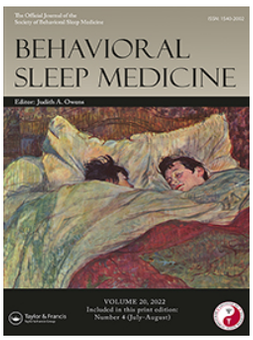

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Differences in Treatment Effects of Cognitive-behavioral Therapy for Insomnia Based on Sleep Reactivity: A Preliminary Study

Kyunga Park, Goeun Kim, Jiyun Lee, and Sooyeon Suh

Department of Psychology, Sungshin University, Seongbuk-gu, Republic of Korea

ABSTRACT

Introduction: Sleep reactivity is the trait-like degree to which stress disrupts sleep, resulting in difficulty falling and staying asleep. Although previous studies have suggested that individuals who have high sleep reactivity may be resistant to cognitive-behavioral therapy for insomnia (CBT-I) effects, there have been no studies that have investigated this empirically. This study explored differential treatment responses in CBT-I based on sleep reactivity levels.

Material and Method: Participants for this study were nineteen insomnia patients who met DSM-5 criteria for insomnia disorder. All participants received four weekly sessions of structured cognitive-behavioral therapy for insomnia (CBT-I). Individuals completed the Insomnia Severity Index (ISI), Korean version of Center for Epidemiologic Studies Depression Scale-Revised (K-CESD-R), Ford Insomnia Response to Stress Test (FIRST), Dysfunctional Beliefs and Attitudes about Sleep Scale-16 (DBAS-16), the Daily Inventory of Stressful Events (DISE) and a sleep diary. Participants were classified into two groups based on sleep reactivity level (high and low sleep reactivity).

Result: Following treatment, significant changes were found for ISI, K-CESD-R, DBAS-16 and FIRST scores, sleep onset latency, wake after sleep onset, sleep efficiency, number of awakenings, sleep quality and feeling refreshed upon awakening in both groups. Improvements in sleep efficiency was lower in the high sleep reactivity group compared to the low sleep reactivity group. No differences in ISI, K-CESD-R, DBAS-16 scores, and stress event frequency during the treatment duration were found between groups.

Conclusion: These findings suggest that sleep reactivity level may be an important factor that affects treatment outcome of CBT-I. Furthermore, the results may suggest that individual response to stress events are more important than the stressor itself.

Introduction

Insomnia is a sleep disorder that causes adverse daytime consequences, such as fatigue, low energy, hypersensitivity, and diminished cognitive function (Morin et al., 2015). Cognitive-behavioral therapy for insomnia (CBT-1) is an evidence-based treatment for insomnia that is recommended as the first line of treatment (Morin et al., 2006, p. 1999; Trauer et al., 2015; Wu et al., 2015). Numerous studies have been published to attest to its effectiveness (Morin et al., 2006; Trauer et al., 2015; Wu et al., 2015). Meta-analysis studies reveal that CBT-I is useful for a broad range of people regardless of age, comorbidity, or hypnotics (Morin et al., 2006; Van Straten et al., 2018). However, the effectiveness of CBT-I varies considerably between individuals. For example, in a recent study, 25 ~ 40% of patients reported that its therapeutic effect was insufficient (Baron & Hooker, 2017; Bastien et al., 2001; Troxel

CONTACT Sooyeon Suh alysuh@sungshin.ac.kr Department of Psychology, Sungshin University, #911 Sungshin Building 2 Bomunro 34-da Gil, Seongbuk-gu 02844, Republic of Korea

et al., 2013). This finding suggests that not all patients experience the same level of effectiveness after receiving CBT-I, which is indicative of the necessity of research for inter-individual variability in treatment response for CBT-I.

Previous studies have reported that specific factors impacting the effectiveness of CBT-I include a chronic course of insomnia with high sensitivity to the environment (Blanken et al., 2019). Sleep reactivity is the trait-like degree to which stress exposure disrupts sleep, resulting in difficulty falling and staying asleep. Individuals' sleep responses to stress are consistent over time and across a variety of stressful stimuli (Bonnet & Arand, 2003; C. C. Drake et al., 2004; Jarrin et al., 2016). Individuals with high sleep reactivity experience drastic deterioration of sleep when stressed, compared to individuals with low sleep reactivity who are able to remain unperturbed during times of stress. High levels of sleep reactivity have a higher vulnerability to sleep disturbance when they encounter a variety of stressors (C. C. Drake et al., 2004; C. L. Drake et al., 2014; Jarrin et al., 2016)

