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ScienceDirect

Behavior Therapy 50 (2019) 994–1001

Behavior Therapy

www.elsevier.com/locate/bt

# Changes in Subjective-Objective Sleep Discrepancy Following Inpatient Cognitive Behavior Therapy for Insomnia

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**Discrepancy between objective and subjective sleep parameters is a frequent symptom in persons suffering from insomnia. Since it has an impairing effect on daytime well-being and neglects possible positive objective improvements, it would be useful if it was treated. Apart from hypnotics, cognitive behavior therapy (CBT-I) is the therapy of choice for chronic forms of insomnia. However, there is limited information about whether CBT-I can also improve subjective-objective sleep discrepancy. We investigated a large sample of patients showing chronic forms of insomnia regarding their subjective-objective sleep discrepancy pre and post CBT-I. Objective sleep data were obtained from 3 nights (2 baseline nights and 1 night after therapy) using polysomnography in our sleep laboratory. All 92 patients participated in a 14-day inpatient program with CBT-I including psychoeducation about subjective-objective sleep discrepancy. Repeated measures analyses showed an improvement in subjective-objective sleep discrepancy parameters after CBT-I. Those parameters were also correlated with perceived quality of sleep. We conclude that CBT-I is a useful tool to improve subjective-objective sleep discrepancy in patients showing chronic forms of insomnia.**

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*Keywords:* insomnia; Cognitive Behavioral Therapy for Insomnia; subjective-objective sleep discrepancy; polysomnography; subjective sleep; sleep state misperception

Discrepancy between sleep estimation and polysomnographic data constitutes a frequent feature of insomnia disorder (Perlis et al., 1997; Harvey & Tang, 2012; Rezaie et al., 2018). Patients with insomnia remember less sleep than measured with polysomnography (Carskadon et al., 1976; Edinger & Fins, 1995; Frankel et al., 1976; Rosa & Bonnet, 2000). Sleep state misperception or subjective-objective sleep discrepancy (SOSD) is not a generic pattern of insomnia sufferers (Means et al., 2003), but one paradoxical phenomenon of insomnia disorder (Edinger & Krystal, 2003; Perlis et al., 1997) that has not been resolved until now (Harvey & Tang, 2012). It has been postulated that it is related to the inability to perceive short episodes of sleep during the night when individuals show frequent awakenings (Knab & Engel, 1988). However, recent research indicates that current measures may not be sensitive enough to detect possible neurophysiological mechanisms (Rezaie et al., 2018). Why is SOSD worth being treated? First of all, patients underestimating objective amounts of sleep probably miss actual improvements after therapy and may develop the self-perception of being a "hopeless case," for example, due to persistent sleep-related worries (Tang & Harvey, 2004). A higher degree of accuracy in estimating their objective sleep would be helpful

in order to benefit from the positive effects of insomnia-specific therapy (Harvey & Tang, 2012). Moreover, clinical experience shows that patients who underestimate their sleep duration feel as exhausted as patients with objectively short sleep (Means et al., 2003). SOSD has an effect on daytime functioning (Semler & Harvey, 2005). Treating SOSD therefore is one important challenge to insomnia therapy.

There is limited information about therapies to improve SOSD in insomnia patients. However, it was shown that benzodiazepines improved the perception of sleep after forced awakening in insomniacs (Mendelson, 1993, 1995). Cognitive behavior therapy for insomnia (CBT-I) is the treatment of choice that is effective in improving sleep as measured by polysomnography (Edinger et al., 2001; Morin et al., 1994; Koffel et al., 2015; Riemann & Perlis, 2009). Unfortunately, there is only a limited amount of data about its effect on SOSD. Lund et al. examined patients showing comorbid insomnia regarding improvement of SOSD pre and post CBT-I and found a correction of discrepancy using home-based polysomnography. They were able to show that a reduction in light sleep (sleep stage NREM 1) as a sign of improved sleep quality was related to improved SOSD (Lund et al., 2013). Kay et al. found a reduction in the discrepancy between subjective and objective wake time after sleep onset recorded with actigraphy in older adults showing insomnia after CBT-I (Kay et al., 2015). One study used a psycho-educative approach (Tang & Harvey, 2004). Persons suffering from insomnia were either informed about the discrepancy between actigraphy results and sleep protocol or not. The "informed group" showed a higher accuracy in the subsequent documentation of sleep with sleep protocol. A similar study was performed on the basis of polysomnography: a series of four patients with paradoxical insomnia were informed about their actual sleep using video and polysomnographical data. Two patients responded well to the novel method, suggesting that sleep education on the basis of polysomnography is a promising way to improve sleep misperception (Geyer et al., 2011) .

