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Sleep Quality Improvement During Cognitive Behavioral Therapy for Anxiety Disorders

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Abstract

Despite the ubiquity of sleep complaints among individuals with anxiety disorders, few prior studies have examined whether sleep quality improves during anxiety treatment. The current study examined pre- to post-treatment sleep quality improvement during cognitive behavioral therapy (CBT) for panic disorder (PD; n = 26) or generalized anxiety disorder (GAD; n = 24). Among sleep quality indices, only global sleep quality and sleep latency improved significantly (but modestly) during CBT. Sleep quality improvement was greater for treatment responders, but did not vary by diagnosis. Additionally, poor baseline sleep quality was independently associated with worse anxiety treatment outcome, as measured by higher intolerance of uncertainty. Additional intervention targeting sleep prior to or during CBT for anxiety may be beneficial for poor sleepers.

Key terms

insomnia; sleep; cognitive behavioral therapy; anxiety disorders

Anxiety disorders, the most common group of psychiatric disorders in the United States (Kessler, Berglund, Demler, Jin, & Walters, 2005), are often accompanied by sleep complaints, which can precede, co-occur, or follow the onset of anxiety disorders (Jansson-Fröjmark & Lindblom, 2008; Johnson, Roth, & Breslau, 2006; Ohayon & Roth, 2003).

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There are several key findings which underscore the importance of the role of sleep within anxiety disorders: 1) Among individuals with anxiety disorders, the presence of sleep difficulties independently impacts functioning over and beyond anxiety disorders alone (Ramsawh, Stein, Belik, Jacobi, & Sareen, 2009; Soehner & Harvey, 2012); 2) sleep complaints are associated with impairment in domains including cognition, social functioning, and performance of daily activities (Soehner & Harvey, 2012); and 3) sleep may be critical for fear extinction learning, memory consolidation, and other learning processes implicated in exposure-based treatments (Kleim et al., 2013; Pace-Schott, Verga, Bennett, & Spencer, 2012).

However, few prior studies have examined whether sleep improves during the course of cognitive behavioral therapy (CBT) for anxiety disorders (Bush et al., 2012; Bélanger, Morin, Langlois, & Ladouceur, 2004; Cervena, Matousek, Prasko, Brunovsky, & Paskova, 2005; Gosselin, Ladouceur, Morin, Dugas, & Baillargeon, 2006). One recent meta-analysis found that of more than 1,200 studies on CBT for anxiety disorders, only 19 studies reported original and interpretable sleep data--based on these limited data, the authors concluded that the overall impact of CBT for anxiety on concomitant sleep problems is currently unclear (Belleville, Cousineau, Levrier, St-Pierre-Delorme, & Marchand, 2010). Of studies reporting sleep data, only a few have examined sleep symptoms using validated measures (Bush et al., 2012; Bélanger et al., 2004; Gosselin et al., 2006). Further, while sleep complaints associated with generalized anxiety disorder (GAD) have been addressed in prior work (Bush et al., 2012; Bélanger et al., 2004; Gosselin et al., 2006), to our knowledge, only one prior CBT study has examined sleep in patients with panic disorder (PD) (Cervena et al., 2005). As at least two-thirds of individuals with PD report moderate to severe sleep difficulties, including difficulty initiating and maintaining sleep, early morning wakenings, nonrestorative sleep, and nocturnal panic attacks (Mellman & Uhde, 1989; Overbeek, van Diest, Schruers, Kruizinga, & Griez, 2005; Stein, Chartier, & Walker, 1993); it would be important to further investigate whether sleep disturbances improve during the course of PD treatment. The primary purpose of the present study was to investigate whether or not sleep quality improves in individuals who have received a course of cognitive behavioral therapy (CBT) for either GAD or PD using a validated measure of sleep quality.

