Published in final edited form as:

J Sleep Res. 2013 June ; 22(3): 266–272. doi:10.1111/jsr.12024.

Ecological momentary assessment of daytime symptoms during Sleep Restriction Therapy for insomnia

Christopher B. Miller, BSc^{1,2,3}, Simon D. Kyle, PhD^{1,2}, Nathaniel S. Marshall, PhD^{3,4}, and Colin A. Espie, PhD^{1,2}

¹University of Glasgow Sleep Centre

²Institute of Neuroscience & Psychology, University of Glasgow

³Centre for Integrated Research and Understanding of Sleep (CIRUS), University of Sydney

⁴Sydney Nursing School, University of Sydney

Abstract

This study profiles changes in self-reported daytime functioning during Sleep Restriction Therapy (SRT) for insomnia. Ecological momentary assessment captured point-in-time symptomatology to map the time course of symptoms. We hypothesised a deterioration (week 1) followed by improvements at week 3 of therapy relative to baseline. Nine patients with Psychophysiological Insomnia completed the Daytime Insomnia Symptom Scale (DISS) at risetime, 12:00hr, 18:00hr, and bedtime, for 1 week before and 3 weeks during SRT. Four validated factors from the DISS were analysed (Alert cognition, Positive mood, Negative mood, and Sleepiness / Fatigue) across 28 days yielding 17,170 data points. Factors evaluated week (baseline v. weeks 1 and 3) and time of day symptomatology. Insomnia Severity Index scores decreased significantly pre-to-post treatment (mean = 18 v. 7). Reflecting acute effects of SRT, significant differences were found for all factors, except Negative mood, between baseline and week 1 of SRT, suggesting adverse effects. By week 3, Sleepiness / Fatigue and Negative mood decreased significantly compared to baseline, and Positive mood showed a trend towards improvement ($p = .06$). Sleepiness / Fatigue displayed a significant week $\times$ time of day interaction, explained by a reduction in Sleepiness / Fatigue at every daytime assessment point (except bedtime, which remained high). A significant interaction for Alert cognition was associated with reduction in alertness at Bedtime by week 3, and an increase in alertness at Risetime; suggesting, SRT not only improves sleep, but moderates alertness and sleepiness in therapeutic ways. Initial SRT is associated with an increase in Sleepiness / Fatigue and a decrease in Alert cognition.

Keywords

Insomnia; Treatment; Sleep Restriction Therapy; Ecological Momentary Assessment; Daytime Functioning; Cognition

Corresponding Author: Christopher B. Miller, Woolcock Institute of Medical Research, University of Sydney, Sydney, Australia, Postal address: PO Box M77, Missenden Road, NSW, 2050, Call: +61 (2)9114 0451; Fax: +61 (2) 9114 0014, chris.miller@sydney.edu.au.

Disclosures/ Conflict of Interest:

All Authors have no conflicts of interest.

1. Introduction

Insomnia is a highly prevalent and costly disorder (Morin et al., 2009). It is associated with workplace deficits, increased health risks and health care utilisation (Siebern and Manber, 2010). Further, health-related quality of life has been found to be impaired and it is thought that this drives treatment-seeking behaviour (Kyle et al., 2010).

One of the main effective treatments for Insomnia is Cognitive Behavioural Therapy (CBT-I) (Morin et al., 2006, Smith et al., 2002). Sleep Restriction Therapy (SRT) has been widely utilised within CBT-I (Edinger and Means, 2005). Indeed, it is nearly always included in single or brief interventions to treat insomnia (Edinger and Means, 2005, Buysse et al., 2011). Anecdotally, it is believed to be one of the most active components within CBT-I. Despite this, our current understanding of how and why this potent and difficult behavioural intervention may work is poorly understood (Morgenthaler et al., 2006). Further, adherence and the perceived benefits of this therapy have previously been linked to a positive treatment response (Harvey et al., 2002). Yet, it may also cause some adverse effects (Kyle et al., 2011). In order to further understand the daytime impact, mechanisms, and adverse effects of this brief behavioural therapy, novel measurement approaches must be implemented.