The objective of this explorative study was to investigate whether insomnia patients participating in a standardized CBT-I program including polysomnography and educational components on SOSD do not only show improved objective and subjective sleep parameters but also show changes in SOSD. We investigated 92 well-defined patients with chronic insomnia regarding their SOSD. All patients participated in an inpatient CBT-I program lasting 14 days (Crönlein et al., 2014). The program included psycho-educative elements about SOSD.

## Methods

### PARTICIPANTS

All patients admitted for an inpatient CBT-I program (Crönlein et al., 2014) between 2009 and 2014 were screened for the study. Only patients showing a chronic form of insomnia were admitted to the program. Inclusion criteria were checked in an interview with each patient based on ICD-10 (by TC, PG and PS): inability to fall into and/or maintain sleep in absence of acute stress or discomfort, impairment of daytime well-being because of disturbed sleep, enhanced focusing on sleep problems and the absence of an untreated sleep apnea or restless legs syndrome. Patients had to be suffering from insomnia symptoms for at least 1 year. Exclusion criteria were untreated or treated other sleep disorders (for example AHI> 15/h), severe psychiatric disorders or medical conditions that would prevent a participation in the program (including sport activities). Different comorbid psychopathologies were assessed by TC and PG. Physical examinations were performed by physicians on ward and supervised by PG and TW.

Two hundred forty-two patients participated in the program. Ten patients were excluded because of preexisting sleep apnea treated with CPAP. Fourteen patients had to be excluded because of untreated sleep apnea apparent in the baseline nights, 10 patients because of severe psychiatric disorders not evident at the first interview, 3 patients quit the program prematurely, and in 6 patients polysomnographic data were not available for technical reasons. Because of the effect on SOSD by hypnotic medication, 107 patients using hypnotic medication at the beginning of the program were excluded from analysis.

### MEASUREMENTS

*Polysomnography*

All subjects underwent a polysomnography for three nights, with two successive baseline nights (baseline 1 and baseline 2) in the beginning and one polysomnography night at the end (post therapy night) of the program. Polysomnography was performed in a separated bedroom in our sleep laboratory located in the clinic. During baseline nights our usual recording times were conducted (10 p.m. to 6 a.m.). In the third night bedtimes according to the fixed bedtime schedule were adopted (0.00 a.m. to 6.a.m.). Full cardio-respiratory polysomnographic recordings were performed and scored according to the manual of the American Academy of Sleep Medicine, version 2.0 (Iber et al., 2007), including electroencephalogram (frontal, central and occipital leads, referenced to the contralateral mastoid),

electrooculogram (alternative derivation, $E_1$-$F_{pz}$ and $E_2$-$F_{pz}$), electromyogram of the chin muscle, tibialis anterior muscles bilaterally, nasal airflow (pressure transducer) thoracic and abdominal respiration (uncalibrated induction plethysmography), oxygen saturation, electrocardiogram and body position. Hypopnea definition B (alternative) was used. Sleep stages, sleep latency, total sleep time, wake time after sleep onset and respiratory events were classified by a trained staff member according to the AASM manual. During the recording and throughout the therapy patients were allowed to keep their watches; however, they were instructed not to look at them during the night.