Previous studies also reported that individuals who have high sleep reactivity may be associated with characteristics that are resistant to CBT-I effects. Nakajima et al. (2014) found in a longitudinal study that high sleep reactivity contributes to the onset and maintenance of insomnia (Jarrin et al., 2014). Kalmbach et al. (2018) found high sleep reactivity was closely related to insomnia with short sleep duration (D. A. Kalmbach et al., 2018; D. A., 2016aa), although one study found that CBT-I was equally effective for insomnia patients with both objective short and normal sleep duration (Cronlein et al., 2020). Another study demonstrated that sleep reactivity was closely associated with depression and anxiety (D. A. Kalmbach et al., 2016; Nakajima et al., 2014), while other studies indicated that sleep reactivity involves cognitive and emotional responses to stress in the process of causing and sustaining insomnia (C. L. Drake et al., 2014; Fernández-Mendoza et al., 2010; D. A. Kalmbach et al., 2018). Despite these reports, no studies have directly investigated sleep reactivity as an important predictor of CBT-I. Our study hypothesized that individuals with high sleep reactivity (observed at baseline) would be predictive of lower treatment response in CBT-I compared to individuals with low sleep reactivity.

Methods

Participants

This study was conducted in adults who met the following criteria: (1) score of at least 15 on the 7-item Insomnia Severity Index (ISI); and (2) meeting Diagnostic and Statistical Manual of Mental Disorders (DSM-5) criteria for insomnia disorder (AmericanPsychiatricAssociation, 2013; Cho et al., 2014). The DSM-5 criteria for insomnia disorder indicates 3 months duration, but we used 6 months duration as the criteria in this study to recruit individuals who had a more chronic course of insomnia.

Participants were recruited through community flyers and online posts, and 128 participants responded to the ad. A total of 21 were selected through two screening procedures to meet study criteria. Participants with factors that may affect compliance with CBT-I were excluded from the study. Exclusion criteria were as follows: (1) prior exposure to CBT-I; (2) traveled to a country in a different time zone in the past 4 weeks; and (3) currently a shift worker; and (4) history of suicide attempts, bipolar disorder and schizophrenia.

A total of 19 participants were used for final analysis. The study was approved by the Ethics Committee of Sungshin Women's University.

Procedure

In this study, we adopted a quasi-experimental pre-post design and provided CBT-I to all study participants. We classified participants into two levels (high and low) of sleep reactivity groups using the baseline median score of the group. We initially intended to use the cutoff score of 18 points on the Ford Insomnia Response to Stress Test (FIRST) to distinguish groups (Kalmbach et al., 2016b).

