Sleep Restriction Therapy for Insomnia is Associated with Reduced Objective Total Sleep Time, Increased Daytime Somnolence, and Objectively Impaired Vigilance: Implications for the Clinical Management of Insomnia Disorder

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Study Objectives: To investigate whether sleep restriction therapy (SRT) is associated with reduced objective total sleep time (TST), increased daytime somnolence, and impaired vigilance.

Design: Within-subject, noncontrolled treatment investigation.

Setting: Sleep research laboratory.

Participants: Sixteen patients [10 female, mean age = 47.1 (10.8) y] with well-defined psychophysiological insomnia (PI), reporting TST ≤ 6 h.

Interventions: Patients were treated with single-component SRT over a 4-w protocol, sleeping in the laboratory for 2 nights prior to treatment initiation and for 3 nights (SRT night 1, 8, 22) during the acute interventional phase. The psychomotor vigilance task (PVT) was completed at seven defined time points [day 0 (baseline), day 1,7,8,21,22 (acute treatment) and day 84 (3 mo)]. The Epworth Sleepiness Scale (ESS) was completed at baseline, w 1-4, and 3 mo.

Measurement and results: Subjective sleep outcomes and global insomnia severity significantly improved before and after SRT. There was, however, a robust decrease in PSG-defined TST during acute implementation of SRT, by an average of 91 min on night 1, 78 min on night 8, and 69 min on night 22, relative to baseline (P < 0.001; effect size range = 1.60-1.80). During SRT, PVT lapses were significantly increased from baseline (at three of five assessment points, all P < 0.05; effect size range = 0.69-0.78), returning to baseline levels by 3 mo (P = 0.43). A similar pattern was observed for RT, with RTs slowing during acute treatment (at four of five assessment points, all P < 0.05; effect size range = 0.57-0.89) and returning to pretreatment levels at 3 mo (P = 0.78). ESS scores were increased at w 1, 2, and 3 (relative to baseline; all P < 0.05); by 3 mo, sleepiness had returned to baseline (normative) levels (P = 0.65).

Conclusion: For the first time we show that acute sleep restriction therapy is associated with reduced objective total sleep time, increased daytime sleepiness, and objective performance impairment. Our data have important implications for implementation guidelines around the safe and effective delivery of cognitive behavioral therapy for insomnia.

Keywords: Adverse effects, CBT, insomnia, sleep restriction therapy, sleepiness, vigilance

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INTRODUCTION

Cognitive behavioral therapy for insomnia (CBT-I) is commonly regarded as the treatment of choice for persistent insomnia disorder.[1-3] CBT-I has been shown to be as effective as pharmacotherapy in the short term but, in contrast to pharmacotherapy, leads to durable improvements in sleep (for up to 2 y post-intervention).[4] One of the frequently cited advantages of CBT-I, and non-pharmacological approaches in general, is the absence of or potential for treatment-related adverse effects.[5,6] This is in contrast to pharmacotherapy where, for example, negative short- and long-term effects of sedative hypnotics have been well described.[7-10] Indeed, adverse effects are routinely assessed in randomized, placebo-controlled clinical trials of hypnotics and guide regulatory approval.[11] Somewhat surprisingly, adverse effects are almost never systematically recorded and/or reported in trials of psychological/behavioral treatments.[5,12-14]

Sleep restriction therapy (SRT), a standard behavioral strategy used within multi-component CBT-I[2] and as a stand-alone intervention,[6,15,16] involves restricting a patient's time in bed (TIB, sleep window) to match their average (self-report) total sleep duration. The sleep window is then titrated, weekly, based on sleep efficiency (the proportion of TIB spent asleep), in order to arrive at the patient's core sleep requirement. Decreasing the opportunity to sleep over successive nights, it is argued, builds homeostatic sleep pressure, stabilizes circadian control of sleep and wakefulness, and dampens presleep cognitive and physiological (hyper)arousal, leading to shorter sleep latencies and more consolidated, uninterrupted sleep.[6,17-20] CBT-I practitioners often advise patients that, because of the reduced opportunity for nighttime sleep, coupled with 'prohibition' of daytime napping, increased sleepiness may emerge during the initial phases of SRT implementation, resulting in a transient worsening of daytime functioning.[18,21] Magnitude of TIB restriction may also be affected by the well-established
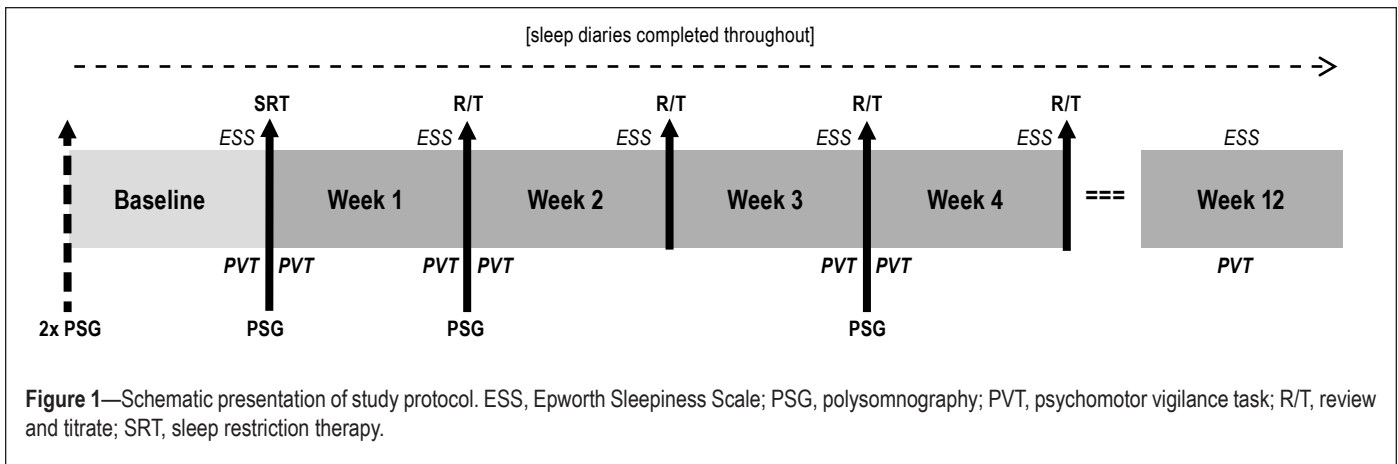
Figure 1—Schematic presentation of study protocol. ESS, Epworth Sleepiness Scale; PSG, polysomnography; PVT, psychomotor vigilance task; R/T, review and titrate; SRT, sleep restriction therapy.