*Psychometric Assessments of Insomnia*

The Regensburg Insomnia Scale (RIS) was developed to assess psychological symptoms of insomnia. It contains 10 items that measure sleep quantity and quality, sleep-related anxiety and worries, hypnotic intake and daytime fitness with a possible score range of 0 to 40 (cut-off for insomnia disorder: >12). Internal consistency is .890 and component analysis revealed four components. It has been cross-validated with the Pittsburgh Sleep Quality Index (PSQI; Crönlein et al., 2013). The PSQI was designed to measure the subjectively perceived sleep quality (Buysse et al., 1989). A score of 6 points and more is considered to be pathological.

*Assessment of Subjective Sleep Data*

Subjective sleep latency in minutes (SubSOL), subjective sleep time in minutes (SubTST), and subjective wake time after sleep onset (SubWASO) were assessed with a morning questionnaire administered after each polysomnography night. Within this questionnaire subjective sleep quality was asked in a Likert scale using following grades: 1 = *very good*; 2 = *good*; 3 = *fairly good*; 4 = *acceptable*; 5 = *not acceptable*; and 6 = *insufficient*.

## PROCEDURE

On the day of admission all patients had a physical and psychological examination conducted by physicians from our sleep laboratory (supervised by PG and TW) and filled out the RIS and the PSQI. Polysomnography was performed as specified above. After each polysomnography night, patients were informed individually about their results (by TC and PG) of objective and subjective sleep and in case of SOSD the gap was explained. Hereby, patients were sat in front of the computer and were informed about their individual polysomnography showing parts of their wave forms and the hypnogram. During the program (conducted by TC), all patients had to keep a 6-hour schedule of bedtime hours. This regime of fixed bedtime is a variation of the original Spielman bedtime restriction. It improves sleep quality (Crönlein et al., 2014) without producing too much tiredness, thus enabling persons to participate in the daily schedule of the program. Patients were only allowed to use the bed for sleeping (stimulus control) and were instructed to leave the bed if awake during the night. They were instructed not to check the time during the night. However, they were allowed to have an alarm clock. They practiced relaxation techniques every day. They were educated about insomnia-specific dysfunctional behavior and cognitions about sleep in group sessions. Information on SOSD as a part of dysfunctional thinking was part of the psycho-educative program. Patients were informed about the possible gap between objective and subjective sleep and were encouraged to give the thought a chance that their body produces more sleep than normally is remembered. CBT-I was conducted in 10 hours of group and 5 hours of single sessions. All patients were treated in groups of eight persons. During the program all patients stayed as normal inpatients on our psychiatric ward. All patients signed an informed consent. The study was approved by the Ethics Committee of the University of Regensburg.

## STATISTICS

The following sleep parameters were studied: sleep onset latency (SOL), total sleep time (TST), sleep efficiency (SE), wake time after sleep onset (WASO), subjective sleep onset latency (SubSOL), subjective sleep time (SubTST) and subjective wake time after sleep onset (SubWASO). Sleep perception parameters were obtained by calculating the difference scores of subjective and objective sleep (in minutes). This procedure was done for sleep latency (SPSOL) parameters, sleep duration (SPTST) parameters and wake time after sleep onset (SPWASO) parameters of all nights.

In order to analyze changes in objective and subjective sleep parameters (SOL, TST, WASO, SE, SubSOL, SubTST and SubWASO) and sleep perception parameters (SPSOL, SPTST and SPWASO) over time, repeated measures analyses of variance (ANOVA) with the within-subjects factor measurement time point (baseline 1, baseline 2, post therapy night) were performed. The sphericity of data was checked with Mauchly-Tests. In case of significant Mauchly-Tests, Greenhouse-Geisser corrections were applied. Bonferroni corrected pairwise comparisons were performed. Non-parametric tests for paired samples were used to calculate differences in subjective sleep quality grades between two baseline nights and between baseline 2 and the post therapy night (Wilcoxon-Test). Non-parametric tests were

Table 1
Epidemiological Data From Patients With Severe and Chronic Insomnia All Participating in a Standardized Program of Cognitive Behavior Therapy for Insomnia

| | |
|---|---|
| Number | 92 |
| Women | 76 |
| Age | 50.9 (± 12.1 years) |
| Mean duration of insomnia | 12.4 (± 11.5 years) |
| Body mass index | 24.3 (± 4.4 kg/m$^2$ ) |
| Mean Score PSQI | 14.3 (± 2.6) |
| Mean RIS Score | 24.1 (± 5.3) |

RIS = Regensburg Insomnia Rating Scale; PSQI = Pittsburgh Sleep Quality Index

used because subjective sleep quality grades are based on the Likert scale.