In order to identify possible predictors of improvement in sleep quality, and to examine whether poor sleep quality itself heralds poor anxiety treatment response, there were three exploratory aims of the current investigation. First, we examined whether treatment responders to CBT for anxiety experienced greater improvement in sleep symptoms. To our knowledge this has not been explored in prior anxiety research, although in the depression literature, participants who were considered partial or non-responders to CBT also had less improvement in sleep (Thase, Reynolds, Frank, & Jennings, 1994). Second, the available data suggest that certain anxiety disorders are more closely associated with sleep problems than others, with GAD emerging as the anxiety diagnosis other than posttraumatic stress disorder that may be most closely associated with sleep disturbance (Marcks, Weisberg, Edelen, & Keller, 2010; Monti & Monti, 2000; Ramsawh et al., 2009). However, as mentioned above, sleep disturbances have also been reported among individuals with PD (Mellman & Uhde, 1989; Overbeek et al., 2005; Stein et al., 1993). We are unaware of prior research that has specifically examined whether sleep improvements vary by diagnosis

during treatment for anxiety. Third, we examined whether poor sleep quality at baseline is associated with treatment outcome in CBT for anxiety. Although some investigators have found a relationship between poor or disturbed sleep at baseline and worse treatment outcome for depression (Buysse et al., 2001; Buysse et al., 1999; Thase, Simons, & Reynolds III, 1996; Troxel et al., 2012), we are aware of only one such study in the anxiety disorders literature, in which Zalta and colleagues (2013) found that sleep quality was a predictor of treatment response to CBT among individuals with social anxiety disorder. Therefore, secondary objectives of the current study were to examine 1) whether sleep quality at baseline predicts anxiety treatment outcome, and 2) whether improvement in sleep quality during CBT for anxiety varies by treatment response or 3) by anxiety diagnosis.

Methods and Materials

Participants

Participants were recruited from primary care and mental health outpatient clinics, and via online and community advertisement, to take part in a larger study evaluating brain changes related to CBT for anxiety disorders. After providing written informed consent, 107 participants were screened with a semi-structured diagnostic interview, the Mini-International Neuropsychiatric Interview (Sheehan et al., 1998). Interviews were conducted by an experienced, doctoral-level clinical psychologist, or by master's level clinicians who were supervised by clinical psychologists. Among those screened, 61 were eligible and met DSM-IV criteria (American Psychiatric Association, 2000) for a primary diagnosis of either PD (n=28, including 26 with agoraphobia), or GAD (n=33). Exclusion criteria included lifetime psychosis, past-year substance dependence, or past-month substance abuse. Other anxiety disorders were permitted, as was co-occurring PD and GAD, although only one disorder was selected as the primary focus of treatment (i.e., primary diagnosis). Of participants with a primary diagnosis of GAD, 4 (12%) had comorbid PD. Of participants for whom PD was the primary diagnosis, 4 (15%) had secondary GAD. Fifty participants who completed 8 or more sessions of the 10-session CBT protocol (n = 26 with GAD, n = 24 with PDA) were considered 'treatment completers' for some study analyses; of these 50 participants, 47 completed all 10 sessions. All participants were required to complete functional magnetic resonance imaging (fMRI) sessions prior to and following CBT as part of the aims of the larger study. Inclusion criteria required all participants to be free of psychotropic medications for at least six weeks prior to study enrollment (only two weeks were required for benzodiazepines, given the often more infrequent usage of benzodiazepines for control of anxiety symptoms on an "as needed" basis). All participants met safety and eligibility criteria for fMRI scanning, including no current neurological conditions, unstable medical conditions, pregnancy, or implanted ferrous metal. Data from 39 of the participants were included in a previous report that did not include treatment data (Ball, Ramsawh, Campbell-Sills, Paulus, & Stein, 2013). This study was approved by the University of California San Diego Human Research Protections Program.

Procedure

Eligible participants were screened with a medical examination consisting of a medical history and physical, clinical laboratory evaluation, EKG, and drug and pregnancy screen.

Participants also completed a battery of self-report measures. In a subsequent testing session, all participants completed an fMRI scan. Following this, they were assigned to receive 10 sessions of weekly individual CBT for treatment of their primary anxiety disorder, either with a clinical psychologist or a master's level clinician. All study clinicians received weekly supervision by licensed clinical psychologists with CBT expertise. The primary focus of the CBT protocol, which has been described elsewhere (Craske et al., 2009), was on anxiety symptomatology; sleep complaints were not specifically addressed in the protocol. Briefly, the treatment protocol consisted of computer-assisted, interactive modules designed to address the primary anxiety disorder. While some modules are relatively generic across anxiety disorders (e.g., the psycheducation module), other modules are tailored to the primary diagnosis through branching mechanisms. The modules address standard CBT concepts including self-monitoring, cognitive restructuring, and exposure to feared internal and external stimuli. The computerized program allows the clinician to enter and store anxiety examples that are specific to each patient, thus allowing individualized data to be tracked and accessed in subsequent sessions. Initial results using this treatment protocol suggest high levels of patient engagement, comprehension of treatment concepts, and high session attendance (Craske et al., 2009).

