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Relationship between maladaptive cognitions about sleep and recovery in patients with borderline personality disorder

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Abstract

Borderline personality disorder (BPD) has been associated with maladaptive cognitive processes including dysfunctional attitudes and a negative attribution style. Comorbid insomnia affects the course of multiple psychiatric disorders, and has been associated with absence of recovery from BPD. Because dysfunctional beliefs and attitudes are common among patients with insomnia, the purpose of this study was to evaluate the association between maladaptive sleep-related cognitions and recovery status (symptomatic remission plus good concurrent psychosocial functioning) in patients with BPD. 223 BPD patients participating in the McLean Study of Adult Development (MSAD) were administered the Dysfunctional Beliefs and Attitudes about Sleep questionnaire (DBAS-16) as part of the 16-year follow-up wave. Maladaptive sleep cognitions were compared between recovered (n=105) and non-recovered (n=118) BPD participants, in analyses that adjusted for age, sex, depression, anxiety, and primary sleep disorders. Results demonstrated non-recovered BPD patients had significantly more severe maladaptive sleep-related cognitions as measured by the overall DBAS-16 score. These results demonstrate an association between dysfunctional beliefs and attitudes about sleep and recovery status among BPD patients. Further research is warranted to evaluate treatments targeted towards maladaptive sleep-related cognitions, and their subsequent effects on the course of BPD.

Keywords

borderline personality disorder; insomnia; DBAS

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1. Introduction

Borderline personality disorder (BPD) is a common psychiatric disorder, that is a significant cause of morbidity and mortality, and is associated with considerable societal costs (Grant et al., 2008; Soeteman et al., 2008). As a disorder, BPD is characterized by pervasive patterns of identity disturbance, interpersonal difficulties, impulsivity, and affective dysregulation that result in significant functional impairment (American Psychiatric Association, 2000). In addition, BPD patients demonstrate disturbed cognitive processes, including non-psychotic thinking (e.g., non-delusional paranoia, unusual perceptions [e.g., depersonalization], and odd thinking [e.g., ideas of reference centering on beliefs that one is stupid, bad, or evil]) and more rarely, quasi-psychotic thinking, which improve, but variably resolve, over the longitudinal course of the disorder (Zanarini et al., 1990; Zanarini et al., 2013). Moreover, patients with BPD may attend to negative stimuli, make biased evaluations, and endorse a range of critical beliefs about themselves and their experiences (Baer et al., 2012). However, it is not clear how such maladaptive cognitive strategies may be related to the development and maintenance of BPD over time (Baer et al., 2012).

Insomnia is an important factor that affects the course of multiple psychiatric disorders. Prospective morbidity studies have demonstrated that untreated insomnia is associated with an increased risk of major depressive disorder (MDD) and anxiety disorders (Ford and Kamerow, 1989; Breslau et al., 1996; Chang et al., 1997; Weissman et al., 1997; Morphy et al., 2007; Neckelmann et al., 2007; Buysse et al., 2008; Jansson-Frojmark and Lindblom, 2008; Szklo-Coxe et al., 2010). In addition, insomnia is a highly treatment-resistant symptom (Carney et al., 2007; Dombrovski et al., 2007), increases the risk of relapse to depressive episodes (Paykel et al., 1995; Karp et al., 2004; Dombrovski et al., 2007), and increases suicidal ideation and the risk of suicide (Goldstein et al., 2008; Wojnar et al., 2009; Fitzgerald et al., 2011). Although not part of the current diagnostic criteria for BPD (American Psychiatric Association, 2000), sleep-related complaints are common in the disorder, with several studies demonstrating subjective sleep disturbance in BPD (Philipsen et al., 2005; Bastien et al., 2008; Schredl et al., 2012). Given the associations between insomnia and the course of other psychiatric disorders, and cross-sectional data which has correlated subjective sleep quality with measures of BPD symptomatology and self-harm inventories (Sansone et al., 2010), research that examines the role of sleep in the course of BPD is an important area of investigation.