objective-subjective sleep discrepancies, known to characterize some patients with insomnia.[22-24] That is, patients may be assigned TIB prescriptions that are significantly lower than pretreatment objective sleep, leading to marked sleep loss over several weeks.[6] Patients are, therefore, advised not to drive or operate heavy machinery if they feel excessively sleepy.[18,21]

Although these guidelines have evolved from clinical experience, there has been little systematic investigation of the nature or magnitude of CBT-I-induced daytime sleepiness and impairment. When investigating the utility of modafinil as an adjunct to CBT-I, Perlis and colleagues[25] showed that those receiving CBT + placebo (n = 10) reported increased Epworth Sleepiness Scale (ESS) scores 1 w post-SRT delivery. In contrast, both the therapeutic arm (CBT + modafinil) and additional control group (modafinil + contact) did not exhibit such a marked increase in ESS scores. Kyle et al.[6] conducted the first in-depth examination of single-component SRT. A mixed-methods approach was applied that involved questionnaire-based measures, semistructured interviews, and real-time audiodiaries to probe the patient experience of treatment. During acute implementation of SRT, patients subjectively reported problems with excessive daytime sleepiness, which negatively affected daytime functioning beyond pretreatment levels. Of note, more than one third of the audiodiary subsample complained, during real-time recordings, that driving was adversely affected [e.g.: "*Woke up bright and breezy, half six, Tuesday morning, raring to go, got into the car…and within 20 min I was absolutely exhausted, so bad that I swear I was nearly falling asleep all the way to work. It was torture, I was cross-eyed, eyes drooping, driving*"; "*driving was a nightmare, and I've never ever had an issue with driving before*"; "*I felt, really, I was a danger on the road.*"][6] Despite these acute difficulties, patients responded well to treatment, evidencing robust improvements in sleep and daytime functioning at 3 mo follow-up. Recently, Miller et al.[20] complemented these qualitative findings using ecological momentary assessment. The authors reported that point-in-time assessments of 'sleepiness/fatigue' increased during w 1 of SRT, whereas 'positive mood' and 'alert cognition' decreased, relative to baseline.

To date, no study has profiled whether subjective reports of treatment-related dysfunction are reflected in objective performance impairments. Moreover, it is unclear to what extent sleep is actually restricted during SRT and whether this is associated with elevated daytime sleepiness, measured with a validated instrument. Information on the magnitude and time course of sleep loss, daytime sleepiness, and performance impairment may have important implications for the future refinement, delivery, and safe dissemination of CBT-I.

METHOD

In the current study, 16 patients with psychophysiological insomnia (PI) took part in a brief regimen of SRT. In order to profile changes in sleep time and objective performance, patients slept in the laboratory on five occasions (twice × baseline, thrice × during acute treatment) and completed a psychomotor vigilance task (PVT) at seven defined time points. The ESS was also completed on a weekly basis (baseline, w 1-4 and at 3 mo) to index changes in daytime somnolence (Figure 1 provides a schematic description of protocol). A comparison group of good sleepers (n = 15) was recruited in order to examine baseline differences in PVT performance.

We hypothesized that acute implementation of SRT would lead to reduced total sleep time (TST), which would be accompanied by impairments in vigilance (increased attentional lapses and slowed reaction time [RT]) and increased daytime sleepiness.

Sample

Sixteen thoroughly screened patients with PI were recruited to take part in SRT for insomnia disorder. Individuals initially responded to media advertisements looking for poor sleepers to sleep for 2 nights in the sleep laboratory, as part of a study into sleep related attentional bias (grant # R01MH077901). This was a noninterventional study, but on completion of the overnight protocol (see following text for details), those without evidence of occult sleep disorder pathology were invited to take part in the current treatment study, using SRT. A group of healthy age- and sex-matched good sleepers (n = 15) was recruited for comparative purposes.

Assessments

Sleep Status

Patients with PI participated in a telephone interview by an expert in behavioral sleep medicine to assess the absence of comorbidities and medication use, as well as the presence of insomnia, defined as satisfying the following criteria for subjective sleep impairment:

- Report of sleep disturbance for at least 3 nights per week for at least 6 mo
- Sleep onset latency (SOL) and/or wake after sleep onset (WASO) > 30 min
- TST ≤ 6 h
- Sleep efficiency < 85%
- Daytime impairment attributed to disturbed sleep
- Insomnia Severity Index (ISI) score ≥ 15

The phone interview was based on guidelines described by Morin and Espie[21] and supplemented with a sleep disorders screening questionnaire.[26] Those deemed eligible were invited to attend a screening day, involving a thorough sleep and psychiatric interview (Mini-international Neuropsychiatric Interview[27]) with a licensed clinical psychologist trained in behavioral sleep medicine, and a medical assessment (electrocardiogram, blood chemistries, medical history, and drug screening) by a certified physician. Patients meeting research diagnostic criteria for PI,[28] and who met all other inclusion/exclusion criteria, subsequently slept for 2 consecutive nights at the University of Glasgow Sleep Centre where they underwent polysomnographic (PSG) assessment (see subsequent text).

