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SCIENTIFIC INVESTIGATIONS

The underestimation of sleep duration phenotype is associated with better treatment response to cognitive behavior therapy for insomnia in patients with chronic insomnia: a preliminary study

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Study Objectives: To examine treatment response to cognitive behavior therapy for insomnia (CBT-I) in patients with chronic insomnia with and without underestimation of sleep duration.

Methods: We studied 41 patients with chronic insomnia who had received 5-week CBT-I. Self-reported and objective sleep were assessed with sleep diary and actigraphy, respectively. Sleep perception was calculated as self-reported total sleep time/objective total sleep time. The underestimation of sleep duration group was defined based on sleep perception less than the median of the overall sample (85%). Insomnia Severity Index was used to assess the severity of insomnia.

Results: The total scores of Insomnia Severity Index decreased significantly after CBT-I in both groups with and without underestimation of sleep duration. Compared to pretreatment, self-reported sleep efficiency increased and total wake time decreased after CBT-I, while the magnitude of changes in sleep efficiency (d = 1.40 vs d=0.81, interaction P = .016) and total wake time (d = −1.82 vs d = −0.85, interaction P < .001) were larger in the underestimation of sleep duration group . Furthermore, self-reported sleep onset latency (interaction P = .520) and wake after sleep onset (interaction P = .052) decreased in the underestimation of sleep duration group (all P < .05), but not in patients without underestimation of sleep duration. Linear regressions showed that lower sleep perception at baseline predicted greater increase in self-reported sleep efficiency (β = −0.99, P < .001) and total sleep time (β = −0.51, P = .006) and greater decrease in self-reported total wake time (β=1.22, P = .023) after CBT-I after adjusting for confounders.

Conclusions: The current preliminary study suggests that sleep perception moderates the self-reported CBT-I effects on chronic insomnia: the phenotype of underestimation of sleep duration is associated with a better response to CBT-I, especially in self-reported sleep parameters.

Keywords: insomnia, sleep perception, underestimation of sleep duration, cognitive behavior therapy for insomnia (CBT-I)

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BRIEF SUMMARY

Current Knowledge/Study Rationale: The prevalence of underestimation of sleep duration is as high as 9.2–50% in patients with chronic insomnia. However, no study has examined the differential treatment response to cognitive behavior therapy for insomnia in patients with insomnia with and without underestimation of sleep duration.

Study Impact: Sleep perception may moderate the cognitive behavior therapy for insomnia effects on chronic insomnia: the phenotype of underestimation of sleep duration may have a better response to cognitive behavior therapy for insomnia .

INTRODUCTION

Chronic insomnia is highly prevalent, affecting 12–24% of the general population.[1–3] Sleep perception, known as the consistency between self-reported and objective sleep parameters, especially the phenotype of underestimation of sleep duration is common in patients with chronic insomnia.[4,5] The prevalence of underestimation of sleep duration has been reported to be as high as 9.2–50% in patients with chronic insomnia[4,6] and has been linked to cognitive arousal[7] and cortical arousal.[8] The clinical characteristics in patients with and without underestimation of sleep duration differ from each other, ie, patients with insomnia with underestimation of sleep duration have better objective sleep compared to patients with insomnia without underestimation of sleep duration,[4,9] suggesting specific treatment and therapeutic goals are needed for each clinical phenotype.

Cognitive-behavioral therapy for insomnia (CBT-I) is recommended as the first-line treatment for chronic insomnia across different age populations according to recent guidelines.[10,11] Vgontzas et al[12] have proposed that insomnia with objective short sleep duration may respond better to biological interventions, ie, pharmacotherapy, whereas those with normal sleep duration may have a more positive response to CBT-I.[9] This hypothesis was confirmed recently by a few studies.[13,14]

For example, Bathgate et al first reported that patients with insomnia with objective short sleep duration (ie, < 6 hours) are less responsive to CBT-I than those with normal objective sleep duration, ie, less improvement in objective sleep efficiency (SE).[13] Most recently, in an open-label preliminary study, Vgontzas et al[14] found that trazodone, but not CBT-I, increased objective sleep duration in patients with insomnia with objective short sleep duration (ie, < 6 hours). It needs to be noted that underestimation of sleep duration is prevalent among patients with insomnia with objective normal sleep duration.[4,9] Taking these together, it appears that patients with insomnia who underestimate their sleep duration may have a better response to CBT-I. However, no studies have examined the moderating effect of sleep perception on the response to CBT-I in patients with chronic insomnia in a clinical setting.