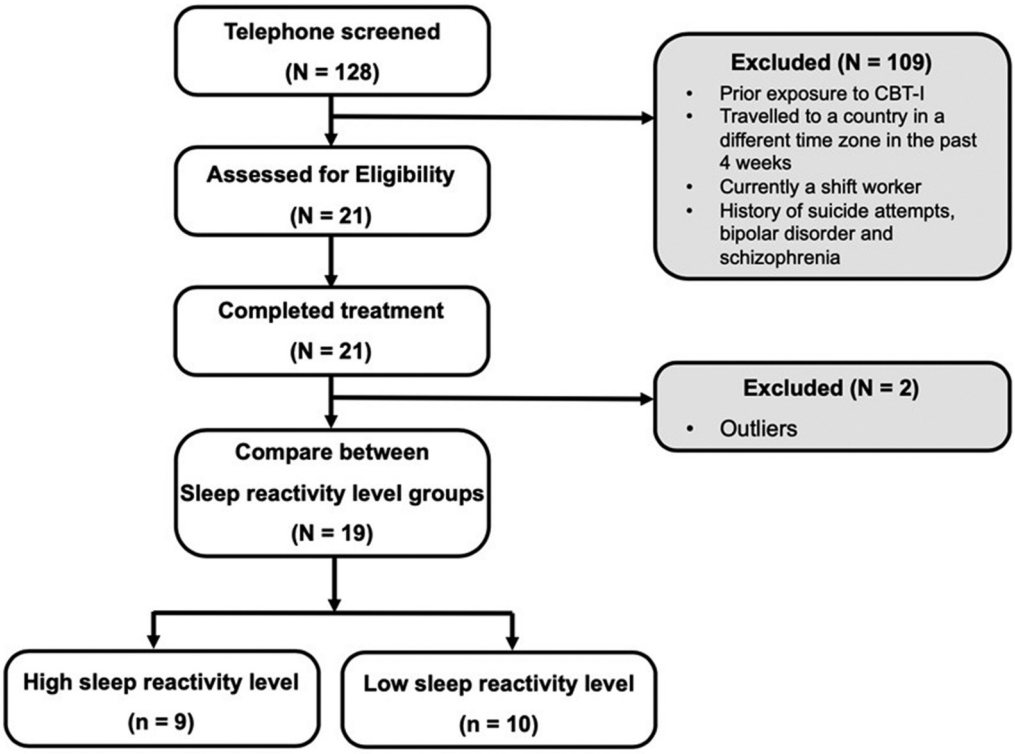

Figure 1 1 Screening procedure

However, only one participant scored below the predefined cutoff point, with potential participants volunteering for the study having overwhelmingly high sleep reactivity. Thus, for the purpose of this study, we used the median sleep reactivity of the participants (median score 28). As a result, we assigned nine out of 19 people to a group with high sleep reactivity and ten to a group with low sleep reactivity. Figure 1 illustrates the procedures for measuring data collection and therapeutic effects.

Intervention

Participants attended weekly individual CBT-I sessions (Edinger et al., 2007). Treatments consisted of four weekly sessions that were manualized to minimize therapist variables. The therapists were two graduate students trained in behavioral sleep medicine. They were supervised by a licensed clinical psychologist who was also behavioral sleep medicine certified (DBSM).

The first session was used to explain the principles of sleep and the mechanism of insomnia through sleep education (explaining the 2 process model of sleep and 3-P model of insomnia), sleep hygiene, and behavioral techniques (sleep restriction, stimulus control). Information about sleep diaries was also presented to the participant (instructions for completing sleep diaries and rationale for using them in the study). Sleep restriction consisted of setting a sleep window based on self-reported TST using the sleep diary, setting 5.5 hours as the minimum TIB. The sleep window was modified by 15 minute increments, extending TIB by 15 minutes when SE > 90% and decreasing TIB by 15 minutes when SE < 85%. The position of the sleep window was tailored to the participant's daily schedule.

The second session consisted of cognitive therapy, and exploration of the dysfunctional beliefs about sleep. Cognitive restructuring was used to replace dysfunctional beliefs with more rational and realistic thoughts. In addition, adjustment to the participant's sleep window was conducted using their previous

weekly sleep diary. The third session included demonstration and practice of relaxation therapy. Techniques such as scheduled worry time were introduced in the session. In the fourth session, participants and therapists evaluated the participant's treatment experience and discussed relapse prevention.

Measures

Sleep diary

Prior to the intervention, participants completed sleep diaries to determine their sleep patterns. One week before the first session, we conducted an online visit that included visual materials to provide instructions on keeping a sleep diary. Participants were requested to complete an online questionnaire upon waking every day. Subsequently, they were asked to keep weekly sleep diaries throughout the remainder of treatment. Sleep diary indices included Sleep Onset Latency (SOL), Number of awakenings (NWAK), Wake After Sleep Onset (WASO), Total Sleep Time (TST), Time In Bed (TIB), Sleep Efficiency (SE; ratio of TST to TIB multiplied by 100%), Sleep Quality (SQ) and Feeling refreshed upon awakening (FRESH).

Insomnia severity index (ISI)

Bastien et al. (2001) developed the Insomnia Severity Index (ISI) to measure insomnia severity (Bastien et al., 2001). The Korean version of the ISI was used and validated by Cho, Song, & Morin (Cho et al., 2014). The ISI consists of seven questions on a five-point Likert scale (0–4 points). The total score ranging from 0 to 28; higher scores are indicative of severe insomnia.

Ford insomnia response to stress test (FIRST)

Drake and colleagues (C. Drake et al., 2004) developed the Ford Insomnia Response to Stress Test (FIRST). The Korean version of FIRST was used and validated by Chang & Suh (Chang & Suh, 2018). The FIRST consists of nine items rated on a four-point Likert scale from 1 to 4. Total scores range from 9 to 36, higher scores are indicative of a greater level of sleep reactivity (Kalmbach et al., 2016b). Cronbach's alpha for the Korean version of FIRST was .85 (Chang & Suh, 2018).