Comparisons of subjective and objective sleep latencies (SubSOL and SOL) as well as subjective and objective sleep duration (SubTST and TST) and subjective and objective wake time after sleep onset (SubWASO and WASO) for all polysomnographic nights were done using paired t-tests.

For all three nights, correlation coefficients (Spearman rho) were calculated between subjective sleep quality and sleep perception parameters (SPSOL, SPTST and SPWASO).

Cohen's d was calculated for all parameters between the two baseline nights and between baseline 2 and the night after therapy.

A level of significance of 0.05 was selected. SPSS 22.0 was used.

## Results

### EPIDEMIOLOGICAL DATA

Ninety-two patients with chronic insomnia were included in the analysis (Table 1). The sample consisted of middle-aged adults (mean age 51 years), predominantly females. They all showed chronic insomnia with a mean duration of 12 years. Self-estimation of severity of insomnia (RIS and PSQI) was above the cut-off scores.

### OBJECTIVE SLEEP DATA

Repeated measures ANOVA showed a significant effect of time for sleep onset latency, $F(1.33, 120.57) = 15.31$; $p < .0005$. Bonferroni corrected pairwise comparisons showed no reduction from baseline 1 to baseline 2 ($p = .133$) and a significant reduction from baseline 2 to the post therapy night ($p = .001$). Repeated measures ANOVA showed a significant effect of time for total sleep time, $F(1.84, 167.57) = 11.56$; $p < .0005$. Total sleep time was enhanced from baseline 1 to baseline 2 ($p < .0005$) and was reduced from baseline 2 to the post therapy night ($p < .0005$) (see Figure 1). However, it should be noted here that post therapy night time in bed was reduced from 8 to 6 hours. A significant effect of time for wake time after sleep onset, $F(1.94, 176.18) = 24.52$; $p < .0005$, was seen. Bonferroni corrected pairwise comparisons showed no reduction from baseline 1 to baseline 2 ($p = .121$) and a significant reduction from baseline 2 to the post therapy night ($p < .0005$). SE showed a significant change over time, $F(1.82, 163.56) = 22.02$; $p < .0005$. Post hoc tests showed an improvement in sleep efficiency from baseline1 to baseline 2 ($p = .001$) and from baseline 2 to the post therapy night ($p = .004$).

### SUBJECTIVE SLEEP DATA

Subjective sleep latencies changed over time, $F(1.77, 160.73) = 9.86$; $p < .0005$. Post hoc tests revealed no