Most previous studies mainly focused on the effects of monotherapy of CBT-I on insomnia.[15,16] However, as high as 90% of patients with chronic insomnia are using hypnotics and/or sedative antidepressants (ie, trazodone) in clinical settings.[17] Based on the above, we examined the moderating effect of sleep perception on the response to CBT-I in patients with chronic insomnia who are using hypnotics and/or sedative antidepressant treatment in a clinical setting. We hypothesized that patients with chronic insomnia with underestimation of sleep duration will have a better response to CBT-I.

METHODS

Participants

Participants for this study were retrospectively selected from the sleep clinic database. Eligible patients were all those who underwent CBT-I in the Sleep Medicine Center between October 2020 to October 2021 due to chronic insomnia. Inclusion criteria were (1) age ≥ 18 years; (2) diagnosis of chronic insomnia disorder based on the *International Classification of Sleep Disorders*, third edition criteria (ICSD-3)[18]; and (3) patients with insomnia on hypnotics/sedative antidepressants when entering the CBT-I program. Exclusion criteria included (1) a major mental disorder (ie, schizophrenia, major depression, and generalized anxiety disorder); (2) substance abuse or dependence; or (3) other sleep disorders, ie, obstructive sleep apnea, narcolepsy, restless legs syndrome, and circadian rhythm disorders based on clinical symptoms and/or relevant screening questionnaires. The diagnosis of insomnia and other sleep disorders was established with a clinical sleep history and semi-structured interview conducted by a clinical physician. In this study, initially, 55 participants were selected for CBT-I. However, 4 patients discontinued (2 dropped at their first visit and 2 dropped at their third visit) due to personal reasons. Moreover, 2 were excluded because they were diagnosed with generalized anxiety disorder, 1 was excluded because she was diagnosed with major depressive disorder, and 7 were excluded because they did not receive hypnotics/sedative antidepressants when undergoing CBT-I. Finally, 41 patients who completed a 5-week CBT-I were included in this study. The study was approved by the Institutional Review Board of Shantou University Mental Health Center (SUMHC202014). All participants signed informed consent and were informed that their personal information would be confidential.

Sample size calculation was conducted based on a previous study examining the treatment response of CBT-I in insomnia patients with self-reported short or normal total sleep duration[19] in which the partial η^2 of time × group effect was 0.276 of self-reported SE and 0.161 of self-reported total sleep time (TST), respectively. Assuming 95% power, using a 2-sided hypothesis test with a significance level of .05, a total of 18 and 34 participants were required to detect a significant time × group effect of self-reported SE and TST, respectively. Thus, at least 34 patients with chronic insomnia were required in the current study. Furthermore, we also conducted power estimation to examine whether the number of participants included allowed a correct estimation of the differences regarding the changes in sleep from pre- to posttreatment between the patients with and without underestimation of sleep duration. Results from power calculation with the G*Power 3.1.9.2 program[20] showed that 93.21%, 88.70%, 78.02%, and 99.99% power to reject the null hypothesis and detect a significant time × group effect of self-reported SE, TST, wake after sleep onset (WASO), and total wake time (TWT) in the current study.

Procedures

The CBT-I protocol used in this study included sleep restriction, stimulus control, cognitive therapy, sleep hygiene, and relaxation therapy.[17] The CBT-I protocol was implemented for over 5 weeks, with a total of 5 weekly consultation/therapy sessions each lasting approximately 60 minutes. The CBT-I was conducted face to face by 1 licensed psychiatrist. In China, a licensed psychiatrist is qualified to prescribe medications and perform psychological therapy. In this study, all participants were using hypnotics and/or sedative antidepressants before the CBT-I, and the dosage of hypnotics and/or sedative antidepressants was gradually reduced by a psychiatrist according to CBT-I protocol.[21] Finally, 15 patients discontinued hypnotics and/or sedative antidepressants after CBT-I.