Dysfunctional beliefs and attitudes about sleep scale-16 (DBAS-16)

Dysfunctional beliefs and attitudes about sleep was measured by the DBAS-16 which was developed by Morin, Vallières, and Ivers (Morin et al., 2007). The Korean version of DBAS-16 was used and validated by Yu and colleagues (Yu et al., 2009).Total scores range from 0 to 100, with higher scores indicating more dysfunctional beliefs and sleep attitudes. Cronbach's alpha for the Korean version of the DBAS-16 was .85 (Yu et al., 2009).

Korean version of center for epidemiologic studies depression scale-revised (K-CESD-R)

In the study, Korean version of Center for Epidemiologic Studies Depression Scale-Revised (K-CESD-R) was used to measure depression, which was closely related to sleep reactivity and insomnia (Lee et al., 2016; Radloff, 1977). The K-CESD-R consists of 20 items rated on a five-point Likert scale from 0 to 4. Total scores range from 0 to 80 with cut off at 13 points. Cronbach's alpha for the Korean version of K-CESD-R was .98 (Lee et al., 2016).

Daily inventory of stressful events (DISE)

Since sleep reactivity is the degree to which sleep is disturbed by stressful events, it is essential to evaluate stress and sleep patterns together. Similar to the sleep diary, we measured daily stress events through an online questionnaire. We used the Daily Inventory of Stressful Events (DISE), developed by Almeida and colleagues (Almeida et al., 2002). The DISE scale measures various aspects of everyday stressors using semi-structured telephone interviews and consists of four elements (Almeida et al., 2002). In this study, we also translated and used Stem Questions to check for stress events experienced

in various life areas. Stem Questions include seven questions to which respondents answer yes/no about stress event experiences. We calculated the average value of the total sum of the cases of "yes" responses.

Statistical analyses

During the first step, frequency analysis and descriptive statistics were used to characterize demographic information using mean and standard deviation of major variables. We used chi-square tests to verify differences between groups according to demographics. Second, we conducted paired t-tests to determine differences in all participants' sleep and psychological characteristics before and after the CBT-I intervention. Third, we calculated the median for FIRST. We classified the group exceeding the median value with a high level of sleep reactivity and the group below the median value with a low level of sleep reactivity. Also, we conducted an intergroup homogeneity test and a 2 (group) × 2 (time) repeated-measures analysis of variance (ANOVA) to compare the differences in treatment effects between the groups. Fourth, we used the Reliable Change Index (RCI) to identify people who showed a significant change level through treatment and calculated the within-group ratio to compare the high-level and low-level sleep-reactive groups' response rates. Finally, we conducted an independent sample t-test to identify the differences in the experience of stressful events during the research period, according to the sleep-reactive group. We performed all analyses with SPSS version 21.0 (IBM Corp., Armonk, NY, USA).

RESULTS

Demographic information

The mean age of participants was 29.11 (±8.50) years. The group included 15 females (78.9%) and four males (21.1%). The median of FIRST scores were used to divide participants into two groups: the high sleep reactivity group (n = 10) included members whose sleep reactivity values were on the median or higher, and the lower group's (n = 9) values were lower than the median. Information on the two groups can be found in Table 1. There were no significant differences for age and gender between groups.

Comparison of results of pre-and post- CBT-I

The comparison between pre-treatment and post-treatment as measured by self-report questionnaires showed that all values significantly decreased from pre-to post-treatment: ISI ($t = 10.29$, $p < .001$), CESD ($t = 6.04$, $p < .001$), DBAS ($t = 3.74$, $p < .01$), and FIRST ($t = 6.38$, $p < .001$) scores. All the sleep indicators from pre-to post-treatment were also significantly decreased: sleep onset latency ($t = 7.35$, $p < .001$), number of awakenings ($t = 3.31$, $p < .01$), wake after sleep onset ($t = 3.46$, $p < .01$). Sleep efficiency ($t = -9.08$, $p < .001$), sleep quality ($t = -2.95$, $p < .01$), and feeling refreshed upon awakening ($t = -5.48$, $p < .001$) increased significantly.

Comparison of treatment effects based on sleep reactivity group

Repeated measures ANOVA was conducted after identifying homogeneity between the groups to examine the differences in sleep reactivity changes from pre- to post-treatment between the higher and lower groups (Table 2 and 3). There was a significant interaction between group and time for sleep efficiency [$F(1,17) = 5.488$, $p < .05$)]. Simple main effects revealed that sleep efficiency significantly improved in both the high ($t = -4.967$, $p < .01$) and low sleep reactivity groups ($t = -9.57$, $p < .001$; Figure 2). Sleep efficiency in the low group (93.37%) after treatment was better than that of the high group (89.59%).