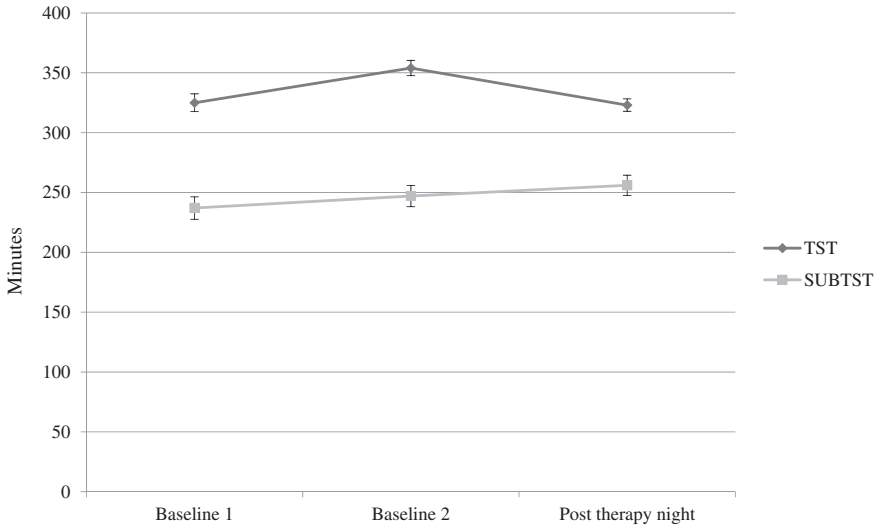


FIGURE 1 Objective (TST) and subjective (SUBTST) sleep duration in minutes in two baseline nights and the night after therapy. Means and standard errors of the means.

significant differences between both baseline nights (p = 1.0), but a significant improvement was seen from baseline 2 to the post therapy night (p = .006). Subjective sleep duration did not show any changes over time, F(2,182) = 2.12; p = .124 (see Figure 1). Subjective wake time after sleep onset changed over time, F(1.95, 177.71) = 17.203; p < .0005. Post hoc tests revealed no significant differences between both baseline nights (p = .869), but a significant improvement was seen from baseline 2 to the post therapy night (p < .0005).

### CHANGES IN SLEEP PARAMETERS AFTER THERAPY

The Wilcoxon Test revealed no significant difference between grades (Quality of Sleep) of the two baseline nights, Z (92) = -.72; p = .471. However, a significant improvement in sleep quality was seen after therapy, Z (92) = -2.39; p = .017.

We could see medium Cohen's d effects after therapy for SOL, WASO, TIB, SPT and SubWASO and small effects regarding the change of SE, NREM3 and SubSOL. Between baseline nights we saw small Cohen's d effect sizes in SOL, TST, WASO and SE (see Table 2).

### PERCEPTION OF SLEEP LATENCY

Paired t-tests revealed significant differences between subjective and objective sleep latencies in all three nights (baseline 1: t(91) = -7.08, p < .0005, baseline 2: t(91) = -7.50; p < .0005; post therapy night: t(91) = -7.70; p < .0005). Sleep latency was overestimated in all nights.

Repeated measures ANOVA showed a change of SOSD parameters (see Table 2) of sleep latency (SPSOL), F(1.66, 151.33) = 3.12; p = .056, however, not reaching a level of significance. Bonferroni corrected pairwise comparisons revealed no difference between both baseline nights (p = 1.0) and a tendency between baseline 2 and the post therapy night (p = .05).

### PERCEPTION OF SLEEP DURATION

Paired t-tests revealed significant differences between subjective and objective sleep duration for all three nights. Sleep duration was underestimated: at all times (baseline 1: t(91) = -10.05, p < .0005, baseline 2: t (91)= 11.33 p < .0005 and the post therapy night 3: t(91) = -7.65; p < .0005.

Repeated measures ANOVA showed a significant change of sleep perception (SPTST) over time, F(1.85, 168.67) = 8.04; p < .001. Post-hoc tests revealed no differences between baseline 1 and baseline 2 (p = .199) and a significant difference between baseline 2 and the post therapy night (p < .0005).

### PERCEPTION OF WAKE AFTER SLEEP ONSET

Paired t-tests revealed significant differences between subjective and objective sleep wake time after sleep onset for all three nights. Wake time after sleep onset was overestimated at all times

Table 2
Sleep Parameters of 92 Insomnia Patients at Two Consecutive Baseline Nights and the Night After Therapy

