (data not presented). Finally, it should be stressed that the data utilized in this study are cross-sectional, though drawn from a longstanding longitudinal study, and thus we can not determine whether sleep quality predicts recovery or loss of recovery, only infer that sleep quality is related to the course of the disorder due to more severe sleep disturbances reported by non-recovered than recovered BPD patients. In this context, it is noteworthy that a high proportion of both recovered and non-recovered BPD subjects had clinically significant sleep disturbance, suggesting that resolution of sleep disturbance is not required for recovery from BPD, and that comorbid sleep disturbance may be a frequent residual problem in BPD, as is the case for mood disorders [57].

In summary, our results demonstrate non-recovered BPD subjects have significantly greater subjective sleep impairments than those who have attained symptomatic and psychosocial recovery. Although speculative, our results suggest the chronicity of certain aspects of the disorder may in part be due to ongoing sleep-related difficulties. Given these results and the role that sleep disturbance plays in the course of affective and anxiety disorders, future studies that examine the role of sleep quality in the longitudinal course of BPD are warranted. Specifically, prospective research that explores the role of sleep quality in attainment and/or loss of recovery in BPD, and the effects of sleep-related interventions (e.g. psychotherapeutic and/or pharmacologic) on BPD symptoms, may provide further insights regarding the role of sleep in the course of the disorder and lead to improved treatment strategies for these patients.

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Table 1

Demographic and Clinical Characteristics of Recovered vs. Non-recovered BPD

	Recovered	Non-Recovered	p-value
Total N	105	118	---
Mean Age±std dev	41.17±5.4	44.21±6.0	<0.0001
Sex N (%)			
Women	87 (82.9%)	94 (79.7%)	0.54
Current Axis I Disorders N (%)			
GAD	3 (2.9%)	5 (4.2%)	0.58
Panic Disorder	4 (3.8%)	20 (16.9%)	0.002
PTSD	3 (2.9%)	27 (22.9%)	<0.0001
Major Depressive Episode	3 (2.9%)	37 (31.4%)	<0.0001
Current Sleep Disorders N (%)			
OSA	8 (7.6%)	23 (19.5%)	0.01
RLS	9 (8.6%)	17 (14.4%)	0.17

Table 2
 Comparisons of Recovered vs. Non-Recovered BPD total PSQI score and self-reported sleep continuity variables

	Unadjusted						Adjusted*			
	Recovered		Non-Recovered		t-ratio	p-value	Recovered	Non-Recovered	t-ratio	p-value
	Mean	Std Dev	Mean	Std Dev						
Overall PSQI	8.28	3.78	11.06	4.43	-5.02	<0.0001	10.73	12.01	-2.18	0.03
SOL (min)	28.68	23.52	40.23	43.35	-2.43	0.02	28.11	39.20	-2.05	0.04
TST (hours)	6.74	1.43	6.72	2.16	0.06	0.94	6.19	6.60	-1.51	0.13
SE (%)	87.6	18.5	84.7	27.8	0.89	0.38	81.2	81.5	-0.07	0.94

SOL: sleep onset latency; TST: total sleep time; SE: sleep efficiency.

* adjusted for age, sex, depression, anxiety disorders (panic disorder, PTSD, and GAD), OSA, and RLS.

Table 3
Comparison of Recovered vs. Non-Recovered BPD ordinal component PSQI scores.

	Unadjusted										Adjusted*					
	Recovered				Non-Recovered						Z	p-value	Odds ratio	p-value		
	Subscale score n(%)				Subscale score n(%)											
0	1	2	3	0	1	2	3	0	1	2	3					
Subjective Sleep Quality (C1)	10 (9.5)	57 (54.3)	29 (27.6)	9 (8.6)	14 (11.9)	43 (36.4)	30,536 (3630.5)	25 (21.2)					-2.19	0.03	1.10	0.49
Sleep Disturbance (C5)	1 (1.0)	54 (51.4)	46 (43.8)	4 (3.8)	0 (0.0)	36 (30.5)	60 (50.9)	22 (18.6)					-4.18	<0.0001	1.36	0.04
Use of Sleeping Medication (C6)	60 (57.1)	9 (8.6)	7 (6.7)	29 (27.6)	34 (28.8)	7 (5.9)	7 (5.9)	70 (59.3)					-4.82	<0.0001	1.49	0.009
Daytime dysfunction (C7)	18 (17.1)	61 (58.1)	23 (21.9)	3 (2.9)	11 (9.3)	46 (39.0)	44 (37.3)	17 (14.4)					-4.23	<0.0001	1.48	0.008

* adjusted for age, sex, depression, anxiety disorders (panic disorder, PTSD, and GAD), OSA, and RLS.