Measures

Clinical history and physical examination

A semistructured questionnaire and a battery of clinical tests were used for assessing each participant's medical history and physical examination. Body mass index (BMI) was calculated based on measured weight (kg)/height (m).[2] STOP questionnaire was used to evaluate the risk of obstructive sleep apnea. The total scores of STOP questionnaire higher than 2 points indicate high risk of obstructive sleep apnea.[22] Insomnia Severity Index (ISI) was used to assess the severity of self-reported insomnia symptoms. The higher total scores of ISI indicate more severe insomnia.[23] Epworth Sleepiness Scale (ESS) was used to assess daytime sleepiness. The higher total scores of ESS indicate more severe daytime sleepiness.[24] The Beck Depression Inventory was used to assess depressive symptoms.[25] The Beck Anxiety Inventory was used to assess anxiety symptoms.[26]

Sleep diary

Self-reported sleep was assessed by sleep diary. Participants were asked to complete a sleep diary right after waking up in

the morning daily during the CBT-I. The sleep diary includes self-reported TST, sleep onset latency (self-reported SOL), WASO (self-reported WASO), TWT (self-reported TWT: total time awake between initial sleep onset and the get-up time), total time in bed (self-reported TIB), and SE (self-reported SE = self-reported TST/self-reported TIB). The mean values of each sleep variable assessed by sleep diary at baseline (pretreatment, 0 week) and posttreatment (4th week) recording periods were used for analysis in this study.

Actigraphy

Objective sleep was assessed by actigraphy. Participants were asked to wear an actigraph (wGT3X-BT, ActiGraph, LLC, Suite C Pensacola, FL) on their nondominant wrists throughout the study (1-week assessment for baseline before treatment and 4 weeks assessments for the therapy period). Objective sleep, including actigraphy measured TST (objective TST), SOL (objective SOL), WASO (objective WASO), TIB (objective TIB), and SE (objective SE) was assessed by actigraphy. The mean values of each sleep variable assessed by actigraphy at pretreatment (0 week) and posttreatment (4th week) recording periods were used for analysis in this study.

Sleep perception was calculated as self-reported TST/objective TST.[27] In this study, the underestimation of sleep duration group (USDG) was defined based on sleep perception less than the median value of the overall sample (< 85%), while the patients without underestimation of sleep duration (control group) were defined based on sleep perception ≥ 85%.

Statistical analysis

Bivariate comparisons between groups were performed using independent *t*-tests or Mann–Whitney *U* tests for normally and nonnormally distributed continuous variables or using the χ^2 test for categorical variables, respectively. Data are presented as means ± standard deviation for continuous variables and percentages for categorical variables. To examine short-term changes (posttreatment vs pretreatment) in self-reported and objective sleep, we conducted 2 × 2 (time × group) analyses of repeated-measures analyses of variance after controlling for age, sex, BMI, and baseline total scores of ISI. Covariates were defined based on the differences between the USDG and the control group (**Table 1**, *P* value < .10) and clinical relevance. Comparisons between pre- and posttreatment sleep characteristics were performed using paired *t*-tests or nonparametric Wilcoxon tests for normally and nonnormally distributed continuous variables. The comparisons of the changes of sleep from pre- to posttreatment between USDG and the control group across each self-reported and objective sleep parameter were conducted using analysis of covariance after controlling for age, sex, BMI, and baseline total scores of ISI. The sleep variables with interaction $P \leq .05$ were entered into a multiple linear regression model as an outcome variable separately and with the continuous values of sleep perception as a predictor to examine the association between sleep perception and the changes in sleep after CBT-I after controlling for age, sex, BMI, and baseline total scores of ISI. Changes in sleep from pre- to posttreatment were calculated as (posttreatment values − pretreatment values)/pretreatment values × 100 (%). A $P \leq .05$ was used to determine statistical significance. Effect sizes (Cohen's *d*) were used as a further indication of the magnitude of changes. G*Power 3.1.9.2 program was used for power calculation.[20] All analyses except for power calculation were conducted using SPSS 23.0 (IBM Corp., Armonk, NY).

RESULTS

Pretreatment characteristics

A total of 41 patients with chronic insomnia with a mean age of 43.29 ± 9.88 years and 29 (70.70%) females were included

Table 1—Sociodemographic and sleep characteristics of study participants.