Considering diminished power due to a median split, additional analysis using regression with sleep efficiency as a continuous variable to predict sleep reactivity scores post-intervention after controlling for baseline intervention sleep reactivity were conducted and found to be not significant. Follow-up

Table 1. Baseline demographic information, clinical indices, and sleep parameters by group

	Total (n = 19)	Low group (n = 10)	High group (n = 9)	p-value
Gender				
Female	15 (78.9%)	7 (46.7%)	8 (53.3%)	.582[a]
Male	4 (21.1%)	3 (75%)	1 (25%)	
Age	29.11 (8.50)	27.50 (±8.89)	30.89 (±8.19)	.401
Questionnaires				
ISI	18.11 (3.68)	18.30 (2.91)	17.89 (4.57)	
CESD	20.79 (11.93)	22.10 (7.94)	19.33 (15.64)	
DBAS	5.90 (1.02)	5.86 (.94)	5.94 (1.15)	
FIRST	27.47 (5.95)	23.10 (4.58)	32.33 (2.35)	
Sleep diary				
SOL (min)	80.37 (36.65)	87.36 (45.00)	72.60 (24.80)	
N_WAK	2.35 (3.67)	2.71 (4.85)	1.94 (1.89)	
TST (min)	339.47 (68.21)	331.80 (56.86)	348.00 (81.70)	
TIB (min)	484.95 (71.02)	492.80 (76.207)	476.22 (68.20)	
WASO (min)	19.74 (17.71)	23.83 (22.33)	15.19 (10.01)	
SE (%)	70.00 (10.79)	67.06 (8.00)	73.26 (12.94)	
SQ	2.63 (0.56)	2.47 (0.48)	2.81 (0.61)	
FRESH	2.10 (0.44)	2.07 (0.52)	2.13 (0.37)	

**Abbreviations: ISI = Insomnia Severity Index; CESD = Center for Epidemiologic Studies Depression Scale; DBAS = Dysfunctional Beliefs and Attitudes about Sleep Scale; FIRST = Ford Insomnia Response to Stress Test; SOL = Sleep Onset Latency; N_WAK = Number of aWAKenings; TST = Total Sleep Time; TIB = Time In Bed; WASO = Wake After Sleep Onset; SE = Sleep Efficiency; SQ = Sleep Quality; FRESH = Feeling Refreshed upon Awakening*

[a] *Fisher's exact test*

Table 2. Clinical indices and sleep parameters of pre- and post-treatment by group.

		Low sleep reactivity group (n = 10)				High sleep reactivity group (n = 9)			
		Pre		Post		Pre		Post	
		M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Clinical indices	ISI	18.30	2.91	8.80	4.18	17.89	4.57	8.56	5.73
	CESD	22.10	7.94	7.20	4.89	19.33	15.64	8.11	7.85
	DBAS	5.86	0.94	3.67	1.79	5.94	1.15	4.53	2.17
Sleep diary parameters	Sleep onset latency	87.36	45.00	15.17	9.96	72.60	24.80	18.64	10.19
	Wake after sleep onset (frequency)	2.71	4.85	2.08	4.09	1.94	1.89	1.27	1.20
	Wake after sleep onset (minutes)	23.83	22.33	3.93	3.10	15.19	10.01	6.92	5.51
	Number of early morning awakenings	4.40	2.07	2.00	1.00	2.67	1.53	1.33	0.58
	Early morning awakenings (minutes)	81.38	33.90	49.33	40.08	59.17	11.27	21.67	7.64
	Sleep Efficiency	67.06	8.00	93.37	3.04	73.26	12.94	89.59	4.97

**Abbreviations: ISI = Insomnia Severity Index; CESD = Center for Epidemiologic Studies Depression Scale; DBAS = Dysfunctional Beliefs and Attitudes about Sleep Scale*

analyses dividing the sample into tertiles to verify the results with low (FIRST scores 0–25), middle (scores 25–30), and high (31 or higher) groups were used as a between group variable in a repeated measures ANOVA with sleep efficiency used as a within groups variable. There was a significant group by time interaction ($p < .001$) similar to the results using the median split two group analyses, with the high group showing attenuated treatment response compared to the other two groups (Table 4).