Ecological Momentary Assessment (EMA) is a methodology which allows participants to report aspects of behaviour prospectively and repeatedly in many conditions and environments (Shiffman et al., 2008). It permits reporting of ecologically valid symptoms within the context of their own environment (Buysse et al., 2007), and so may be useful to profile the daytime impact, potential mechanisms of action, and adverse effects of standalone SRT on a day-by-day basis through the course of treatment.

The aim of this study was to profile self-report daytime functioning over the course of three weeks of SRT for Insomnia. We hypothesised that daytime levels of Alert cognition, Sleepiness / Fatigue, Positive and Negative mood, from the Daytime Insomnia Symptom Scale (DISS; Buysse et al., 2007) would initially deteriorate in line with an acute restriction of sleep opportunity, but then improve beyond baseline levels through SRT in line with a treatment response.

2. Methods

Nine participants who responded to advertisements were recruited from the general population and were screened for Psychophysiological Insomnia (Edinger et al., 2004). Participants slept for two nights in the University of Glasgow Sleep Centre, Scotland, prior to study enrolment as part of a concurrent insomnia trial (grant # R01MH077901). Each subject gave informed consent to participate in the study.

Two nights of polysomnography (PSG) were used to exclude sleep-related co-morbidities and to acquire a baseline assessment of sleep. A standard PSG montage was used, involving Electroencephalographic (EEG: Fp1 (neutral), C3, P3 (reference), O1, Fpz, Fz, Cz, Pz, Oz, F4, C4), Electro-oculographic (EOG: horizontal and vertical) and Electromyographic (submental) recordings. On night 1, 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% inter-scorer reliability) according to criteria by Rechtschaffen and Kales (1968). For study inclusion, patients were required to have an Apnoea Hypopnoea Index (AHI) and Periodic Limb Movements of Sleep (PLMS) index < 10. This study was reviewed and approved by the West of Scotland NHS research ethics committee (protocol #10/SO701/85).

SRT comprised one, 40-minute face-to-face session, using Spielman et al's., (1987) instructions, with minimum time in bed set at five hours. The intervention was standardised and supported by a brief set of digital slides (eight in total) and an information manual (11 pages; in plain English). Two further in-person sessions and two telephone calls (each 5–10 minutes) were provided to review sleep efficiency and titrate the sleep window. For titration, time in bed was modified on a weekly basis. Changes were made to time in bed incrementally, depending upon the achievement of "good sleep efficiency"; 85–89% no change, 90% increase by 15 minutes, less than 85% decrease by 15 minutes (cf. Spielman et al., 1987). Time out of bed was rarely changed. In nearly all cases time to bed was modified in line with Spielman et al's., (1987) initial instructions (see Table 1). No other CBT-I components were addressed. The experimenters (SDK, CBM) acted as therapists in this study. Home adherence to SRT was monitored via subjective sleep diary (throughout therapy), and also via a specific sleep restriction adherence scale (SRAS; Kyle & Crawford, unpublished), completed on a week-by-week basis. The SRAS is loosely based on the Medical Outcomes Study general adherence scale (MOS-A; Kravitz et al., 1993), but is modified to probe adherence to different aspects of SRT, through five specific questions (e.g. Item 4: "I got up at my calculated 'rising time' on weekdays..." / Response selection: 1= 'None of the time'; 2= 'A little of the time'; 3= 'Some of the time'; 4= 'A good bit of the time'; 5= 'Most of the time'; 6= 'All of the time'). Possible total SRAS scores range from 5–30, with higher scores being indicative of greater levels of adherence. Preliminary (unpublished) psychometric evaluation of the SRAS, with 42 insomnia patients undergoing SRT, reveals high levels of internal consistency (Cronbach's $\alpha = .92$; range of item-deletion $\alpha = .89-.93$, mean $\alpha = 0.91$). The Insomnia Severity Index (ISI) (Morin, 1993) was completed before and after therapy to monitor more global effects of treatment. Pairwise comparisons were used to evaluate differences.