Self-report measures

Sleep quality—The well-validated Pittsburgh Sleep Quality Index (PSQI) (Buysse, Reynolds III, Monk, Berman, & Kupfer, 1989) was administered at baseline and at end of treatment in the current study. This self-report measure yields a global score as well as seven component scores measuring: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbance, sleep medications, and daytime dysfunction due to sleepiness. Both PSQI global and component scores were included in the primary analyses. Participants with global scores > 5 were considered poor sleepers, per recommended guidelines (Buysse et al., 1989). For descriptive purposes, additional PSQI data were reported as follows: total sleep time in hours (TST), time in bed in hours (TIB), sleep efficiency calculated as TST divided by TIB (SE%), and sleep latency in minutes (SLmin).

Treatment response—The Overall Anxiety Severity and Impairment Scale (OASIS) (Norman, Hami Cissell, Means-Christensen, & Stein, 2006) was administered at baseline and every two weeks during treatment. It is a 5-item measure of anxiety severity and impairment with good psychometric properties, including excellent 1-month test-retest reliability and evidence of convergent and divergent validity (Norman et al., 2006). The OASIS was chosen as the primary measure of treatment response in the current study because it can be used across anxiety disorders. Participants with at least a 50% reduction from baseline to end of treatment on the OASIS were deemed to have had a "clinically meaningful response" to treatment and were considered treatment responders.

Other clinical measures—Participants were administered additional self-report clinical measures as part of the study battery: 1) an abbreviated version of the well-validated Penn State Worry Questionnaire (PSWQ) (Hopko et al., 2003; Meyer, Miller, Metzger, & Borkovec, 1990), a measure of the core "worry" construct in GAD, 2) the Anxiety Sensitivity Index (ASI) (Reiss, Peterson, Gursky, & McNally, 1986), an established index of

anxiety sensitivity ("fear of fear") and a core construct in the psychological conceptualization of PD, 3) the Intolerance of Uncertainty Scale (IUS) (Buhr & Dugas, 2002), a validated index of intolerance of uncertainty, often elevated in anxiety disorders and a core psychological construct for GAD, and 4) the Quick Inventory of Depressive Symptomology (QIDS) (Rush et al., 2003), a reliable measure of depressive symptoms. Although the constructs assessed by these measures were not the focus of the current inquiry, baseline data are presented for descriptive purposes, and select data are presented as part of a set of secondary, exploratory analyses (See Statistical analysis section below).

Statistical analysis

Bivariate tests (chi-squares, independent sample t-tests, or the appropriate nonparametric alternative) were used to examine demographic and clinical differences by primary diagnosis, treatment completion, and treatment response. To answer the current study's primary aim—whether sleep quality improves by the end of a course of CBT treatment for anxiety—a general linear model (GLM, SPSS Version 20) approach to repeated measures analysis of covariance (ANCOVA) was utilized. These analyses were limited to treatment completers, for whom end of treatment PSQI scores were available (n = 50). Alpha level for statistical significance was set at $p \leq .05$ for all analyses. Partial eta squared, η_p^2, an estimation of effect size appropriate for analysis of variance procedures (Cohen, 1988), is also reported.

Several secondary, exploratory aims were also examined. To address the first and second exploratory aims—whether sleep quality improvement varies by treatment response or diagnosis, respectively—treatment response and primary diagnosis were entered as covariates in GLM ANCOVA models. To address the third exploratory aim—whether poor sleep at baseline (i.e., PSQI global score) was associated with treatment outcome—linear regression analyses with adjustments for demographics (age, years of education) and primary diagnosis (GAD or PD) were carried out. End of treatment measures relevant to overall anxiety impairment (OASIS) as well as disorder-specific constructs (PSWQ, IUS, and ASI Total scores) were examined. For participants who were not treatment completers, but completed at least 4 sessions of CBT (n = 9), a last-observation-carried-forward-approach was used, bringing total sample size for regression analyses to n = 59.