The cognitive model of psychopathology, which has been applied to myriad psychiatric disorders, including BPD, suggests that the processing of external events and internal stimuli is biased, leading to distortion of a patient's construction of his/her experiences and resultant cognitive errors. Dysfunctional beliefs become incorporated into cognitive schemas, which tend to bias information processing and produce typical cognitive content of a given disorder (Beck, 2005). Cognitive models for insomnia have also been developed (Harvey, 2002), in which individuals with insomnia tend to be overly worried about their sleep and the consequences associated with their sleep disturbance, resulting in counterproductive behaviors and erroneous beliefs about sleep, which, in turn, exacerbate and perpetuate insomnia. Targeting dysfunctional beliefs about sleep is an important component of cognitive-behavioral therapy for insomnia (CBT-I), a highly efficacious therapy for both primary insomnia and insomnia comorbid with psychiatric disorders (Edinger et al., 2009; Morin and Benca, 2012). Notably, CBT-I results in decreases in maladaptive cognitions about sleep that further correlate with other areas of clinical improvement in insomnia sufferers, such as enhanced sleep efficiency and quality, suggesting that interventions targeted toward maladaptive sleep cognitions may have broad therapeutic effects (Edinger et al., 2001; Morin et al., 2002).

Despite the predisposition for maladaptive cognitive processes among BPD patients, and reports of sleep disturbance in the disorder, to our knowledge, prior studies have neither examined dysfunctional beliefs about sleep in BPD, nor assessed the association between maladaptive sleep cognitions and recovery from the disorder. Longitudinal data, collected over 16 years of prospective follow-up, from the McLean Study of Adult Development (MSAD), one of two longitudinal studies to investigate the course of BPD, 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 patients have a subsequent loss of recovery status (Zanarini et al., 2012). Recent cross-sectional analysis of MSAD data from the 16-year follow-up wave has further demonstrated an association between sleep disturbance, as measured by the Pittsburgh Sleep Quality Index (Buysse et al., 1989), and recovery status, such that non-recovered BPD patients were more likely to demonstrate impairments in global sleep quality, increased difficulties with sleep initiation, and higher rates of sedative-hypnotic use compared to recovered BPD patients, even when age, sex, and comorbid sleep and psychiatric disorders were included as covariates in adjusted analyses (Plante et al., 2013). Given the role dysfunctional sleep-related cognitions play in the development and perpetuation of insomnia, the contribution insomnia makes to the course of other psychiatric disorders, and cross-sectional data demonstrating association between sleep disturbance and absence of recovery from BPD, we examined maladaptive sleep-related cognitions in subjects participating in MSAD to explore the relationship between these dysfunctional thought processes and recovery from BPD. We hypothesized that non-recovered BPD patients would have more severe maladaptive cognitions about sleep compared to participants who had attained recovery from the disorder.

2. Methods

2.1. Subjects

The current study utilized data from the McLean Study of Adult Development (MSAD), a naturalistic, longitudinal study of the course of BPD, the methodologic details of which are described in detail elsewhere (Zanarini et al., 2003). In brief, participants were initially identified during hospitalization at McLean Hospital in Belmont, Massachusetts. Inclusion criteria at baseline 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 English fluency. 362 subjects were initially enrolled: 290 meeting criteria for BPD, 72 subjects meeting criteria for other personality disorders.

After study procedures were explained, written informed consent was obtained. A masters-level interviewer conducted a comprehensive diagnostic assessment blinded to the subject's clinical diagnoses. 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) (Zanarini et al., 1987; Zanarini et al., 1989; Spitzer et al., 1992). Inter-rater and test-retest reliability of these three measures have been found to be good to excellent (Zanarini and Frankenburg, 2001; Zanarini et al., 2002).

Participants have been subsequently reassessed at two-year intervals. At each follow-up wave 24 months apart, staff members have reassessed Axis I and II psychopathology blind to baseline diagnoses. Informed consent has been re-obtained again in each case, and the aforementioned diagnostic battery re-administered. The follow-up inter-rater and longitudinal reliabilities of these measures have also been found to be good to excellent (Zanarini and Frankenburg, 2001; Zanarini et al., 2002). In addition to semi-structured interviews, as part of the 16-year follow-up assessment, participants completed the

Dysfunctional Beliefs and Attitudes about Sleep, 16-item version (DBAS-16; see below for details) (Morin et al., 2007). In addition, subjects were queried regarding various aspects of their medical health, which specifically included self-report of physician's diagnosis of obstructive sleep apnea and restless legs syndrome, which are common sleep disorders that can disrupt sleep and diminish sleep quality (Earley and Silber, 2010; Luyster et al., 2010).

Study retention has been high, with 87.5% (N=231/264) of surviving borderline patients (13 died by suicide and 13 died of other causes) re-interviewed at all eight follow-up waves. The current study utilized 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.