Participants completed paper-based versions of the DISS at 4 assessment points per day (risetime, 12:00hr, 18:00hr, and bedtime) for 1 week before the SRT intervention (baseline) and for 3 weeks during the intervention (weeks: 1, 2, and 3), representing the acute treatment phase (See Figure 1). The DISS consists of 20 visual analogue scales (ranging from 0 to 100 on a 100 mm line). The participant is asked to mark along the line where they believe to agree or disagree with each of the twenty statements at the specified moment in time. For example, question one asks; "How alert do you feel" with "Very little" at the extreme left of the 100 mm line and "Very much" at the right.

Eighteen of the 20 items were utilized, forming 4 previously validated factors; Sleepiness / Fatigue (containing adjectives; Sleepy, Fatigued, and Exhausted), Negative mood (Anxious, Stressed, Tense, Sad, & Irritable), Positive mood (Relaxed, Energetic, Calm, Happy, & Efficient), and Alert cognition (Forgetful, Clear-headed, Concentrate, Effort, & Alert) (see Buysse et al., 2007). In order to improve adherence to this procedure, participants were offered SMS text message reminders to their mobile phone. Two participants opted to receive these at the 12:00hr and 18:00hr time points only.

A linear mixed model analysis using SPSS software (SPSS IBM v 19.0.0, USA) was implemented for each factor. Fixed effects included: week (B, 1, 2, 3) of the intervention, and for time of day of DISS completion (risetime, 12:00hr, 18:00hr, and bedtime). Random effects were run to account for between-subject variation. Only random effects were included for the intercepts. Uncorrected Pairwise comparisons based on estimated marginal means were implemented to investigate the primary outcomes.

3. Results

In total, 9 patients (6 females; mean age = 46.4yr, range = 34–58) thoroughly screened for Psychophysiological Insomnia (Edinger et al., 2004) completed this study. No participants were taking prescribed sleep promoting hypnotics during the course of this study.

Confirming the expected therapeutic benefits of SRT, sleep diary data reveals significant and robust improvements, baseline to week 4, in both sleep efficiency (73% v. 93%; $p < .01$) and sleep quality ratings (1.8 v. 2.3; $p < .01$). Significant pre-to-post reductions ($p < .001$) were found for; sleep onset latency (33.7 v. 10.7 minutes), time awake after sleep onset (58.7 v. 15), number of awakenings during the night (2.5 v. 1.6), and total time spent in bed (489 v. 367). Total sleep time marginally reduced (357 v. 340); this was not significant ($p > .05$). A significant reduction was found for both total sleep time (357 v. 285; $p < .001$) and time in bed (489 v. 355; $p < .001$) between baseline and week 1 of SRT (see Table 1). ISI scores decreased significantly at post treatment ($M = 18$ (5) v. 7 (5), $p < .05$). The SRT self-report adherence mean scores remained high across the weeks of therapy (week; 1 = 26.1 (3.5), 2 = 24.0 (3.8), 3 = 25.1 (1.3), 4 = 23.1 (6.1); with a range of 10–30.

Turning to the primary purpose of the study, the completion rate for the DISS questionnaire throughout the 4 weeks of data collection was highly satisfactory at 94.6% (ranging from 76% to 100%), yielding 19,075 data points out of a possible 20,160. Each participant was asked to complete the 20 item DISS at 4 times per day everyday for 4 weeks (B, 1, 2, 3). For the primary analysis 18 questions evaluated 4 dimensions of interest; Sleepiness / Fatigue, Alert cognition, Positive and Negative mood. This resulted in 17,170 data points (out of a possible 18,144). The following sections report findings.

In order to firstly evaluate differences for time of questionnaire completion, we looked to evaluate potential daytime patterns to each of the four factors by analyzing the main effect of time of day. Significant differences were found across the 4 daytime assessment points for 3 factors; Sleepiness / Fatigue, Alert cognition, Positive mood [$F(3, 930)$, Sleepiness / Fatigue = 63.97, Alert cognition = 82.93, Positive mood = 42.28]; (all $p < .001$). Negative mood displayed no diurnal change [$F(3, 930) = 0.48$]; ($p = .699$).