Proportion of individual changes depending on sleep reactivity

To compensate for the small sample size, we calculated the RCI developed by Jacobson and Truax (1991). We identified the levels of individual changes through CBT-I. The RCI helps determine if each participant's changes from pre- to post-treatment are statistically significant. Its merits include calculating changes without referring to control group statistics (Lambert & Ogles, 2009; Maassen,

Table 3. Comparison of treatment effects by group.

			Sum of Squares	*df*	Mean of Squares	F	*p*
Clinical indices	ISI	Group	1.018	1	1.018	.033	.857
		Time	840.066	1	840.066	99.694	.000***
		Group*Time	.066	1	.066	.008	.931
	CESD	Group	8.155	1	8.155	.056	.815
		Time	1616.141	1	1616.141	35.258	.000***
		Group*Time	32.035	1	32.035	.699	.415
	DBAS	Group	2.156	1	2.156	.793	.386
		Time	30.647	1	30.647	13.375	.002**
		Group*Time	1.433	1	1.433	.625	.440
Sleep diary parameters	Sleep onset latency	Group	301.660	1	301.660	.398	.536
		Time	37,686.364	1	37,686.364	53.474	.000***
		Group*Time	786.433	1	786.433	1.116	.306
	Wake after sleep onset (frequency)	Group	5.970	1	5.970	.256	.619
		Time	3.971	1	3.971	10.342	.005**
		Group*Time	.002	1	.002	.006	.937
	Wake after sleep onset (minutes)	Group	75.447	1	75.447	.430	.521
		Time	1879.702	1	1879.702	12.108	.003**
		Group*Time	320.463	1	320.463	2.064	.169
	Number of early morning awakenings	Group	5.400	1	5.400	2.571	.160
		Time	13.067	1	13.067	5.627	.055
		Group*Time	1.067	1	1.067	.459	.523
	Early morning awakenings (minutes)	Group	2332.603	1	2332.603	2.501	.165
		Time	4534.567	1	4534.567	4.693	.073
		Group*Time	27.870	1	27.870	.029	.871
	Sleep Efficiency	Group	13.882	1	13.882	.161	.693
		Time	4305.248	1	4305.248	100.327	.000***
		Group*Time	235.521	1	235.521	5.488	**.032***

**Abbreviations: ISI = Insomnia Severity Index; CESD = Center for Epidemiologic Studies Depression Scale; DBAS = Dysfunctional Beliefs and Attitudes about Sleep Scale*
**p < .05, **p < .01, ***p < .001*

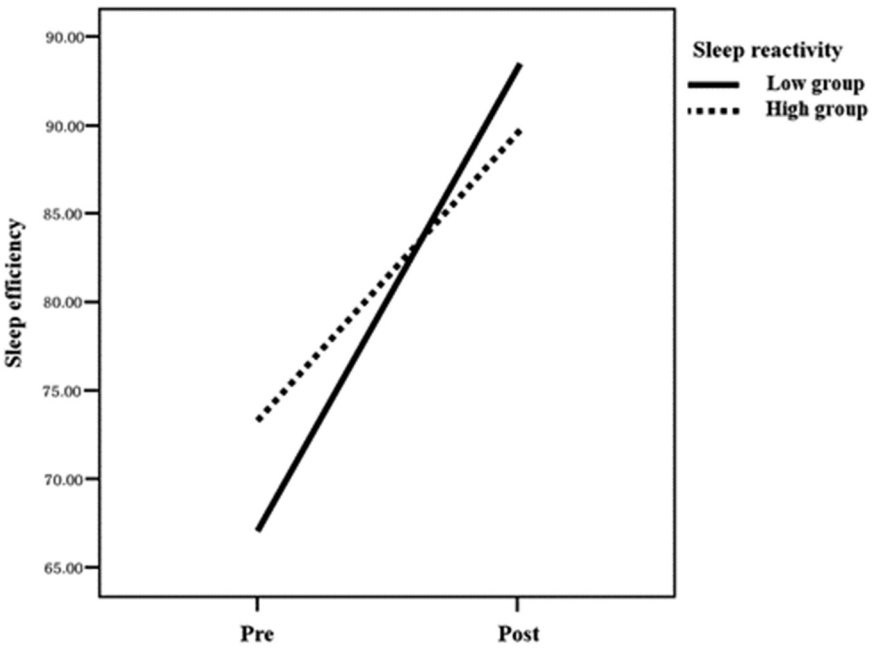

Figure 2 Changes in sleep efficiency between groups according to pre- and post-treatment

Table 4. Sleep efficiency changes post-intervention by sleep reactivity (tertiles).