| Sleep parameters | Baseline night1 | Baseline night2 | Cohen's d | Post Therapy night | Cohen's d | F; p |
|---|---|---|---|---|---|---|
| Sleep onset latency | 23.8 ± 37.0 | 12.6 ± 16.3 | - 0.39 | 6.2 ± 7.1 | - 0.51 | 15.31; p < .0005 |
| Total sleep time | 325.5 ± 70.8 | 354.0 ± 41.8 | 0.49 | 323.6 ± 50.0 | - 0.66 | 11.56; p<. 0005 |
| WASO | 80.0 ± 50.4 | 67.1 ± 51.9 | - 0.25 | 40.8 ± 29.1 | - 0.62 | 24.54; p< .0005 |
| Time in Bed | 436.3 ± 29.8 | 441.0 ± 41.1 | 0.13 | 376.8 ± 39.1 | - 1.6 | 130.21; p< .0005 |
| Sleep efficiency (%) | 74.0 ± 15.0 | 79.9 ± 12.4 | 0.43 | 84.6 ± 12.6 | 0.38 | 22.02; p< .0005 |
| Sleep period time | 402.7 ± 51.3 | 420.1 ± 47.4 | 0.15 | 364.3 ± 48.4 | - 1.16 | 43.81; p< .0005 |
| NREM1 (% of SPT) | 11.1 ± 5.8 | 10.6 ± 5.5 | - 0.09 | 10.3 ± 5.8 | 0.05 | .854; p = .431 |
| NREM2 (% of SPT) | 41.6 ± 10.4 | 42.2 ± 10.5 | 0.06 | 43.9 ± 9.6 | 0.17 | 1.70; p =.185 |
| NREM3 (% of SPT) | 13.4 ± 8.9 | 14.5 ± 8.5 | 0.13 | 18.0 ± 11.7 | 0.34 | 13.17; p<.0005 |
| REM (% of SPT) | 15.2 ± 16.7 | 16.4 ± 6.0 | 0.10 | 15.9 ± 5.6 | - 0.09 | .431; p = .651 |
| SubSOL (min.) | 68.4 ± 61.5 | 61.1 ± 65.4 | - 0.11 | 38.0 ± 39.9 | - 0.43 | 9.86; p < .0005 |
| SubTST (min.) | 237.5 ± 89.9 | 247.6 ± 89.5 | 0.11 | 256.8 ± 80.2 | 0.11 | 2.11; p = .124 |
| SubWASO (min.) | 128.5 ± 91.0 | 117.7 ± 97.9 | - 0.11 | 74.0 ± 64.1 | - 0.53 | 17.203; p = .215 |
| Sleep quality | 3.5 ± 1.0 | 3.4 ±1.0 | | 3.1 ± 0.9 | | $Chi^2$ 12.741; p < .0005 |
| SPSOL (min.) | 44.5 ± 60.3 | 48.5 ± 62.0 | 0.06 | 31.8 ± 39.6 | - 0.32 | 3.12; p = .056 |
| SPTST (min.) | -88.0 ± 84.0 | -106.4 ± 90.0 | - 0.21 | -66.8 ± 83.7 | 0.46 | 8.04; p =.001 |
| SPWASO (min.) | 48.5 ± 89.8 | 50.6 ± 81.7 | 0.02 | 35.2 ± 64.0 | - 0.21 | 1.55; p = .215 |

*Note.* *p* values from ANOVA and Cohen's *d* are reported. WASO = Wake time after sleep onset, SPT = Sleep period time, NREM= Non-Rapid eye movement sleep, REM = Rapid eye movement sleep, SubSOL = Subjective sleep latency, SubTST = Subjective sleep time, SubWASO = Subjective wake time after sleep onset; Sleep quality = grades 1 to 6; SPSOL = Difference between subjective and objective sleep latency, SPTST = Difference between subjective and objective sleep time, SPWASO = Difference between subjective and objective wake time after sleep onset.

(baseline 1: t(91) = -5.18, p < .0005, baseline 2: t (91)= -5.941; p < .0005 and therapy night 3: t(91) = -5.28; p < .0005). Repeated measures ANOVA did not show a significant change of perceived wake time after sleep onset, F(1.98; 180.37) = 1.55; p = .215 (see Table 2).

We could see small Cohen's d effect sizes in SPSOL, SPTST and SPWASO after therapy.