To test the initial hypothesis for a deterioration in symptoms at week 1 of SRT, main effects were analyzed for week, yielding significant results for all 4 factors [$F(3, 930)$, Sleepiness / fatigue = 14.30, Alert cognition = 6.54, Negative mood = 7.75, Positive mood = 6.81]; (all $p < .001$). We then compared baseline to week 1 of SRT for all factors. Pairwise comparisons revealed a significant increase for Sleepiness / Fatigue ($p < .01$), a significant decrease for Alert cognition ($p < .001$), and Positive mood ($p < .05$), and no change for Negative mood. This deterioration in symptoms at week 1 was in line with our initial hypothesis regarding early adverse effects due to acute sleep restriction as part of therapy. To test the second part of our hypothesis (that daytime insomnia symptoms would reduce through SRT) we evaluated differences between baseline and week 3. Consistent with expectation, a significant decrease ($p < .001$) was found for Sleepiness / Fatigue and Negative mood, with a trend for improvement in Positive mood ($p = .06$). Alert cognition, however, was not significantly different (see Table 1).

Next, a time of day $\times$ week interaction was used to examine potential diurnal changes in the daytime assessment of symptoms through SRT between baseline and week 3. Sleepiness / Fatigue [$F(9, 930) = 2.54$]; ($p < .01$), and Alert cognition [$F(9, 930) = 2.42$]; ($p < .05$), displayed significant interactions. This was non-significant for Positive [$F(9, 930) = 1.24$]; ($p = .266$) and Negative mood [$F(9, 930) = 0.36$]; ($p = .956$). Specifically, Pairwise comparisons revealed a decrease at week 3 compared to baseline for Sleepiness / Fatigue at the risetime (61.9, 95% CI [51.6, 72.2] v. 52.2, 95% CI [42.0, 62.5]; $p < .01$), 12:00hr (47.9,

95% CI [37.6, 58.2] v. 37.0, 95% CI [26.7, 47.3]; $p < .01$), and 18:00hr (48.3, CI [38.1, 58.6] v. 39.6, CI [29.3, 49.9]; $p < .05$), time points. The bedtime time point increased but not significantly ($p = .172$). For Alert cognition, comparisons revealed a significant increase for risetime (42.2, CI [33.8, 50.5] v. 48.5, CI [40.2, 56.7]; $p < .05$) and a significant decrease at the bedtime assessment point (49.4, CI [41.1, 57.7] v. 40.3, CI [32.0, 48.6]; $p < .01$) at week 3 of SRT. With no significant differences for the 12:00hr and 18:00hr time points (see Figure 2).

4. Discussion

As a prelude to discussing the impact of SRT on daytime variables, it is important to first note the following about this exploratory treatment study; participants adhered to the SRT instructions (as evaluated by the SRT adherence scale), SRT was effective in improving sleep through a reduction in insomnia severity, and the completion rate for the DISS questionnaire was high.

Our primary aim was to examine self-report daytime functioning during the acute course of SRT and to evaluate any initial impairment compared to baseline. Initial implementation of SRT led to significant elevations in Sleepiness / Fatigue and significant reductions in Alert cognition and Positive mood (week 1 compared to baseline), suggesting a general deterioration in symptoms in line with previously observed adverse effect data (Kyle et al., 2011). Negative mood remained unchanged. Our data suggest a reduction of symptoms by week 3 of SRT compared to baseline, with Sleepiness / Fatigue and Negative mood both reducing significantly. Specifically, Sleepiness / Fatigue reduced at risetime, 12:00hr, and 18:00hr, but the bedtime assessment increased (but not significantly). Alert cognition did not change overall but instead evidenced changes at specific time-points; increasing at risetime and decreasing at bedtime (both significantly), suggesting improvements due to SRT (see Figure 2).

Modification in daytime 'Alert cognition' may suggest a potential mechanism by which SRT exerts its therapeutic effect. That is, SRT may help re-structure cognitive arousal, inhibiting it at sleep-onset (to permit sleep) and restoring it at risetime (to permit optimum daytime functioning). Interestingly, baseline Alert cognition scores resemble those previously found in insomnia; by week 3 of treatment, however, these scores resemble that of good sleeping controls (cf. Buysse et al., 2007). Specific testing of this normalisation of Alert cognition at risetime and bedtime may help uncover the role of cognitive arousal in Insomnia in response to effective behavioural treatment. Further trials are required with untreated waitlist controls in order to fully understand if these changes are related to SRT.