Results

Table 1 shows baseline demographic and clinical characteristics of the sample. At baseline, 35 (57.4%) participants were poor sleepers on the PSQI, with GAD participants having worse overall sleep quality than PD participants. Treatment completers (n = 50, 82% of the total sample) did not differ significantly from non-completers on demographic or clinical characteristics. Among participants who completed treatment, approximately half (n = 24, 48%) were treatment responders. At baseline, treatment responders did not differ significantly from non-responders on any demographic or clinical variables with the exception of frequency of taking sleep aid medications, Mann-Whitney $U = 356.5$, $p = .031$. However, as very few participants reported taking sleep medications (n = 0 for treatment

responders and n = 6 for treatment non-responders at baseline), this component had a markedly skewed distribution, and this variable was not examined further.

Primary analyses: Main effects of time

Table 2 shows change in PSQI global and component scores from baseline to end of treatment. Significant improvements in PSQI global score [$F(1, 36) = 4.122$, $p = .05$] and sleep latency component score [$F(1, 41) = 6.637$, $p = .014$] were observed, yielding medium to large effect sizes. All other baseline to end-of-treatment changes on PSQI indices were nonsignificant.

Secondary analyses

Time x responder status—First, we examined whether improvement over time in sleep quality varied by treatment response (Table 2). Change in sleep latency component score [$F(1, 41) = 12.623$, $p = .001$] significantly varied by responder status, with a large effect size observed. Specifically, post-hoc analyses stratified by responder status showed that treatment responders had significant improvement in their sleep latency component score from pre-to post treatment [$F(1, 20) = 14.439$, $p = .001$], whereas treatment non-responders did not show such improvements [$F(1, 21) = 0.098$, $p = .757$].

Time x diagnosis—Next, we examined whether improvement over time in sleep quality varied by diagnosis (Table 2). There were no statistically significant results at $p \leq .05$.

Association between baseline sleep quality and treatment response—Finally, a multivariate linear regression approach was used to examine the independent association between poor baseline sleep quality (PSQI global score) and treatment response. When examining treatment response as measured by end of treatment overall anxiety impairment (OASIS score), the relationship failed to reach significance in a multivariate model adjusted for demographics and primary diagnosis, B = 0.251, SE B = 0.143, 95% CI B = −0.037–0.539, $\beta = 0.236$, $t(51) = 1.753$, $p = .086$. However, there was a significant independent association between PSQI global score and IUS, B = 2.152, SE B = 0.873, 95% CI B = 0.399–3.906, $\beta = 0.315$, $t(50) = 2.465$, $p = .017$. Results are not shown for PSWQ and ASI, as the omnibus models were nonsignificant.

Discussion

Sleep disturbance is a common concern in individuals with anxiety disorders. Three-quarters of individuals with GAD (Anderson, Noyes, & Crowe, 1984; Bélanger et al., 2004) and two-thirds with PD report moderate to severe sleep difficulties (Overbeek et al., 2005; Stein et al., 1993). Few studies have investigated sleep quality in patients being treated with CBT for anxiety disorders. In the current study, poor sleep, as measured by a widely validated self-report instrument of sleep quality (Buysse et al., 1989), was endorsed by most participants at baseline, and baseline sleep quality was worse for those with GAD relative to PD. Overall, sleep quality improved modestly during treatment for anxiety. Among those that did respond to anxiety treatment, they also had improvements in sleep latency (i.e., they took less time to fall asleep), whereas non-responders showed no such improvements in sleep latency.

Further, improvement in sleep quality did not vary by diagnosis, at least in this relatively small sample and for these disorders. Finally, controlling for demographics and primary diagnosis, baseline sleep disturbance was independently associated with worse treatment response. In particular, those with poorer sleep at baseline had higher end-of-treatment scores on a measure of intolerance of uncertainty.

Sleep quality improved significantly during CBT from baseline to end of treatment, as has been reported in prior literature (Bélanger et al., 2004; Gosselin et al., 2006), with medium to large effect sizes observed. This can be viewed in two ways. First, it is encouraging that individuals receiving CBT for anxiety disorders also made gains in sleep quality, even without any explicit focus on sleep in the treatment protocol. In particular, global sleep quality improved, and sleep latency component score decreased. One interpretation of these data might be that when clinicians focus on the primary anxiety diagnosis, improvement in sleep tends to follow. However, significant improvements did not extend to other sleep quality domains (e.g., subjective sleep quality, sleep duration, sleep efficiency, daytime dysfunction). The current results are similar to those of another recent study in which the investigators found that PSQI global sleep quality, sleep latency, and subjective sleep quality improved over time, but not other PSQI sleep quality indices, in individuals receiving CBT for GAD (Bush et al., 2012). Furthermore, the mean global sleep quality score at Session 10 of the current study was 5.8, still above the cutoff of > 5 for poor sleep quality (Buysse et al., 1989). These data indicate that significant areas of sleep disturbance remain at the end of treatment. Indeed, other CBT studies of anxiety disorders have also found residual sleep complaints following treatment (Cervena et al., 2005; Galovski, Monson, Bruce, & Resick, 2009; Zayfert & DeViva, 2004). As residual sleep symptoms can put anxiety-prone individuals at risk for relapse (Ohayon & Roth, 2003), such patients may benefit from further intervention targeting sleep.