2.2. Definition of recovery from borderline personality disorder

Recovery from BPD was defined as achieving remission of symptoms on both BPD interviews (DIB-R and DIPD-R), as well as attaining good vocational and social functioning during the preceding two years (Zanarini et al., 2010; Zanarini et al., 2012). Consistent with prior studies from our laboratory and retrospective studies on the course of BPD, a global assessment of functioning (GAF) score of 61 or higher (which no participant had at baseline) was utilized to define good psychosocial functioning (Plakun, et al., 1985; McGlashan 1986; Paris et al., 1987; Stone 1990; Zanarini et al., 2010; Zanarini et al., 2012). To operationalize and thus enhance the reliability and meaning of this measure, a GAF score ≥61 required symptomatic remission from BPD and at least one emotionally sustaining relationship with a close friend, life-partner, or spouse, as well as consistent and competent participation in full-time work or school. 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.

2.3. Measures of maladaptive sleep-related cognitions

The psychometric evaluation most commonly used to assess maladaptive cognitions about sleep is the Dysfunctional Beliefs and Attitudes about Sleep (DBAS) Scale, which contained in its original version 30 items that were rated on a 100-mm analog scale (Morin, 1993; Morin, 1994). To make the scale more user-friendly, a 16-item version (DBAS-16) was developed and validated that utilizes a Likert-type response (0–10) scale (Morin et al., 2007), which is the version utilized in this study. The DBAS-16 has been demonstrated to be reliable and valid, as evidenced by adequate internal consistency and temporal stability, as well as convergent validity demonstrated by significant correlations with other conceptually related self-report measures (Morin et al., 2007). Questions on the DBAS-16 fall into one of four thematic subscale/factors: 1) perceived consequences of insomnia (Consequence), 2) worry/helplessness about insomnia (Worry/Helplessness), 3) expectations about sleep (Expectations), and 4) attitudes about sleep medications (Medication). Five questions comprise the Consequence factor, each related broadly to the subject's perception of how insomnia affects energy, mood, and overall functioning. The Worry/Helplessness subscale is comprised of 6 questions, focused on perceptions and beliefs about how worried a subject may be about the unpredictable nature of his/her sleep, and how it may affect vitality and health. The Expectations factor is composed of two questions, regarding the belief that one cannot function without eight hours of sleep, and that napping or sleeping in the next day is necessary to catch up on sleep if preceded by a night of insomnia. Finally, the Medication subscale is comprised of 3 questions, which query the participant regarding beliefs that insomnia is essentially the result of a chemical imbalance and can only be managed by medications. The average of all 16 questions forms the overall DBAS-16 score, with higher scores indicating a greater degree of maladaptive sleep-related cognitions. The values of

questions relating to each thematic subscale may also be averaged to calculate scores for each of the four factors, again, with higher scores indicative of more severe maladaptive beliefs and attitudes about sleep (Carney et al., 2007).

2.4. Statistics

Chi-Squared and t-tests were utilized to compare demographic and clinical characteristics between recovered and non-recovered BPD groups. Simple linear regression was conducted to determine if DBAS-16 overall and factor scores (dependent variables) differed between recovered and non-recovered BPD groups (independent variable). Linear regression models included age, sex, current major depressive episode, current 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 adjusted analyses. The anxiety disorders (GAD, panic disorder, and PTSD) were included in the analyses as these are the primary anxiety disorders most frequently associated with sleep disturbance (Mellman, 2006). Statistical significance was fixed at $\alpha=0.05$. Statistical analyses were performed using JMP®Pro 10.0 (SAS Institute, Inc., Cary, NC).

3. Results

Demographic variables and rates of comorbid sleep and psychiatric disorders among recovered and non-recovered BPD patients are presented in Table 1. Non-recovered BPD subjects were significantly older than recovered subjects, however, both groups had a mean age in the fourth decade of life, and were separated by approximately three years. There was no between group difference in the percentage of men and women. The non-recovered BPD group had greater proportions of current major depressive episode, panic disorder, PTSD, and obstructive sleep apnea, relative to the recovered BPD group.

Overall DBAS-16 score, which was the primary outcome of interest, was significantly different between non-recovered and recovered BPD subjects in the unadjusted analysis (5.25±1.80 vs. 3.95±1.58; $p<0.0001$) (Table 2). Moreover, a greater proportion of non-recovered participants had overall DBAS scores greater than 3.8, considered to reflect a clinically significant degree of maladaptive sleep-related cognitions (83.1% vs. 54.3%; $p<0.0001$)(Carney et al., 2010). Significantly higher overall DBAS-16 scores remained among non-recovered compared to recovered BPD participants after adjustment for age, sex, and comorbid sleep and psychiatric disorders (adjusted means 5.66 vs. 4.80; $p=0.0009$) (Table 2).