Good sleepers participated in the same phone interview to assess inclusion/exclusion criteria, defined as the absence of sleep, psychiatric or (unstable) medical disorder, and the endorsement of good quality, restorative sleep, in addition to the following:

- SOL and WASO < 15 min
- Number of nighttime awakenings ≤ 2
- TST > 6 h
- Sleep efficiency > 85%
- Stable sleep period between 22:00 and 08:00

All study participants completed a 7-day sleep diary[21] to assess sleep continuity and quality and help rule out circadian phase disturbance. Patients completed sleep diaries for 6 w in total (baseline, treatment w 1-4, and at 3 mo). Participants also completed the Hospital Anxiety and Depression Scale (HADS),[29] supplementing the psychiatric screening interview and helping to rule out clinical level anxiety/affective disorders. Patients completed the ISI,[30] a sensitive measure of insomnia severity, at baseline, 4 w (posttreatment), and 3 mo. Finally, patients completed the ESS[31] at six time points (baseline, w 1-4, and 3 mo). The standard ESS does not include a specified time frame and thus for the purpose of the current study, modifications were made so that patients completed the ESS with reference to "in the last week…", permitting assessment of weekly sleepiness levels.

It should be noted that matching between patients and good sleepers was initiated on a subject-by-subject basis, with each patient matched with a corresponding good sleeper (GS) in terms of sex and age ± 2 y. Successful one-to-one matching was achieved for 14 of 16 patients.

Polysomnography

A standard PSG montage was used, involving electroencephalographic [EEG: Fp1 (neutral), C3, P3 (reference), O1, Fpz, Fz, Cz, Pz, Oz, F4, C4], electrooculographic (EOG: horizontal and vertical) and electromyographic (submental) recordings. On night 1 of the baseline phase, all participants were screened for sleep disordered breathing and periodic limb movements through monitoring of abdominal and thoracic effort, nasal airflow, oximetry, and bilateral tibialis anterior EMG. Sleep was recorded on a Lifelines Trackit™ ambulatory recorder and scored visually by two experienced scorers (> 90% interscorer reliability) according to criteria by Rechtschaffen and Kales.[32] For study inclusion, patients were required to have an apnea-hypopnea index and periodic limb movements of sleep arousal index < 10. This initial night served as screening and adaptation to the sleep environment, whereas night 2 of the baseline phase was used as a comparator to index change during SRT. During baseline PSG assessment, patients implemented normal, 'at-home' bed and rise-times (guided by sleep diary records).

For the SRT intervention, patients slept in the sleep laboratory on three additional nights (SRT nights 1, 8, and 22; Figure 1) where sleep parameters were recorded (EEG, EMG, EOG) during implementation of a prescribed sleep window (based on sleep diary reports of TST; see details of SRT intervention in the following paragraphs). For the purpose of the current study, PSG-defined TST was the only selected variable of interest, to index magnitude of sleep reduction between baseline and SRT nights. Future reports will focus on changes in objective sleep continuity parameters, as well as sleep macroarchitecture and microarchitecture, in relation to treatment response.

Psychomotor Vigilance Task

The PVT is a frequently used task in sleep research to assess the effect of sleep restriction, total sleep deprivation, or altered sleep timing on basic vigilant attention. Evidence also exists that PVT metrics relate to driving simulator performance during sleep deprivation[33] and that PVT performance is reliable across repeated administrations.[34] The version of the PVT used in the current study has been applied in studies of insomnia and sleep perturbation.[35,36] In the task, participants are asked to respond with a left mouse click, as quickly as possible, to the presence of an asterisk located in the center of the computer screen. Interval onset for asterisks varied between 1 and 10 sec in duration and there were 110 experimental trials. Participants completed five practice trials at the beginning of the session to aid task familiarity. The PVT was programmed in E-prime (http://www.pstnet.com/eprime.cfm) and completed on a Dell laptop computer, at a viewing distance of 40 cm. Task duration was approximately 13 min. PVT testing took place at 18:00. The following PVT metrics[37] were analyzed: (1) attentional 'lapses' (defined as RTs > 500 msec); and (2) 1/Mean RT [lower values indicating a slowing in response speed].

Intervention

There are several different variants of SRT used in clinical practice, but our intervention was based on the original seminal work by Spielman et al.[17] and previously published protocols from our group.[6] Specifically, the SRT intervention involved one main session for delivery of treatment rationale and instructions, and four additional brief, in-person or telephone interactions to review progress and titrate sleep efficiency (Figure 1). Treatment was delivered by experts in behavioral sleep medicine via PowerPoint slides to two patients at a time, and covered SRT rationale, sleep window calculation, and troubleshooting around potential implementation difficulties. The sleep window

Table 1—Demographic and sleep characteristics for patients with psychophysiological insomnia and good sleepers

	GS (n = 15)	PI (n = 16)
Age (SD)	47.1 (10.5)	47.1 (10.8)
Sex% (F:M)	66.7/33.3	62.5/37.5
ISI	0.8 (1.1)	17.8[a] (2.8)
HADS-A	2.1 (2.3)	6.4[a] (4.0)
HADS-D	0.9 (1.6)	4.0[a] (2.2)
SOL (min)	7.1 (7.9)	38.8[a] (32.4)
WASO (min)	6.8 (11.2)	62.6[a] (58.8)
No. Awak	1.2 (1.4)	2.1[b] (1.3)
TST (min)	449.9 (41.7)	338.7[a] (57.4)
TIB (min)	503.1 (51.0)	490.6 (66.8)
SE (%)	89.9 (6.3)	69.3[a] (12.3)
SQ (0-4)	3.3 (0.4)	1.7[a] (0.6)