Sleep quality was found to vary by treatment response, with a large effect size observed, such that treatment responders had improvements in sleep latency, whereas non-responders had essentially no change in sleep latency during CBT for anxiety. Therefore, the current data suggest that for those individuals who do respond to anxiety treatment, they also tend to have less difficulty falling asleep by the end of treatment. We could find no previous studies in the existing literature that have examined whether sleep varies by anxiety treatment response.

Given the close association between GAD symptoms and self-reported sleep complaints (Marcks et al., 2010; Monti & Monti, 2000; Ramsawh et al., 2009), we examined whether individuals with GAD would experience differential improvements in sleep relative to participants with PD over the course of CBT for anxiety. However, sleep improvement did not significantly vary by diagnosis on any measure of sleep quality. This finding should be reexamined in future investigations with larger sample sizes, as the current study may have been underpowered for detecting such subgroup effects. Future studies should also include additional anxiety disorders, as well as PTSD, given the well-known associations between PTSD and sleep disturbance (Germain, 2013; Ross, Ball, Sullivan, & Caroff, 1989).

In the current study, baseline sleep difficulties were not independently associated with overall treatment response to CBT, but were associated with higher end-of-treatment intolerance of uncertainty. Intolerance of uncertainty, characterized by elevated distress and avoidance in the face of ambiguous future events, is a construct most closely associated with GAD but also elevated across anxiety disorders (Carleton et al., 2012). We are unaware of prior research demonstrating a link between subjective sleep disturbance and intolerance of uncertainty, and so the current finding, while intriguing, awaits further investigation. Analogous studies of depression have also found an association between poor sleep at baseline and worse treatment response (Buysse et al., 2001; Buysse et al., 1999; Thase et al., 1996; Troxel et al., 2012). In the one prior study we are aware of that has examined this association in anxiety disorders, participants with social phobia and poor sleep at baseline had greater social anxiety symptom severity at the end of treatment relative to those without poor baseline sleep (Zalta et al., 2013). The current study extends the prior findings limited to social phobia by examining sleep quality and treatment response in individuals with GAD and PD. Taken together with the findings of Zalta and colleagues (2013), these data suggest that poor sleep may be a general prognostic indicator among individuals with anxiety disorders. As such, there may be treatment implications. Individuals with anxiety disorders and poor self-reported sleep quality at treatment baseline may benefit from additional measures that target sleep in order to achieve the maximum benefit possible from anxiety treatment, such as CBT for insomnia (CBT-I) prior to CBT for anxiety, or augmentation of CBT for anxiety to address sleep difficulties concurrently.

There are several significant limitations that should be mentioned. First, there were no corrections for multiple comparisons in the current, exploratory study. Second, generalizability may be limited, as the sample consisted of participants in a larger fMRI study, and primary analyses concerning sleep quality improvement were limited to treatment completers. However, treatment completers did not differ significantly from non-completers on available demographic and clinical variables. Third, the current sample size is relatively modest, which may have led to inflated Type II error, particularly with regard to subgroups analysis (e.g., diagnostic subgroups). Fourth, as there was no control group, it is not possible to conclude that sleep indices improved as a direct result of the anxiety intervention. Finally, the current investigation was limited by its use of a single, subjective sleep quality measure. Future investigations of sleep disturbance in individuals with anxiety disorders should include objective sleep measures (e.g., polysomnography, actigraphy) as part of a more comprehensive battery than what was possible in the current study.