	All (n = 41)	Control group (n = 20)	USDG (n = 21)	*P*
Age (years)	43.29 ± 9.88	40.50 ± 9.34	45.95 ± 9.85	**.077**
Sex (Female, %)	29 (70.70)	13 (65.00)	16 (76.20)	.431
BMI, kg/m^2	21.79 ± 2.65	21.47 ± 2.58	22.09 ± 2.73	.735
Baseline medication type				.459
Benzodiazepine receptor agonists	18 (43.90)	7 (35.00)	11 (52.38)	
Nonbenzodiazepine receptor agonists	13 (31.70)	8 (40.00)	5 (23.81)	
Sedative antidepressants	10 (24.40)	5 (25.00)	5 (23.81)	
ISI score	20.76 ± 4.79	19.05 ± 5.38	22.38 ± 3.57	**.027**
STOP questionnaire score				
0 (%)	39 (95.12%)	19 (95.00%)	20 (95.24%)	.972
1 (%)	2 (4.88%)	1 (5.00%)	1 (4.76%)	
ESS score	3.95 ± 3.90	4.90 ± 4.75	3.05 ± 2.69	.274
BDI score	9.41 ± 5.73	9.40 ± 6.01	9.43 ± 5.59	.988
BAI score	31.12 ± 6.09	31.80 ± 6.06	30.48 ± 6.20	.469

All values are presented as mean ± SD. Values in bold indicate *P* values < .10. BAI = Beck Anxiety Inventory, BDI = Beck Depression Inventory, BMI = body mass index, ESS = Epworth Sleepiness Scale, PSQI = Pittsburgh Sleep Quality Index, USDG = underestimation of sleep duration group.

(Table 1). Eighteen were taking benzodiazepine receptor agonists, 13 were taking nonbenzodiazepine receptor agonists and 10 were taking sedative antidepressants at baseline. Two patients got 1 point in STOP questionnaire and the rest scored 0 points. Among the 41 patients included, 20 were categorized as control group, and 21 were USDG. Compared to the control group, total scores of ISI were significantly higher in the USDG at baseline. Age, sex, BMI, medication type, the total scores of STOP questionnaire, ESS, Beck Depression Inventory, and Beck Anxiety Inventory did not differ significantly between the 2 groups at baseline.

Self-reported sleep

As expected, the USDG presented worse self-reported sleep compared to the control group. Specifically, the USDG had lower SE (54.67 ± 11.76% vs 80.07 ± 8.58%, $P < .001$), shorter TST (303.68 ± 71.16 minutes vs 431.63 ± 52.89 minutes, $P < .001$), longer SOL (53.64 ± 26.22 minutes vs 29.42 ± 19.66 minutes, $P = .002$), longer WASO (57.71 ± 59.62 minutes vs 21.13 ± 17.13 minutes, $P = .013$), and longer TWT (165.14 ± 72.13 minutes vs 55.56 ± 29.63 minutes, $P < .001$) as measured by sleep diary at baseline (Table 2).

Objective sleep

Objective TST as measured by actigraphy was significantly longer in the USDG (475.64 ± 59.81 minutes vs 437.60 ± 53.18 minutes, $P = .038$) compared to the control group. No significant differences in other objective sleep parameters between the USDG and control group were observed (Table 2).

Treatment effects

Self-reported sleep

Among the 41 patients included, compared to pretreatment, the total scores of ISI (13.10 ± 5.28 vs 20.76 ± 4.79, $P < .001$), SOL (29.47 ± 25.30 minutes vs 41.83 ± 26.03 minutes, $P = .007$), WASO (17.56 ± 20.97 minutes vs 39.87 ± 47.53 minutes, $P < .001$), and TWT (36.12 ± 31.75 minutes vs 111.69 ± 78.06 minutes, $P < .001$) decreased significantly, and SE (81.72 ± 12.51% vs 67.06 ± 16.41%, $P < .001$) increased significantly after CBT-I. Furthermore, TST (337.80 ± 68.83 minutes vs 366.09 ± 89.74 minutes, $P = .010$) decreased significantly, and the total scores of ESS (6.27 ± 4.37 vs 3.95 ± 3.90, $P < .001$) increased significantly after CBT-I.