GS, good sleepers; HADS-A/D, Hospital Anxiety and Depression Scale; ISI, Insomnia Severity Index; No. Awak, number of awakenings; PI = psychophysiological insomnia; SE, sleep efficiency; SOL, sleep onset latency; SQ, sleep quality; TIB, time in bed; TST, total sleep time; WASO, wake after sleep onset. [a]$P < 0.01$, [b]$P < 0.10$ for group comparison.

was initially calculated based on 1 w of baseline sleep diaries, with TIB prescriptions reflecting average TST. The sleep window was subsequently titrated each week according to the following guidelines: sleep efficiency < 85%, decrease by 15 min; sleep efficiency ≥ 85-89%, no change; and sleep efficiency ≥ 90%, increase by 15 min.[17] The minimum sleep window was set at 5 h. For those patients in whom the sleep window was deemed too difficult, restrictive, or impossible to adhere to, a compromise was established between therapist and the patient. No other components of CBT-I were addressed during the intervention. Beyond the 4-w acute interventional phase, patients completed sleep diaries and sleep efficiency grids and continued to self-titrate the sleep window according to the aforementioned sleep efficiency criteria.

The study protocol was reviewed and approved by the West of Scotland NHS Research Ethics Committee (protocol no. 10/SO701/85).

Analysis

Group differences (patients versus good sleepers), with respect to demographic and sleep related variables, were assessed using independent *t*-tests. Treatment-related change in subjective sleep diary outcomes (SOL, WASO, sleep efficiency) and insomnia severity (ISI) were assessed with repeated-measures analysis of variance (ANOVA), across baseline, posttreatment (w 4) and 3-mo follow-up. PSG-TST (min), daytime sleepiness (ESS) and vigilance (lapses, RT) were similarly assessed with repeated-measures ANOVA. Logarithmic transformations were performed on variables exhibiting skewed distributions. For those variables failing to meet the sphericity assumption, degrees of freedom and corresponding probability were adjusted using the Greenhouse-Geisser correction. Significant main effects were followed up using paired *t*-tests. PSG-TST was compared across 4 nights [baseline (night number 2), and treatment nights (1, 8, and 22)], vigilance across seven time

points [day 0 (baseline), day 1,7,8,21,22 (acute treatment) and day 84 (3 mo)] and sleepiness across six time points [baseline, w 1-4, 3-mo follow-up]; with comparisons focused on change from baseline assessments. Effect sizes (ES) for paired data were calculated as follows: [mean difference / standard deviation (SD) of difference]. All comparisons were two-sided, with $P < 0.05$ indicating statistical significance, but given the *a priori* nature of our directed hypotheses, P values and effect size data are also reported for $P \leq 0.10$.

Although the primary analyses of interest focussed on assessment of within-subject change for vigilance, sleepiness and PSG-TST, recruitment of a group of good sleepers also permitted between-group comparisons at baseline, with respect to PVT lapses and RT (using independent *t*-tests).

RESULTS

Sample

Sixteen patients [10 female, mean age = 47.1 (10.8) y] initially enrolled in the study and completed session 1. One patient dropped out in the first week due to concerns about the effect of SRT on work functioning. The 15 remaining participants completed the full protocol (five laboratory nights and seven neurocognitive assessments), including 12-w follow-up. Mean age of the remaining 15 patients was 47.2 y (SD = 10.4) and 10 (66.6%) were female. The comparison group of good sleepers were identical in both age (47.1, SD = 10.5) and sex (10 female [66.6%]). As expected, patients with PI demonstrated significant sleep disturbance at baseline relative to GS (Table 1), and reported greater levels of anxiety and depression. Of note, and consistent with the diagnosis of PI, anxiety and depression scores were in the mild range and approximate those found in large nonclinical samples.[38]

Subjective Sleep: Manipulation Check of the SRT Protocol

The average prescribed sleep window for the first week of therapy was 347.0 min (SD = 32.0), which increased by 15 min over the 4-w acute SRT phase (w 4 = 362.0 min, SD = 33.0; Figure 2). Sleep diary records of TIB decreased from a baseline of 483.2 min (SD = 74.1) to 353.2 min (SD = 36.1) during w 1, in line with prescribed sleep window times, indicating close adherence to the SRT protocol (Figure 2).