Conclusions

In conclusion, most individuals receiving CBT for anxiety disorders have poor self-reported sleep quality at baseline, which in turn is associated with poorer response to anxiety treatment. Only global sleep quality and sleep latency showed some improvement during treatment in the current sample. In addition, improvement in sleep latency was observed among anxiety treatment responders, but not among non-responders. If the current results are replicated, further investigation should determine the possible mechanism(s) driving improvements in sleep during CBT for anxiety, such as decreased worry in GAD, or decreased cognitive or physical arousal prior to falling asleep in PD. Future research should

also include larger samples and both subjective and objective measures of sleep disturbance and quality. As only a few sleep quality indices showed improvements, augmentation of CBT for anxiety—or interventions that specifically target sleep, such as CBT-I prior to or following anxiety treatment—could possibly lead to a more complete resolution of both sleep and anxiety symptoms in individuals with anxiety disorders.

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Table 1

Baseline demographic and clinical characteristics of the sample.

Variable	GAD (n = 33)	PD (n = 28)	p-value
Age (M, SD)	34.1 (10.4)	29.5 (7.5)	0.052
Gender (% female)	23 (69.7)	22 (78.6)	0.432
Non-Hispanic White (%)	26 (78.8)	17 (60.7)	0.123
Years of education (M, SD)	15.6 (2.2)	14.7 (1.7)	0.084
Employment			
Employed full or part-time (%)	28 (84.8)	17 (60.8)	**0.033**
Unemployed (%)	2 (6.1)	3 (10.7)	
Homemaker/student/retired	3 (9.1)	8 (28.6)	0.092
PSQI			
Global	7.7 (3.6)	5.6 (3.1)	**0.027**
Component 1 (subjective sleep quality)	1.6 (0.8)	1.2 (0.6)	0.075
Component 2 (sleep latency)	1.8 (1.2)	1.3 (1.1)	0.153
Component 3 (sleep duration)	0.7 (0.7)	0.5 (0.8)	0.472
Component 4 (habitual sleep efficiency)	0.5 (0.9)	0.3 (0.7)	0.269
Component 5 (sleep disturbance)	1.5 (0.6)	1.2 (0.6)	0.065
Component 6 (sleep medications)	0.4 (0.9)	0.0 (0.2)	0.057
Component 7 (daytime dysfunction)	1.4 (0.7)	1.1 (0.8)	0.147
SLmin	30 (17.5–52.5)	23 (15–60)	0.45
TST	6.9 (1.2)	7.3 (1.3)	0.222
TIB	7.9 (1.1)	7.9 (1.7)	0.856
SE%	92.3 (79.9–95.5)	94.7 (86.7–100)	0.069
PSQI poor sleepers (%, global score > 5)	23 (69.7)	12 (42.9)	0.094
OASIS responders (%)	11 (33.3)	13 (46.4)	0.266

GAD = generalized anxiety disorder; PD = panic disorder with or without agoraphobia; OASIS = Overall Anxiety Severity and Impairment Scale; PSQI = Pittsburgh Sleep Quality Index

Owing to skewness, medians and interquartile ranges are displayed for selected data.

P-values ≤ .05 are bolded.

Table 2

Repeated measure analysis of covariance results for sleep quality from baseline to end of treatment.

Variable	Baseline	Session 10	Time			Time x Diagnosis			Time x Responder status		
			f	p-value	η_p^2	f	p-value	η_p^2	f	p-value	η_p^2
PSQI											
Global	6.9 (3.7)	5.8 (3.8)	4.122	**.050**	0.103	0.873	0.356	0.024	2.328	0.136	0.061
Component 1 (subjective sleep quality)	1.4 (0.7)	1.1 (0.7)	3.174	0.082	0.072	2.598	0.115	0.06	0.871	0.356	0.021
Component 2 (sleep latency)	1.5 (1.1)	1.2 (1.2)	6.637	**0.014**	0.139	2.851	0.099	0.065	12.623	**0.001**	0.235
Component 3 (sleep duration)	0.5 (0.7)	0.5 (0.7)	0.048	0.828	0.001	0.003	0.956	0	0.148	0.702	0.004
Component 4 (habitual sleep efficiency)	0.4 (0.7)	0.3 (0.8)	0.738	0.396	0.018	0.006	0.937	0	2.506	0.121	0.059
Component 5 (sleep disturbance)	1.3 (0.6)	1.3 (0.6)	0.001	0.981	0	0.539	0.46	0.015	0.559	0.460	0.015
Component 7 (daytime dysfunction)	1.2 (0.7)	1.0 (0.9)	2.423	0.127	0.056	0.001	0.976	0	0.783	0.381	0.019

PSQI = Pittsburgh Sleep Quality Index.

P-values ≤ .05 are bolded.