As shown in Table 2, the total scores of ISI decreased significantly in both control group (11.55 ± 4.06 vs 19.05 ± 5.39, $P < .001$) and USDG (14.57 ± 5.96 vs 22.38 ± 3.57, $P < .001$) after CBT-I compared to pretreatment, while the magnitude of changes in ISI between the 2 groups did not show statistical difference ($d = -1.41$ vs $d = -1.04$, time × group interaction $P = .954$) after controlling for age, sex, BMI, and baseline total scores of ISI. Furthermore, self-reported SE ($P = .016$), TST ($P = .026$), WASO ($P = .052$), and TWT ($P < .001$) showed significant time × group interactions after controlling for age, sex, BMI, and baseline total scores of ISI, indicating that CBT-I treatment effects on self-reported SE, TST, WASO, and TWT differed significantly between the control group and USDG. Compared to pretreatment, self-reported SE (USDG: 76.04 ± 14.63% vs 54.67 ± 11.76%, $P < .001$; control group: 87.68 ± 5.59% vs 80.07 ± 8.58%, $P = .002$) increased significantly, and self-reported TWT (USDG: 46.02 ± 38.38 minutes vs 165.14 ± 72.13 minutes, $P < .001$; control group: 25.73 ± 18.71 minutes vs 55.56 ± 29.63 minutes, $P = .001$) decreased significantly after CBT-I in both groups, while the magnitudes of changes in SE ($d = 1.40$ vs $d = 0.81$, time × group interaction $P = .016$) and TWT ($d = -1.82$ vs $d = -0.85$, time × group interaction $P < .001$) were greater in the USDG after controlling for age, sex, BMI, and baseline total scores of ISI. Furthermore, self-reported WASO decreased significantly in the USDG (21.87 ± 26.57 minutes vs 57.71 ± 59.62 minutes, $P = .001$, $d = -0.85$), but not in the control group (13.03 ± 11.85 minutes vs 21.13 ± 17.13 minutes, $P = .075$, $d = -0.42$). Moreover, self-reported TST decreased significantly in the control group (376.05 ± 47.88 minutes vs 431.63 ± 52.89 minutes, $P < .001$, $d = -1.24$), but not in the USDG (301.36 ± 66.63 minutes vs 303.68 ± 71.16 minutes, $P = .889$, $d = -0.03$). In addition, although without a significant time × group interaction, self-reported SOL (36.35 ± 29.21 minutes vs 53.64 ± 26.22 minutes, $P = .029$, $d = -0.51$) decreased significantly after CBT-I in the USDG, but not in the control group (22.24 ± 18.49 minutes vs 29.42 ± 19.66 minutes, $P = .116$, $d = -0.37$). The total scores of ESS (control group: 6.75 ± 4.87 vs 4.90 ± 4.75, $P = .035$; USDG: 5.81 ± 3.91 vs 3.05 ± 2.69, $P = .003$) increased significantly after CBT-I in both groups, while the magnitude between the 2 groups did not show significant difference ($d = 0.45$ vs $d = 0.79$, time × group interaction $P = .591$) after controlling for age, sex, BMI, and baseline total scores of ISI. Considering that baseline medication type (ie, benzodiazepine receptor agonists, nonbenzodiazepine receptor agonists, and sedative antidepressants) may affect the results, in sensitivity analyses, we further adjusted for baseline medication type, and findings were similar. No difference regarding the withdrawal rate of hypnotics/sedative antidepressants after CBT-I between the 2 groups was observed (control group: 40%, USDG: 33.3%, $P = .658$).

Figure 1 depicts the comparisons of changes in self-reported sleep from baseline to posttreatment. Compared to the control group, the changes of self-reported SE (40.50 ± 30.25% vs 14.40 ± 29.96%, $P = .012$) and TWT (−78.50 ± 71.00% vs −33.90 ± 64.85%, $P = .043$) from pre- to posttreatment were significantly greater in the USDG after CBT-I after controlling for age, sex, BMI, and baseline total scores of ISI. However, changes in self-reported TST, SOL, WASO, and objective sleep parameters did not differ significantly between the 2 groups. In sensitivity analyses, after further adjusting for baseline medication type, findings were similar.