Insomnia severity (measured with the ISI) significantly reduced across assessment points [$F_{(2,24)} = 85.07$, $P < 0.001$], decreasing from 17.4 (SD = 2.8) at baseline to 7.7 (SD = 3.9) at 4 w ($P < 0.001$). Further reductions were observed between w 4 and 3 mo (5.08, SD = 4.1; $P = 0.004$, and $P < 0.001$ for comparison with baseline). Subjective reports of SOL similarly changed over assessment period [$F_{(1.04,11.39)} = 16.24$, $P = 0.002$], reducing from 32.2 (SD = 21.8) min at baseline to 9.4 min (SD = 5.4) at 4 w ($P < 0.01$) and remaining at this level (8.1, SD = 5.2) at 3-mo follow-up. Both WASO and sleep efficiency showed robust changes over time [WASO: $F_{(1.04,11.41)} = 9.04$, $P = 0.011$ and sleep efficiency: $F_{(1.07,11.75)} = 28.34$, $P < 0.001$]. WASO significantly reduced from 66.8 (SD = 60.7) min at baseline to 12.4 min (SD = 10.1; $P = 0.01$) at posttreatment, remaining at this level at 3 mo (16.2 min, SD = 16.7). Changes in WASO and SOL were reflected in improved sleep efficiency, increasing from 68.0%
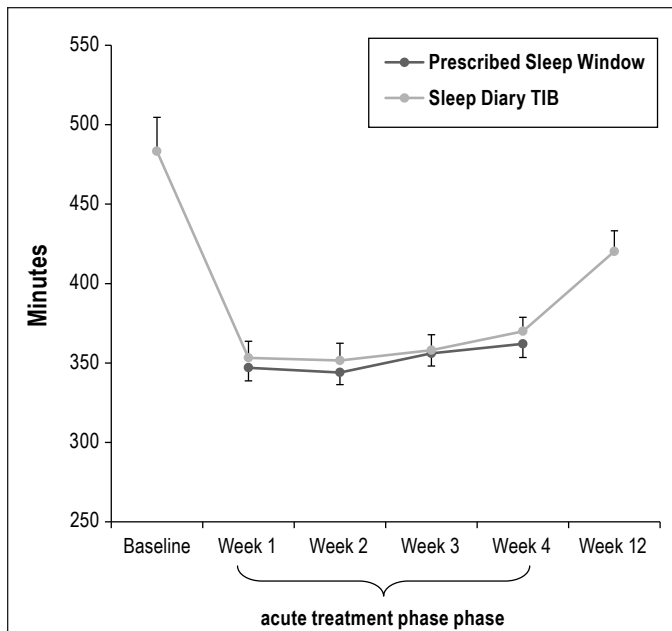
Figure 2—Descriptive profiles of mean (± standard error) sleep window prescriptions (w 1-4) and sleep-diary reported time in bed (TIB) over the course of sleep restriction therapy protocol.
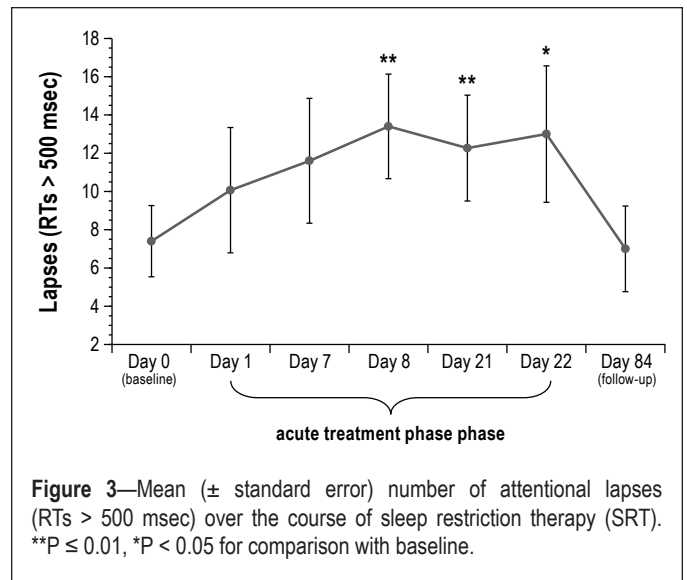

Figure 3—Mean (± standard error) number of attentional lapses (RTs > 500 msec) over the course of sleep restriction therapy (SRT). **$P \leq 0.01$, *$P < 0.05$ for comparison with baseline.

Figure 4—Psychomotor vigilance task (PVT) RT (1/mean RT ± standard error) over the course of sleep restriction therapy (SRT). Lower scores indicate a slowing in RT. **$P \leq 0.01$, *$P < 0.05$ for comparison with baseline.

(SD = 13.7) at baseline, to 90.7% (SD = 4.4; $P < 0.001$) at 4 w, which was maintained at 3 mo (91.3%, SD = 4.8). Finally, subjective TST estimates showed fluctuation over assessment points [$F(2,22) = 13.04$, $P = 0.001$]. Although there was no change in TST between baseline and posttreatment (326.8, SD = 61.1 versus 334.9, SD = 37.1; $P = 0.50$), by 3 mo TST had improved by approximately 56 min, to 383.2 min (SD = 49.3; $P < 0.01$ for baseline comparison).
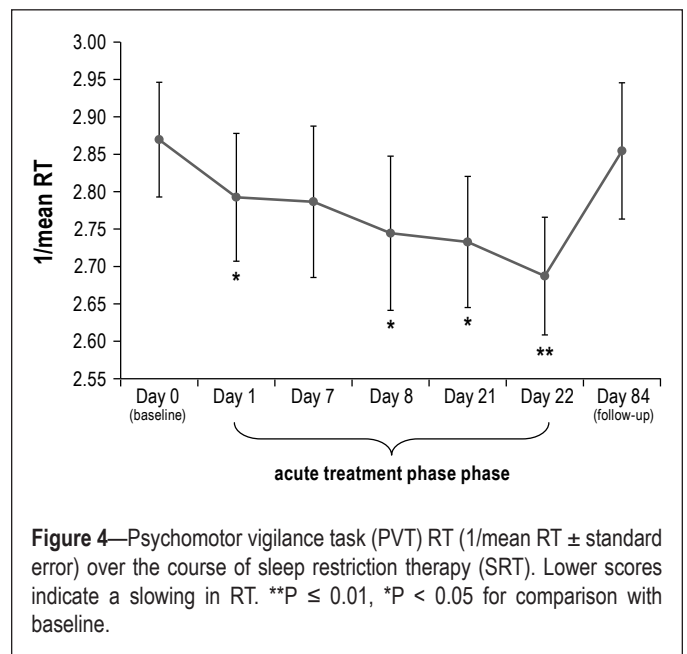
PVT Performance

PVT performance was first compared across GS (n = 15) and patients with PI (n = 15; pretreatment). Independent t-tests did not reveal any significant baseline group differences for number of attentional lapses [PI = 7.4 (SD = 7.2) versus GS = 7.2 (SD = 10.7); $t = 0.49$, $P = 0.62$] or 1/mean RT [PI = 2.87 (SD = 0.30) versus GS = 3.03 (SD = 0.36); $t = 1.31$, $P = 0.20$].