### CORRELATION BETWEEN SLEEP PERCEPTION AND SLEEP QUALITY

SOSD in sleep time was associated with less positive sleep quality. This association was found in all three nights: baseline 1: Spearman rho = -.52; p < .0005; baseline 2: Spearman rho = -.41; p < .0005; night after therapy: Spearman rho = -.53; p < .0005. We found a positive correlation between sleep quality and SOSD of sleep latency in all three nights. Baseline 1: Spearman rho = .28; p = .007; baseline 2: Spearman rho = .24; p = .021; the night after therapy: Spearman rho = .35; p < .0005. A smaller magnitude of overestimation of sleep latency was associated with a better subjective quality of the night. Moreover, there was a positive correlation between perception of wake time after sleep onset and estimation of sleep quality in all nights: baseline 1: Spearman rho = .44; p < .0005; baseline 2: Spearman rho = .46; p < .0005; the night after therapy: Spearman rho = .43; p < .0005. Thus accuracy of sleep perception was correlated with subjective quality of the night (see Figure 2).

## Discussion

To our knowledge this is the first study investigating the effect of CBT-I on SOSD in insomnia disorder using polysomnography in a large sample of patients. Our sample consisted of patients suffering from a chronic and severe form of insomnia disorder, which is apparent in their high PSQI and RIS scores. Patients showed an underestimation of sleep time and overestimation of sleep latency and wake time after sleep onset, which is very frequent in insomnia disorder. We are aware that not all patients show SOSD (Edinger & Krystal, 2003) and the new classification system ICSD-3 ceased to use subtype insomnia disorders such as paradoxical insomnia. However, SOSD is still often found in insomnia patients and more research is needed to disentangle its underlying mechanisms.

In our sample, the degree of SOSD was consistent over two consecutive baseline nights. After participating in a 14-day CBT-I program, an improvement in estimating sleep duration was observed. Our data are in line with earlier studies (Kay et al., 2015).

What are possible explanations for better sleep perception after CBT-I? One reason may be an improvement in objective sleep quality. After CBT-I, sleep latency was shorter and sleep efficiency increased. Thus, it is possible that a better sleep efficiency may have an impact on SOSD. It was shown earlier that SOSD seems to be associated with the number of nightly wake time periods (Knab & Engel, 1988). By reducing nightly wake time after

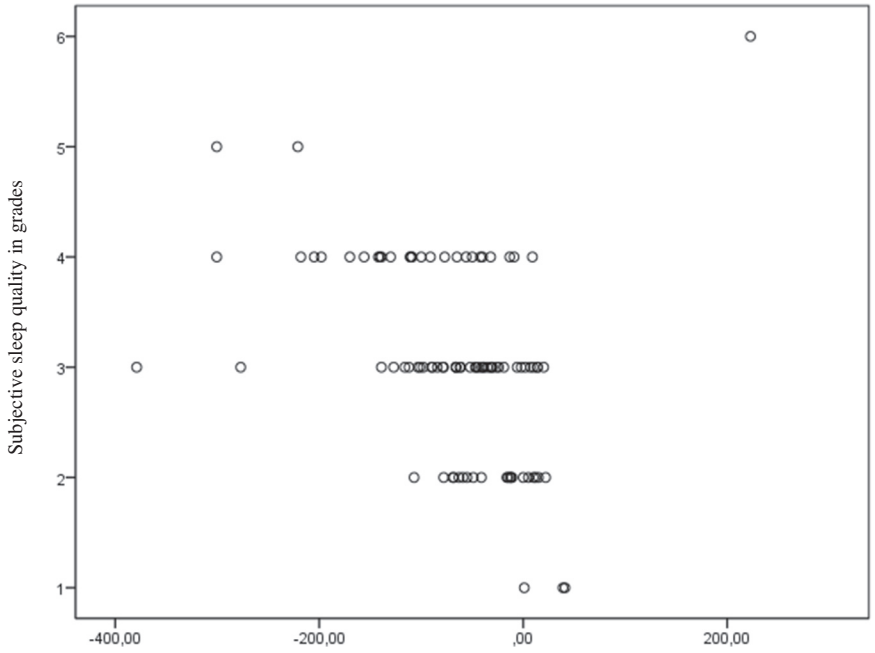


FIGURE 2 Subjective sleep quality expressed in grades and sleep time perception parameters in the night after therapy. Spearman rho = .53; p< .0005.

sleep onset, it is possible that sleep may be perceived more accurately according to polysomnographic measurements. Future studies are necessary to investigate the effect of consolidated versus unconsolidated sleep on SOSD. However, our data point in this direction.