Further, the increase in the factor Sleepiness / Fatigue at bedtime at week 3 of SRT is indicative of heightened sleep pressure (reflected, also, in reduced sleep latencies). This may help to overcome cognitive arousal (as previously described in the reduction in Alert cognition), suggesting a validation of alterations to the sleep experience and cognitive arousal facilitating the initiation of sleep. This has previously been postulated as a mechanism of action within SRT (Spielman et al., 1987, Pigeon and Perlis, 2006, Kyle et al., 2011). Future studies, with larger samples and in the context of a more controlled design, should assess whether changes in Sleepiness / Fatigue relate to changes in cognitive arousal. As for the two affect factors; Positive mood increased overall through SRT (but not significantly), with a slight trend for higher scores at risetime. Negative mood displayed no daytime differences (unlike the other 3 factors). This may be attributable to these factors all assessing the same fundamental construct, a lack of power to test this hypothesis, or no dysphoric symptoms within the subject group. Of note, Negative mood did reduce

significantly at week 3 compared to baseline (the mean difference was only 3.6; see Figure 2).

This is the first study to map changes in daytime functioning, using an EMA micro approach as opposed to a macro between subjects approach through brief SRT for Psychophysiological Insomnia. Previously, researchers have focused on differences between groups of insomniacs and good sleepers in order to profile differences in daytime functioning (Levitt et al., 2004, Buysse et al., 2007). Two studies have validated the use of the DISS via EMA methods in insomnia and healthy good sleepers, but not through treatment. It is, however, difficult to compare the present study findings with that of previous studies as these initially used a non-validated version of the DISS (Levitt et al., 2004) and also solely reported factor scores for the groups of insomniacs and good sleeping controls (Buysse et al., 2007). One other previous study has attempted to compare the differences between good-sleeping control participants and insomniacs via ambulatory assessment (Varkevisser et al., 2007). In this study, the authors attempted to probe differences in performance and well-being (concentration, fatigue, mood, and sleepiness). No differences were found on the performance measures but they did discover that subjective wellbeing was compromised in insomniacs compared to controls. Further trials using EMA methods are now required to evaluate the role of daytime symptoms in response to other single components of CBT for insomnia.

It is also important to note that although the term 'sleep restriction' (SR) is more widely used in sleep science - usually in studies where healthy participants are systematically denied a predefined period of sleep to investigate the effects of sleep loss upon cognitive and physiological functioning (Dinges et al., 1997, Van Dongen et al., 2003) - the context between these protocols and the present study, where SRT is used therapeutically, differ greatly. Nevertheless, some of the effects of SR in these carefully conducted laboratory studies are what have prompted our taking a careful look at the potential adverse effects of SRT insomnia patients. Results suggest that total sleep time reduced significantly between baseline and week 1, but not between baseline and week 4 of SRT. Therefore, the changes that we see at week 1 in the three factors (Alert cognition, Sleepiness / Fatigue, and Positive mood) may be attributable to changes in perceived sleep time. It is possible that participants adapted over time to the initial effects of this type of sleep restriction by week 3 (Belenky et al., 2003). Yet, this does not fully explain the significant reduction in Sleepiness / Fatigue and Negative mood at week 3 compared to baseline levels. Further controlled studies are required to evaluate acute sleep restriction in patients with insomnia compared to healthy good-sleeping controls.

A number of limitations regarding this study must be addressed. A regression to the mean may explain the reduction in daytime impairments in Alert cognition and Sleepiness / Fatigue. However, an initial decrease in Alert cognition and an elevation in Sleepiness / Fatigue at week 1 and the subsequent reversal of this at week 3 suggests a contrary conclusion. The experimenters (SDK, CBM) also acted as therapists in this study. Therefore, it is difficult to rule out experimenter, performance, and demand effects. It could be argued that a lack of participants in this study represents low statistical power and increases the probability of obtaining a Type II statistical error. Also, the precision of estimating unknown parameters may be compromised by the lack of statistical power. Participants were asked to repeatedly respond to items on a visual analogue scale, a fine-grained continuous outcome measure with increased statistical power compared to more standard ordinal measures. Further, we were not able to determine objective adherence to EMA questionnaire completion. Previously, hand-held computers with time-stamped recordings have been used to determine exactly when a participant completes a questionnaire (Buysse et al., 2007). Use

of such devices may be more reliable but they do also bring about difficulties such as battery life and learning how to use them.