Changes in patient PVT performance across the treatment protocol [days 0, 1, 7, 8, 21, 22, 84] were next examined with repeated-measures ANOVA. A main effect of time was observed for attentional lapses [$F(6,84) = 4.45$, $P = 0.001$] and a significant quadratic trend [$F(1,14) = 30.52$, $P < 0.001$; Figure 3]. Relative to baseline (day 0), number of lapses increased (nonsignificantly) at day 1 ($P = 0.10$; ES = 0.45) and 7 ($P = 0.075$; ES = 0.50), and were significantly elevated at days 8 ($P = 0.010$; ES = 0.77), 21 ($P = 0.009$; ES = 0.78) and 22 ($P = 0.018$; ES = 0.69) of SRT. By day 84 (3 mo), lapses returned to baseline levels [3 mo = 7.0 (SD = 8.7) versus baseline = 7.4 (SD = 7.2); $P = 0.43$].

Similar findings were observed for RT, reflected in a significant main effect of time [$F(6,84) = 3.11$, $P = 0.008$], accompanied by a significant quadratic trend [$F(1,14) = 7.59$, $P = 0.015$; Figure 4]. Relative to baseline (day 0), patient RTs increased at day 1 ($P = 0.042$; ES = 0.58), day 8 ($P = 0.045$; ES = 0.57), day 21 ($P = 0.034$; ES = 0.61) and day 22 ($P = 0.004$; ES = 0.89).
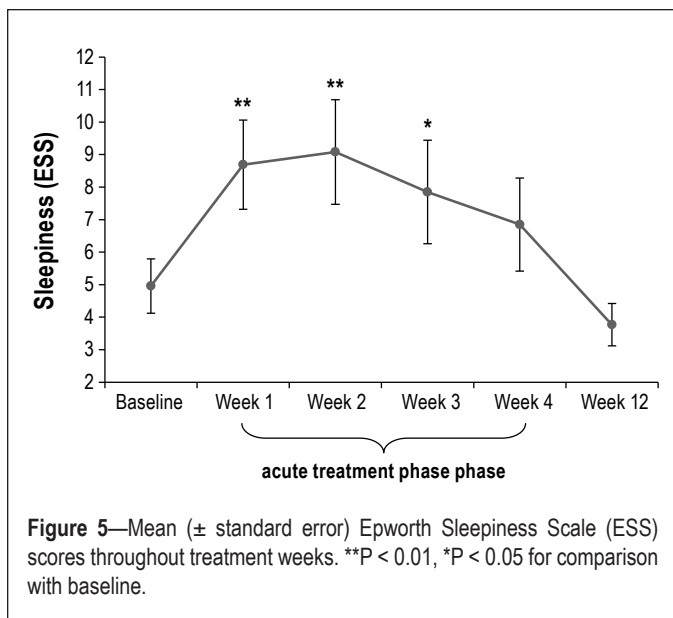
By day 84 (3 mo), RTs had returned to baseline levels (2.85, SD = 0.35 versus 2.87, SD = 0.30; $P = 0.78$).

Daytime Sleepiness

Sleepiness evidenced a significant main effect of time [$F(5,60) = 7.26$, $P < 0.001$] and a significant quadratic trend [$F(1,12) = 11.58$, $P = 0.005$; Figure 5]. ESS scores significantly increased from baseline to w 1 [4.95, SD = 3.02 versus 8.69, SD = 4.96; $P = 0.004$, ES = 0.98], w 2 [9.08, SD = 5.84; $P = 0.006$, ES = 0.92], and w 3 [7.85, SD = 5.8; $P = 0.035$, ES = 0.66]. There were no significant differences between ESS scores at baseline and w 4 (6.85, SD = 5.18; $P = 0.112$) or between baseline and week 12 (3.80, SD = 4.96; $P = 0.652$).

PSG-Defined TST

We next assessed the magnitude of change in PSG-TST, from the baseline PSG night (pretreatment) relative to SRT acute implementation, and the extent to which TST varied across the three SRT laboratory nights (nights 1, 8, and 22). There was

Figure 5—Mean (± standard error) Epworth Sleepiness Scale (ESS) scores throughout treatment weeks. **P < 0.01, *P < 0.05 for comparison with baseline.

Figure 6—Mean (± standard error) polysomnographically (PSG)-determined total sleep time (TST, min) at pretreatment and during sleep restriction therapy (SRT). **P < 0.001 for comparison with baseline.

a significant main effect of time [$F(3,39) = 27.03$, P < 0.001; Figure 6]. Baseline TST was 393.6 min (SD = 43.0), which decreased by 91 min on the first night of SRT (302.4, SD = 53.0; P < 0.001, ES = 1.62), remaining significantly reduced at night 8 (315.6, SD = 26.7; P < 0.001, ES = 1.80) and night 22 (324.6, SD = 34.6; P < 0.001, ES = 1.60). TST exhibited a trend toward improving between night 1 and 22 (by approximately 22 min; P = 0.052, ES = 0.57).