Another reason may be an improvement in perceived sleep quality. The patients' evaluation of parameters of sleep quality improved at the end of therapy. There was a significant correlation between subjective parameters and subjective-objective sleep time discrepancy. It is possible that there is an interaction of perceived quality of sleep and the accuracy of estimating sleep duration. Hence, experiencing bad sleep may impair the capability to estimate objective sleep duration. It is possible that patients are judging according to plausibility: I feel badly, therefore I must have slept less. Future studies here should highlight this particular association of emotion and sleep estimation.

Regarding the effect of CBT-I on sleep perception, we suppose that psycho-educative strategies about sleep, especially with respect to the differences of objective and subjective sleep, could be further reason for the improvement. This would be in line with the findings of Tang et al., who improved sleep perception by explaining to the patients what their measured sleep actually was (Tang & Harvey, 2004). Improved sleep perception therefore could be a consequence of a specific education.

It is necessary mentioning that although there was a statistical improvement in our data, we saw that SOSD did not cease altogether. As Tang and Harvey (2004) have found, there may be a correlation with persistent sleep-related worries we did not measure. However, in a post hoc analysis we could see a significant correlation between the degree of SOSD and the RIS item ("I have the feeling of not having slept at all"; Spearman rho = .367; p = .0005). This indicates that some patients may not only have problems with estimating sleep time correctly but in feeling sleep at all (McCall & Edinger, 1992), which again fuels the discussion about the validity of current sleep measurements (Rezaie et al., 2018).

### LIMITATION

A limiting factor is the lack of a sham treatment as a control condition so that no statement about an effect of CBT-I on SOSD is allowed. However, we have two baseline nights, so that an influence of CBT-I is at least possible. Further studies using sham treatments are necessary to prove any effects. A further limitation of the study was the lack of sleep diaries for pre and post therapy and the fact that no distinction between different grades of SOSD severity was conducted. However, since subtypes such as paradoxical insomnia were abandoned in newer classification systems, no subtyping was performed in our study. In addition, we did not discriminate between the effect of educating about SOSD and the other CBT-I tools. We had a rather explorative interest in whether participating at CBT-I including psycho-educative components about SOSD would change it per se. At this point it is important to mention that patients were allowed to have an alarm clocks in their rooms. Tang et al. have shown the negative effects on the process of falling asleep and overestimating sleep latency (Tang et al., 2007). However, the patients were explicitly informed about the relationship of clock watching to impaired sleep and were strictly instructed to turn away the displays. In addition, the fact that the third night was conducted with the 6-hour bedtime restriction schedule could be seen as a limiting factor as well. This might reflect not a naturalistic result in case of an improvement of more than a 6-hour sleep duration. However, our sample did not show an improvement in total sleep time, which is consistent with former polysomnographic data on CBT-I effects (Kyle et al., 2014).

Our inpatient setting differs from normally delivered CBT-I, since there is closer contact between therapist and patients and the possibility to control the compliance every day. The setting also differs regarding the aspect of two polysomnographic baseline nights, comprising the possibility to educate the patients about their individual sleep and possible SOSD. Thus, further studies should show whether including an educational component about SOSD in an outpatient setting has a similar effect.

## Conclusion

CBT-I may improve SOSD in patients with chronic insomnia. Possible factors could be better sleep efficiency together with a better understanding of SOSD mediated by specific psycho-educative elements of CBT-I.

### Conflict of Interest Statement

This was not an industry-supported study. The authors have indicated no conflicts of interest related to the study.

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Received: September 1, 2018
Accepted: March 15, 2019
Available online: 23 March 2019