Sleep reactivity group	SE at baseline	SE at intervention
Low	71.23	92.33
Medium	61.45	93.34
High	76.25	89.41

Sleep reactivity groups based on FIRST score tertiles: Low (0–25), Medium (25–30), High (31 and higher)

Table 5. Reliable Change Index between low sleep reactivity groups and high sleep reactivity groups

		Low group (n = 10)		High group (n = 9)	
		N	%	N	%
Questionnaire	ISI	9	90.0	5	55.6
	CESD	6	60.0	3	33.3
	DBAS	5	50.0	4	44.4
Sleep diary	SOL	8	80.0	8	88.9
	WASO (min)	3	30.0	0	0.0
	SE (%)	10	100.0	7	77.8

**Abbreviations: ISI = Insomnia Severity Index; CESD = Center for Epidemiologic Studies Depression Scale; DBAS = Dysfunctional Beliefs and Attitudes about Sleep Scale; SOL = Sleep Onset Latency; WASO = Wake After Sleep Onset; SE = Sleep Efficiency*

2000). As seen in Table 5, low sleep reactivity groups (vs. high sleep reactivity groups) had significantly more participants who showed reliable improvements on the ISI (90%), CESD-R (60%), DBAS (50%), SOL (80%), WASO (30%) and SE (100%).

Differences in experiencing stressful events depending on levels of sleep reactivity

Our study examined the relationship between experiencing stressful events and sleep reactivity levels to ensure that neither group had more actual stressful events during the course of the treatment. During the research period, participants were asked to record the number of daily stressful events (using DISE) along with their sleep journal. For each participant, we calculated the frequency of stressful events and the mean value. Our calculation included dividing the frequency of such incidents by the number of days the person participated in the research. There was no difference in frequency of experiencing stressful events based on levels of sleep reactivity. The analytical results are in Table 6.

Discussion

This preliminary study investigated the differences in treatment effects of CBT-I based on sleep reactivity level. Treatment successfully improved insomnia severity, depression and dysfunctional beliefs and attitudes about sleep in both low and high sleep reactivity groups. Sleep onset latency, wake after sleep onset decreased, in addition to sleep efficiency and sleep quality improving in both groups. These result supports the overall effectiveness of CBT-I as identified in numerous precedent studies despite the inter-individual variance shown in treatment effects (Morin et al., 2006; Van Straten et al., 2018).

High sleep reactivity as a predictor of treatment

The main results of our study indicated there was less improvement in sleep efficiency in response to treatment in the high sleep reactivity group compared to the low sleep reactivity group. While we did not find a significant result when using sleep reactivity as a continuous variable due to limited sample size, similar results were found using tertiles and the RCI, which demonstrated that a significant change level was smaller in the individuals with high sleep reactivity scores

Table 6. Differences in frequency of stressful events between low sleep reactivity groups and high sleep reactivity groups during treatment

	Low group (n = 10)		High group (n = 9)			
	M	SD	M	SD	t	p
Week 1	0.96	0.62	0.73	0.76	.736	.472
Week 2	0.28	0.31	0.46	0.55	−.884	.389
Week 3	0.46	0.38	0.73	0.68	−1.024	.325

compared to individuals with low sleep reactivity scores for sleep efficiency. These results indicate that changes in sleep efficiency through treatment may vary depending on the level of sleep reactivity, but should be further investigated with bigger sample sizes that are powered to detect an effect. Since increased sleep efficiency is an important treatment outcome for CBT-I, our results support the possibility that a high level of sleep reactivity moderated the treatment effects of CBT-I.

Additionally, there were no differential treatment effects between groups for insomnia severity, depression, dysfunctional beliefs and attitudes about sleep and wake after sleep onset. However, when comparing pre-and post-treatment results of all study participants, the RCI showed a diminished treatment effect in the high sleep reactivity group compared to the low sleep reactivity group for insomnia severity, depression, dysfunctional beliefs and attitudes about sleep and wake after sleep onset. Based on these results, our study confirmed that high sleep reactivity would be predictive of lower treatment response in CBT-I compared to individuals with low sleep reactivity.