Unadjusted analysis of DBAS-16 factor scores demonstrated significantly higher scores on Consequence, Worry/Helplessness, and Medication components among non-recovered relative to recovered BPD patients ($p<0.0001$ for each unadjusted comparison) (Table 2). Component scores for Consequence (adjusted means 6.12 vs. 5.32; $p=0.011$), Worry/Helplessness (adjusted means 5.29 vs. 4.54; $p=0.019$) and Medication (5.01 vs. 3.74; $p=0.0005$) remained significantly elevated among non-recovered subjects after adjustment for all covariates. The Expectation component score was not significantly different between groups in either unadjusted or adjusted analyses (Table 2).

4. Discussion

Our results confirm the hypothesis that non-recovered BPD patients have more severe and clinically significant maladaptive sleep-related cognitions compared to those who have recovered from the disorder. In addition, greater severity of dysfunctional beliefs and attitudes about sleep remains among non-recovered BPD patients after controlling for several covariates that could affect sleep including age, sex, comorbid depressive episode,

anxiety disorders, obstructive sleep apnea, and restless legs syndrome. 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 of potential confounders and assessment of recovery status. Our results are congruent with several prior reports of subjective sleep disturbance in BPD (Philipsen et al., 2005; Bastien et al., 2008; Sansone et al., 2010; Schredl et al., 2012), as well as recent data from our laboratory which have demonstrated an association between sleep disturbance and recovery from BPD (Plante et al., 2013). This study further extends this area of inquiry by demonstrating that maladaptive cognitions about sleep are associated with an absence of recovery from the disorder.

Although the primary outcome measure, the overall DBAS-16 score, was significantly higher among non-recovered compared to recovered participants, secondary evaluation of individual factors of the DBAS-16 suggests suggest particular areas in which non-recovered BPD patients may have prominent maladaptive sleep-related cognitions. Specifically, even after controlling for covariates, non-recovered BPD participants demonstrated more severe concern about the consequences associated with their sleep problems, greater worry and helplessness about their insomnia, and more fixed beliefs that medication was necessary to treat their sleep disturbance. Although these data do not allow for ascertainment of the causative factor(s) involved in these dysfunctional beliefs and attitudes about sleep, they do suggest potential areas in which cognitive-behavioral treatments may be optimally focused in the management of insomnia associated with BPD.

It is in some ways not surprising that non-recovered BPD subjects demonstrated higher (i.e. worse) thematic subscale scores regarding consequences of and worry about insomnia, given the aforementioned tendency of patients with BPD to have a negative attribution style and endorse critical beliefs about their experience (Mathews and MacLeod, 2005; Baer et al., 2012). In addition, BPD patients have previously demonstrated self-perceived helplessness (Butler et al., 2002), which may be reflected in elevated scores in the Worry/Helplessness subscale. Furthermore, by definition, non-recovered BPD subjects had lower psychosocial functioning than recovered participants, and thus higher scores in the Consequence factor may reflect this delineation between groups, as questions in this thematic subscale involve functional impairments that are perceived to be associated with insomnia. Although speculative, given the chronic dysphoria experienced by many with BPD (Korner et al., 2008), non-recovered BPD subjects may both overestimate the severity of their daytime dysfunction resulting from their insomnia, and/or fail to recognize that negative sleep-related cognitions may affect the quality of their sleep. Thus, correcting maladaptive sleep-related cognitions related to these themes using CBT-I may be a fruitful area of future investigation, given clinical improvement in insomnia observed in other psychiatric disorders using this psychotherapeutic modality (Manber et al., 2008; Edinger et al., 2009).