DISCUSSION

CBT-I is widely regarded as the most effective treatment option for chronic insomnia. Similar to psychological therapies in other fields, CBT-I is promoted as a safe and adverse-effect-free intervention. Our clinical and research experience suggests that CBT components, particularly SRT, may be associated with some negative effects, but examination and evidence are lacking.[6,21] Understanding possible treatment-related adverse effects has important implications for patient care. In the current study, we aimed to quantify the effect of SRT on objectively defined vigilance, daytime sleepiness, and objective TST.

The first thing to note is that our SRT intervention effectively improved the core symptoms of insomnia. That is, by w 4, diary ratings of SOL, WASO, and SE had all improved relative to baseline (with corresponding large effects). Changes in sleep diary parameters were also reflected in reduced ratings of overall insomnia severity. These findings were maintained (or enhanced) at 3 mo follow-up. Of course, our aim was not to test the effectiveness of SRT *per se*, but results from our (uncontrolled) work support the growing literature that SRT is an effective, single-component intervention.[15,16] Furthermore, improvements in sleep, coupled with reductions in diary-reported TIB during SRT—almost overlapping with prescribed sleep window times—suggests that patients followed the protocol faithfully.

Despite these posttreatment improvements in sleep continuity and insomnia severity, PVT performance was found to deteriorate during acute SRT implementation, reflected in a greater number of attentional lapses and slowed RT. To our knowledge, this is the first evidence that SRT (or any component
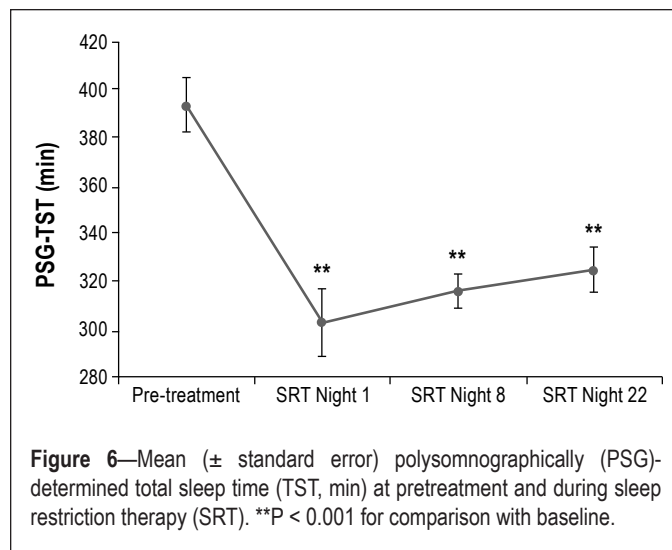
of CBT-I) is associated with objective performance impairment. Performance was impaired on three of five assessment points for attentional lapses and four of five assessment points for reaction time, relative to baseline (medium-to-large effects). By 3 mo, performance had returned to baseline levels. Consistent with meta-analytic data,[39] patients did not differ from GS at baseline with respect to PVT performance; therefore, in this study sample and protocol, insomnia *per se* was not associated with impaired vigilance, but acute treatment was.

Deterioration in PVT performance was paralleled by increased daytime sleepiness as reflected in ESS scores. Patients reported significantly elevated ESS scores during w 1-3 of treatment (medium-to-large effects). By 3 mo, however, and similar to PVT performance, ESS scores had returned to baseline levels. A reduction in TST is the most intuitive explanation for degraded performance and increased sleepiness during treatment. Comparing PSG nights, we observed a large reduction in TST by approximately 91 min on SRT night 1, 78 min on night 8, and 69 min on SRT night 22. Chronic sleep restriction protocols in healthy subjects, even with sleep curtailment of just 1.5 h, reveal cumulative impairments in PVT performance over a 14-day period.[40,41] Although we were not able to assess vigilance or objective sleep on a daily basis, it is interesting that RT performance appears to follow a relatively linear (cumulative) decline throughout the acute phase of SRT, with impairments tending to be most pronounced on days 8, 21, and 22. It is also clear that PSG-defined TST is relatively stable over the 3 assessment nights (increasing by 22 min from night 1 to night 22), and the prescribed sleep window was extended by just 15 min over the entire 4-w treatment protocol (Figure 2).

Our findings are difficult to compare with published literature because few studies have investigated the acute phase of insomnia treatment; instead, tending to focus on pretreatment to posttreatment outcomes. Previous work by our group[6,20] and others[17,25,42,43] provide both systematic and clinical evidence of treatment-related difficulties, including self-reported sleepiness, cognitive impairment, and implementation challenges, but longitudinal tracking of sleep and functioning is lacking. Treatment studies that have used PSG to assess sleep outcomes, before and after CBT-I, have not found convincing evidence of change in TST[4] and, to our knowledge, no published study has

examined the magnitude of PSG-defined TST reduction during acute implementation (although TIB reduction, similar to our study, has been shown to exceed 2 h).[17] However, inspection of published CBT-I trial data, where both objective (PSG) and subjective (sleep diary) baseline data are reported, indicates that TST discrepancies often range between 50 and 60 min[44-46] (indeed, in one study, as high as 83 min)[46]; and it is well known that a general objective-subjective sleep discrepancy exists in some patients with insomnia.[23,24,47] This discrepancy has important implications for sleep window calculation and the degree to which patients may be sleep restricted during, and possibly after, CBT treatment monitoring.