High levels of sleep reactivity can impair treatment effects due to high levels of awakening. Falling asleep requires de-arousal and a reduction in the wake state, which can be disturbed by physiological, cognitive, and emotional hyperarousal that is caused by stress. Insomnia exhibits difficulty in converting from wake to sleep, or maintaining the sleep state. Kay and Buysse (2017) proposed a sleep-wake heuristic model that is biologically based on specific states and specific brain regions, consisting of three main components: sleep drive, wake drive, and conscious awareness level. Based on these three components, the authors propose that it is possible to tailor individualized treatment to the patient based on the status of each component (Kay & Buysse, 2017). Specifically, patients presenting with high sleep reactivity (and subsequently higher stress) may have greater wake drive, and may benefit from treatment components that focus on reducing arousal, such as relaxation and scheduled worry. Additional treatments such as mindfulness and/or acceptance and commitment therapy have not been directly studied with sleep reactivity, but may be promising treatment options for individuals with high sleep reactivity. Further empirical studies investigating differential characteristics associated with each of the components of the conceptual model and how they map on to individualized treatment are needed to guide treatment in clinical settings.

Sleep reactivity and the stress response

In our study, the frequency of stress events was not significantly different for each sleep reactivity group. The experience of stress can be divided into actual stressors and an individual stress response. We verified that the actual number of stressors did not differ between the low and high sleep reactivity group. However, there can be individual variability in how one responds to a stress event, such as evaluation of the stress event, individual coping styles, and emotional reactions that moderate the impact of the stressor on sleep (Fortunato & Harsh, 2006; Morin et al., 2003; Pillai et al., 2014). Future studies investigating the mechanisms of sleep reactivity by studying the link between the stressor, the individual stress response, and the impact on sleep are needed.

Sleep reactivity as a trait vs. state

Previous studies have maintained that sleep reactivity is consistent regardless of the type of stressor, time change, age, and genetic factors (Altena et al., 2017; Bonnet & Arand, 2003; C. C. Drake et al., 2004; C. L. Drake et al., 2011; Jarrin et al., 2016). Interestingly, our study demonstrated a significant reduction of sleep reactivity through CBT-I. This raises the question of whether sleep reactivity can be conceptualized as both a trait and state, such as anxiety. It is possible that while genetic predispositions may determine high trait-like sleep reactivity, this can also be modified through interventions that can help reduce state-like sleep reactivity, which subsequently might be useful in improving sleep disturbance. This finding suggests the necessity of further studies differentiating whether these two domains exist, and whether they merit attention for intervention.

Limitations

The limitations of this study and the suggestions for follow-up studies are as follows. First, this study has limitations in generalizing the effectiveness of the intervention because of the small sample size. The preliminary intervention was conducted as a quasi-experimental single group pre-post study design at an exploratory level. While the results are promising and may lay further ground for bigger clinical trials, the preliminary nature of the study should be noted and results should be interpreted with caution. Future studies with larger samples and preregistered study designs and statistical analysis plans are needed to replicate the study results.

Second, this study used an arbitrary cutoff point for differentiating high and low sleep reactivity. The groups were classified based on the median values of sleep reactivity from our sample, which reduces power to detect a difference if it exists. We emphasize that our results are preliminary and merit further investigation with larger sample sizes using sleep reactivity as a continuous variable. Our sample demonstrated that having insomnia also accompanied high sleep reactivity, therefore making it difficult to clearly define what was high and low sleep reactivity in a clinical sample. Further studies are needed to explore the level of sleep reactivity that can accurately predict low treatment response in clinical samples.

Third, there was a low RCI for WASO in our sample. The low sleep reactivity group had a difference in WASO of 19.9 minutes, and the high sleep reactivity group had a difference in WASO of 8.27 minutes (both improved). Based on a meta-analysis by Trauer et al. (2015), WASO improved on average by 26 minutes (range 15.48 ~ 36.52 minutes). We believe that the results we found are within the range of previous studies. However, because this was a 4-session structured treatment that focused heavily on behavioral components of CBT-I (stimulus control and sleep restriction), less focus on relaxation or other cognitive components could have been reflected on low RCI for WASO in our sample.

Finally, this study did not collect data using objective sleep measures. Future studies are needed to collect objective sleep data through actigraphy or polysomnography.

Conclusions

Despite these limitations, this study found differential treatment effects between groups depending on the level of sleep reactivity, despite having the same level of stressors in their daily life. This suggests that high sleep reactivity, and interventions targeted toward modifying the stress response to minimize its effect on sleep can be an important area to explore in clinical settings. These results contribute to expanding the understanding of sleep reactivity as a concept and the clinical implications it has on treatment.

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ORCID

Sooyeon Suh http://orcid.org/0000-0003-0644-8634

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