Dysfunctional beliefs and attitudes about the necessity of sedative-hypnotic medications to treat insomnia, which were more severe in non-recovered BPD patients in this study, may be a particularly important issue in the management of insomnia in patients with BPD. Prior work from our laboratory has demonstrated BPD patients are approximately three times more likely to take sedative hypnotic medications compared to Axis II comparison subjects, even when adjusted for comorbid mood and anxiety disorders (Plante et al., 2009). Additionally, recent analysis of data from the MSAD 16-year follow-up wave suggests non-recovered BPD patients are significantly more likely to take medications to help them sleep than recovered BPD patients, even after adjustment for other demographic and psychiatric comorbidities (Plante et al., 2013). When taken in the context of other investigations that have demonstrated BPD outpatients are prescribed more psychotropic medications relative to patients with other psychiatric disorders (Sansone et al., 2003), it is conceivable that

medications to treat insomnia may be used in excess in patients with BPD. The results of this study suggest that patient factors, specifically beliefs related to the necessity of soporific medications to treat insomnia, may be at least partially responsible for higher rates of sedative-hypnotic use in BPD patients. However, the use of a sleeping pill by a patient requires that the medication be prescribed by a treating clinician, and thus, higher rates of sedative hypnotic use among BPD patients may also be due to factors related to the treating clinician, such as absence of knowledge regarding the efficacy of non-pharmacological treatments for insomnia, which were not ascertained by this study. Given the potential for sedation, tolerance/dependence, and other side effects from sleeping medications, future research that examines both patient and provider-based factors that may contribute to increased use of sedative-hypnotics in BPD patients is warranted.

Limitations of this study are primarily related to the study design, which utilized cross-sectional data and regression analyses to control for potential confounders. The inclusion of self-reported sleep disorders that were not confirmed with clinical evaluation and/or polysomnography (obstructive sleep apnea and restless legs syndrome) 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 excluded from regression models (data not presented). Conversely, there may have been important covariates that were not included in the adjusted analyses that may have affected findings, however, controlling for comorbid mood, anxiety, and primary sleep disorders, which collectively are the most common disorders comorbid with insomnia, minimizes this risk (Buysse et al., 1994a; Buysse et al., 1994b). Also, this study was only able to assess maladaptive beliefs surrounding the use of sleeping medications, and was not able to determine the types and frequency of sedative-hypnotic use among MSAD participants. Finally, although the data from this investigation are taken from a longitudinal study, they are cross-sectional, and we can not determine whether maladaptive sleep-related cognitions predict recovery or loss of recovery from BPD, only infer that such maladaptive beliefs may be related to the course of the disorder based on observed differences between recovered and non-recovered participants. It is noteworthy that although a significantly larger percentage of non-recovered BPD patients had clinically significant levels of maladaptive sleep-related cognitions, the majority of both groups had overall DBAS-16 scores greater than 3.8, suggesting that resolution of clinically significant dysfunctional beliefs and attitudes are not required for recovery from BPD, and that interventions to target these maladaptive cognitions could potentially be of utility in BPD patients who suffer from insomnia, even if they have recovered from the disorder.

In summary, our results demonstrate non-recovered BPD subjects have significantly worse maladaptive cognitions about sleep than those who have attained symptomatic and psychosocial recovery. 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 dysfunctional beliefs and attitudes about sleep in the longitudinal course of BPD are warranted. Specifically, prospective research that explores the efficacy of CBT-I on insomnia symptoms in BPD, as well as the effects of such therapies on attainment of recovery, may provide further insights into the role of sleep-related cognitions in the course of BPD and lead to improved treatment strategies for these patients.

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Table 1

Demographic and Clinical Characteristics of Recovered vs. Non-recovered Borderline Patients

	Recovered	Non-Recovered	p-value
Total N	105	118	---
Demographics			
Mean Age±std dev	41.17±5.4	44.21±6.0	<0.0001
Women N (%)	87 (82.9%)	94 (79.7%)	0.54
Current Axis I Disorders N (%)			
Major Depressive Episode	3 (2.9%)	37 (31.4%)	<0.0001
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
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 Borderline Patients total and component DBAS scores

	Unadjusted						Adjusted*			
	Recovered		Non-Recovered				Recovered	Non-Recovered		
	Mean	Std Dev	Mean	Std Dev	t-ratio	p-value	Adjusted means	Adjusted means	t-ratio	p-value
Overall DBAS	3.95	1.58	5.25	1.80	−5.72	<0.0001	4.80	5.66	−3.35	0.0009
Consequence	4.38	1.92	5.72	2.22	−4.76	<0.0001	5.32	6.12	−2.56	0.011
Worry/Helplessness	3.31	1.86	4.66	2.31	−4.74	<0.0001	4.54	5.29	−2.37	0.019
Expectations	5.97	2.54	6.49	2.48	−1.56	0.12	5.91	6.58	−1.75	0.081
Medication	3.13	2.37	4.82	2.40	−5.28	<0.0001	3.74	5.01	−3.55	0.0005

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