On this point, Morin and colleagues[48] reported that PSG-defined TST was significantly reduced (medium-to-large effect) in the CBT treatment arm at 6 w (post-treatment), and Buysse et al.[49] reported significant reductions in actigraphy-defined TST relative to an information-only control group after 4 w of brief behavioral therapy (SRT + stimulus control therapy). Thus, it would appear that TST reduction during CBT-I is likely the norm, rather than the exception, but the field lacks consistent (week-by-week) process data to answer this question definitively. An important point is that TST appears to return to at least baseline levels during follow-up PSG assessments,[45,46,48,50] suggesting that CBT exerts its therapeutic effect, at least in part, through correction or restoration of sleep-wake perception.[44] Priming sleep pressure through TST reduction may also be necessary to overcome cognitive arousal and consolidate sleep,[6,19,20,51] but these putative mechanistic routes require further experimental attention. Importantly, there exists the possibility that some patients, perhaps treatment nonresponders, continue to implement SRT for a prolonged period of time which, if associated with chronic sleep restriction, could have detrimental health effects.[52]

Limitations

Our findings must be interpreted within the context of several limitations. Principally, our sample size was small and we did not include an untreated (patient) control group. This limitation is partially mitigated through triangulation of methodologies (PSG, performance impairment, self-reported sleepiness), coupled with normalized trajectories of sleepiness and vigilance, at follow-up, giving us some confidence in our conclusions. Nevertheless, we cannot conclude with certainty that SRT was responsible for the observed effects. Recruitment of a group of patients with untreated insomnia, a group receiving another CBT-I component or an inactive intervention should be considered in future research studies. We also realize that SRT is often introduced within the context of a full CBT-I package and so our results may not generalize to all CBT-based interventions. An important point to remember is that SRT is commonly introduced in the second or third session of CBT-I protocols[53-56] and as such the sleep window may not be calculated based on pre-treatment diary values, but instead from sleep parameters measured during the first 2 w of CBT-I. This would potentially lead to longer sleep window prescriptions, because sleep may already be improving, than if the sleep window were based on pre-treatment data. However, this remains an empirical question that could be addressed through reanalysis of existing datasets.

Moreover, our SRT protocol may not generalize to all CBT-based approaches that include a sleep restriction component, because we (1) recruited patients reporting ≤ 6 h TST and (2) there are many variants of SRT (e.g., some involve prescribing a sleep window based on TST plus 30 min,[50] whereas others may permit daytime napping[45]), which may differentially affect sleepiness and vigilance. Nevertheless, our SRT protocol, although shorter in duration, was based on guidelines set forth by Spielman et al.,[17] which are commonly implemented in clinical practice and CBT-I trials.

A related point to consider is that in-laboratory SRT, due to increased monitoring and strict scheduling of the sleep window, may have led to greater adherence and possibly enhanced impairment. In practice, it is likely that patients tend to modify the duration and timing of the sleep window (in the home environment) based on individual preferences and ability to function. Nevertheless, it is important to understand the full effect of SRT when patients adhere faithfully to the prescribed program. Convergence of diary-recorded TIB and prescribed sleep window durations (Figure 2) would support this conclusion. It is also worth noting that vigilance was impaired relative to baseline, regardless of whether patients had slept, the previous night, in the laboratory or at home; ruling out the possibility that performance was impaired simply because of the laboratory environment.

In the current study our intention was to isolate SRT, because this intervention has been found to be very effective (when used in single-component interventions), yet difficult to implement,[43] and our early work suggested the possibility of treatment-related impairment.[6,20] It is worth pointing out, however, that stimulus control therapy may also be associated with acute sleep loss, and possible impairment. Future work should attempt to characterize the magnitude and time course of stimulus-control-related impairment (in isolation) as well as in combination with SRT, because many programs combine these two behavioral interventions.[49,53-57]

Finally, because we did not assess performance beyond 3 w (or sleepiness beyond 4 w), during the acute treatment phase, we cannot determine exactly when vigilance started to normalize. From ESS data it would appear that, by w 4, sleepiness was beginning to weaken, but future work should profile daytime performance (including objective measures of sleep debt, e.g., Multiple Sleep Latency Test) for several weeks beyond active treatment/monitoring. The negative effects were most pronounced in our study within the first 3 w but it is worth noting that CBT-I and classic SRT, according to Spielman et al.,[17] often last for 6-8 w, reinforcing the need to extend measurement beyond the 3-4 w in our trial.

Clinical Implications

We think it reasonable, even mandatory, to reflect on what might be the clinical impact of our results. Assuming there is a "necessary pain to achieve gain" with SRT, clinicians should emphasize that CBT-I may negatively affect vigilance levels, and those that are identified as excessively sleepy, pretreatment, or appear to report gross subjective-objective sleep discrepancies, should be assigned a more liberal sleep window. There currently exists variation in the minimum TIB sleep window used in SRT, as well as variation in time in bed calculation and

titation method.[18] The field should aim to reach a consensus on what is the recommended SRT protocol as well as any required modifications for specific populations (e.g., cancer, major depression, bipolar disorder, comorbid chronic pain).[55,58] Consensus should be guided by experimental manipulations, which are needed to reveal treatment mechanisms[6,20] and to provide empirical data on the 'dose' of sleep restriction required to bring about treatment response. Related to this, the suitability and feasibility of using objective measures to guide sleep window generation and titration should also be considered.

Finally, we realize that some laboratories and therapists set a minimum TIB as low as 4.5 h.[17,58] Indeed, had we set this as our minimum TIB, three participants would have been assigned a 4.5-h sleep window and another patient 4.75 h. It remains possible that minimum TIB as low as 4.5 h may lead to impairment greater than that observed in the current study. Going forward, the standardization of SRT procedures, often regarded as the most effective ingredient of CBT-I interventions, should be considered a research and clinical priority for Behavioral Sleep Medicine specialists.

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DISCLOSURE STATEMENT

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