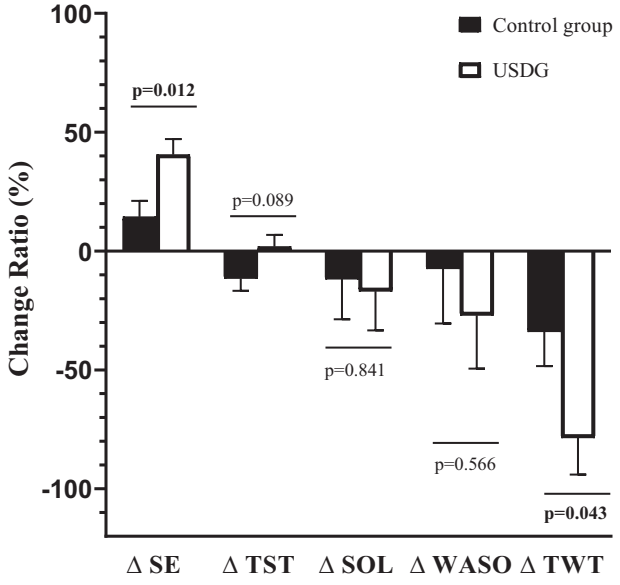
Multiple linear regressions showed that lower sleep perception at baseline was significantly associated with greater increase in self-reported SE ($\beta = -0.99$, $P < .001$) and TST ($\beta = -0.51$, $P = .006$) and greater decrease in self-reported TWT ($\beta = 1.22$, $P = .023$) after CBT-I after controlling for age, sex, BMI, and baseline total scores of ISI. However, sleep perception at baseline was not associated with changes in self-reported WASO from pre- to post-CBT-I treatment. In sensitivity analyses, after further adjusting for baseline medication type, findings were similar.

Table 2—Sleep characteristics of study participants pre- and post-CBT-I treatment.

	Overall	Control Group (n = 20)		USDG (n = 21)								
	Pretreatment	Pretreatment	Posttreatment	Pretreatment	Posttreatment	*P*	*P**	*P*†	Cohen *d*1	Cohen *d*2	Time effect# F/*P*	Time × Group Effect# F/*P*
Self-reported sleep												
SE (%)	67.06 ± 16.41	80.07 ± 8.58	87.68 ± 5.59	54.67 ± 11.76	76.04 ± 14.63	**< .001**	**.002**	**< .001**	**0.81**	**1.40**	2.18/.149	**6.41/.016**
TST (min)	366.09 ± 89.74	431.63 ± 52.89	376.05 ± 47.88	303.68 ± 71.16	301.36 ± 66.63	**< .001**	**< .001**	.889	**−1.24**	−0.03	0.56/.463	**5.41/.026**
SOL (min)	41.83 ± 26.03	29.42 ± 19.66	22.24 ± 18.49	53.64 ± 26.22	36.35 ± 29.21	**.002**	.116	**.029**	−0.37	−0.51	**4.29/.046**	0.42/.520
WASO (min)	39.87 ± 47.53	21.13 ± 17.13	13.03 ± 11.85	57.71 ± 59.62	21.87 ± 26.57	**.013**	.075	**.001**	−0.42	**−0.85**	2.80/.103	**4.03/.052**
TWT (min)	111.69 ± 78.06	55.56 ± 29.63	25.73 ± 18.71	165.14 ± 72.13	46.02 ± 38.38	**< .001**	**.001**	**< .001**	**−0.85**	**−1.82**	**4.20/.048**	**23.80/< .001**
ISI	20.76 ± 4.79	19.05 ± 5.39	11.55 ± 4.06	22.38 ± 3.57	14.57 ± 5.96	**.027**	**< .001**	**< .001**	**−1.41**	**−1.04**	0.10/.750	0.01/.954
ESS	3.95 ± 3.90	4.90 ± 4.75	6.75 ± 4.87	3.05 ± 2.69	5.81 ± 3.91	.274	**.035**	**.003**	0.45	0.79	0.24/.628	0.29/.591
Objective sleep												
SE (%)	84.21 ± 4.78	83.30 ± 3.76	83.67 ± 6.14	85.07 ± 5.54	83.98 ± 6.00	.241	.536	.498	0.06	−0.14	0.30/.585	0.73/.399
TST (min)	457.08 ± 59.18	437.60 ± 53.18	371.34 ± 50.01	475.64 ± 59.81	377.14 ± 61.89	**.038**	**< .001**	**< .001**	**−1.63**	**−1.30**	3.85/.058	2.79/.104
SOL (min)	20.35 ± 16.30	21.71 ± 17.67	18.29 ± 18.86	19.06 ± 15.20	16.92 ± 16.57	.715	.550	.709	−0.15	−0.10	1.57/.25	0.06/.812
WASO (min)	46.51 ± 15.18	48.68 ± 14.04	45.44 ± 14.87	44.45 ± 16.26	43.64 ± 16.67	.380	.296	.958	−0.18	−0.04	0.08/.781	0.13/.726

All values are presented as raw data of mean ± SD. *P* values for between-group comparisons regarding self-reported and objective sleep parameters were presented after adjusting for age, sex, BMI, and baseline total scores of ISI; *P* value for between-group comparison regarding ISI was adjusted for age, sex, and BMI. *P*, Control group vs USDG at pretreatment; *P**, pretreatment vs posttreatment in the control group; *P*†, pretreatment vs posttreatment in the USDG; Cohen's *d1*, pretreatment vs posttreatment in the control group; Cohen's *d2*, pretreatment vs posttreatment in the USDG. Values in bold indicate absolute values of Cohen's $d \geq 0.80$ or *P* values ≤ .05. ESS = Epworth Sleepiness Scale, PSQI = Pittsburgh Sleep Quality Index, SE = sleep efficiency, SOL = sleep onset latency, TST = total sleep time, WASO = wake after sleep onset, TWT = total wake time, USDG = underestimation of sleep duration group.

Figure 1—The estimated changes in self-reported sleep from baseline to posttreatment.



The data were presented after controlling for age, sex, BMI, and baseline total scores of ISI. Error bars represent standard errors. △ = ([posttreatment values minus pretreatment values]/pretreatment values), Control group = patients without underestimation of sleep duration, SE = sleep efficiency, SOL = sleep onset latency, TST = total sleep time, TWT = total wake time, USDG = underestimation of sleep duration group, WASO = wake after sleep onset.

Objective sleep

Among the 41 patients included, objective TST decreased (374.31 ± 55.78 minutes vs 457.08 ± 59.18 minutes, $P < .001$) significantly after CBT-I compared to pretreatment. No significant differences were observed in objective SE, SOL, or WASO between pre- and post-CBT-I treatment.

As shown in **Table 2**, objective TST (control group: 371.34 ± 50.01 minutes vs 437.60 ± 53.18 minutes, $P < .001$, $d = -1.63$; USDG: 377.14 ± 61.89 minutes vs 475.64 ± 59.81 minutes, $P < .001$, $d = -1.30$) decreased after CBT-I in both groups, but without a significant time × group interaction (interaction-$P = .104$) after controlling for age, sex, BMI, and the baseline total scores of ISI. No significant differences were observed regarding changes in objective SE, SOL, or WASO from baseline to posttreatment in either control group or USDG.

DISCUSSION

To our knowledge, up to now, this is the first study comparing the treatment response to CBT-I between patients with chronic insomnia using hypnotics/sedative antidepressants with and without underestimation of sleep duration in a clinical setting. Our findings suggest that patients with chronic insomnia with underestimation of sleep duration at baseline have a better response to CBT-I, especially in self-reported sleep.

Bathgate et al[13] reported that patients with insomnia with normal sleep duration had a better response to CBT-I, and, interestingly, the group of patients with insomnia with objective normal sleep duration also presented underestimated sleep duration at baseline.[13] This study supports our findings of patients with insomnia who underestimate their sleep duration at baseline appear to respond better to CBT-I. Recent studies reported that patients with objective short sleep duration (TST < 6 hours) at baseline were less likely to show sleep improvements after CBT-I treatment compared to those with relatively normal sleep duration (TST ≥ 6 hours).[13,28] Of note, those with normal sleep duration were more likely to report underestimation of sleep.[4,9] Indeed, objective TST was significantly longer in the patients with underestimation of sleep duration compared to those without underestimation of sleep duration in the current study. These findings also indirectly support our findings of insomnia with underestimation of sleep duration responding better to CBT-I. Underestimation of sleep duration in patients with insomnia is quite a challenge and its underlying mechanisms remain unclear.[29] It has been suggested that underestimation of sleep duration is associated with psychological characteristics and cortical hyperarousal as measured by EEG beta activity during sleep in patients with insomnia.[12,30,31] Cognitive therapy improves psychological distress and decreases emotional arousal and cognitive arousal.[17] The latter has been linked to cortical arousal, and might plausibly be effective in the phenotype of underestimation of sleep duration in chronic insomnia. Furthermore, based on our data, although self-reported SE increased significantly in patients with underestimation of sleep duration after CBT-I treatment, posttreatment self-reported SE did not reach the pretreatment level of the control group. This could be due to the relatively short follow-up duration. We speculate that self-reported SE would further increase after prolonged CBT-I therapy period and/or follow-up duration. Future studies with longer CBT-I therapy periods and follow-up duration may verify our hypothesis.

In this study, we found that objective TST measured by actigraphy decreased significantly in both patients with and without underestimation of sleep duration. The decrease in TST during treatment may seem counterintuitive to achieve insomnia remission, however, previous studies support this trend.[13,16] As reported by Buysse et al[16] insomnia remission was observed despite a significant reduction in actigraphy-measured TST from pre- to posttreatment in older adults with chronic insomnia who received brief behavioral treatment. Later, a similar finding in middle-aged and older adults with chronic insomnia who receive CBT-I was observed by Bathgate and colleagues.[13] We did not observe significant changes in other objective sleep characteristics (ie, SE, SOL, and WASO) after a 5-week CBT-I. These findings are in line with previous studies that CBT-I has little effect on objective sleep parameters.[32–36] Recently, a meta-analysis also reported the sleep benefits of CBT-I are more pronounced in the self-reported vs objective sleep.[37] The relatively long objective sleep duration in our study for patients with chronic insomnia could also be associated with the use of hypnotics and/or sedative antidepressants at baseline. However, despite the relatively long objective sleep duration, all included patients in our study met the diagnostic criteria for chronic insomnia based on ICSD-3.

Strengths and limitations

The strength of this study is that Beck Depression Inventory and Beck Anxiety Inventory were used to further exclude

patients with emotional disorders. However, several limitations should be acknowledged. First, the relatively small sample size of this study, although it met the limited sample size requirement based on sample size calculation. Future large multicenter clinical trials are needed. Second, we included patients who were using hypnotics/sedative antidepressive medications at baseline, thus the current findings might not be generalized to the insomnia patients who are not being treated with hypnotics/sedative antidepressive medications. However, a significant percentage of patients with chronic insomnia still report severe insomnia symptoms even after being treated with hypnotics. Thus, we believe that our study is a pragmatic study reflecting the real-world clinical practice. In addition, using hypnotics/sedative antidepressive medications may affect patients' recall/perception.[38] Most randomized control trials (RCTs) showed that self-reported sleep improved after hypnotics treatment[39–41] in mitigation. Because the severity of insomnia is mainly based on self-reported symptoms, it appears that hypnotics improve sleep perception. Future studies should be conducted in unmedicated insomnia patients. Third, we did not perform polysomnography to exclude obstructive sleep apnea and periodic limb movement disorder. However, polysomnography is not routinely recommended based on American Academy of Sleep Medicine guidelines for patients with insomnia in clinical practice[42] and we used valid questionnaires to rule out these potential patients. In mitigation, findings of STOP questionnaire showed that all included patients were at a low risk of sleep apnea. Fourth, our study could not provide evidence of the long-term effect of CBT-I in patients with chronic insomnia with and without underestimation of sleep duration. Future studies with longer follow-up duration should be conducted.

CONCLUSIONS

This preliminary study suggests that sleep perception moderates the CBT-I effects on patients with chronic insomnia who are using sleep medications: the phenotype of underestimation of sleep duration (ie, paradoxical insomnia) is associated with a better response to CBT-I, especially in self-reported sleep parameters.

ABBREVIATIONS

BMI, body mass index
CBT-I, cognitive behavior therapy for insomnia
ESS, Epworth Sleepiness Scale
SE, sleep efficiency
SOL, sleep onset latency
TIB, time in bed
TST, total sleep time
TWT, total wake time
USDG, underestimation of sleep duration group
WASO, wake after sleep onset

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