In summary, results demonstrate that the DISS, completed via EMA, can profile changes in self-report daytime functioning through SRT for Psychophysiological Insomnia. Changes at risetime and bedtime for Alert cognition and Sleepiness / Fatigue seem like candidates for further study as mechanisms of action in treatment response; potentially reflecting changes to the input and output of the sleep homeostat and cognitive arousal. Further objective assessments of insomnia treatment via repeated-measures are required in order to elucidate the role of sleep and arousal in response to SRT for insomnia. Results may help uncover mechanisms of action within single components of CBT-I. Those delivering SRT as part of CBT-I should be mindful of an initial deterioration in symptoms at week 1 (cf. Kyle et al., 2011). It may be necessary to tailor SRT to suit the needs of the patient in order to reassure and overcome the initial negative symptoms of therapy. Such understanding may help to safely disseminate this treatment option.

Acknowledgments

This research was supported by:

The Chief Scientist Office (Scotland, UK),

National Health and Medical Research Council (NHMRC, Australia), Centre for Integrated Research and Understanding of Sleep (CIRUS),

The Sackler Institute of Psychobiological Research (University of Glasgow Sleep Centre)

Funding Source:

The Chief Scientist Office (Scotland) Grant # CZG/2/503

NHMRC Centre for Integrated Research and Understanding of Sleep (CIRUS) Grant # 571421

National Institutes of Health (NIH) Grant # R01MH077901

References

- Belenky G, Wesensten NJ, Thorne DR, et al. Patterns of performance degradation and restoration during sleep restriction and subsequent recovery: a sleep dose-response study. *J Sleep Res.* 2003; 12:1–12. [PubMed: 12603781]
- Buysse DJ, Germain A, Moul DE, et al. Efficacy of Brief Behavioral Treatment for Chronic Insomnia in Older Adults. *Arch Intern Med.* 2011; 171:887–895. [PubMed: 21263078]
- Buysse DJ, Thompson W, Scott J, et al. Daytime symptoms in primary insomnia: A prospective analysis using ecological momentary assessment. *Sleep Med.* 2007; 8:198–208. [PubMed: 17368098]
- Dinges DF, Pack F, Williams K, et al. Cumulative sleepiness, mood disturbance, and psychomotor vigilance performance decrements during a week of sleep restricted to 4–5 hours per night. *Sleep.* 1997; 20:267–277. [PubMed: 9231952]
- Edinger JD, Bonnet MH, Bootzin RR, et al. Derivation of research diagnostic criteria for insomnia: report of an American Academy of Sleep Medicine Work Group. *Sleep.* 2004; 27:1567–1596. [PubMed: 15683149]
- Edinger JD, Means MK. Cognitive-behavioral therapy for primary insomnia. *Clinical Psychology Review.* 2005; 25:539–558. [PubMed: 15951083]
- Harvey L, Inglis SJ, Espie CA. Insomniacs' reported use of CBT components and relationship to long-term clinical outcome. *Behav Res Ther.* 2002; 40:75–83. [PubMed: 11762429]

- Kravitz RL, Hays RD, Sherbourne CD, et al. Recall of recommendations and adherence to advice among patients with chronic medical conditions. *Arch Intern Med.* 1993; 153:1869–1878. [PubMed: 8250648]
- Kyle SD, Morgan K, Espie CA. Insomnia and health-related quality of life. *Sleep Med Rev.* 2010; 14:69–82. [PubMed: 19962922]
- Kyle SD, Morgan K, Spiegelhalter K, Espie CA. No pain, no gain: an exploratory within-subjects mixed-methods evaluation of the patient experience of sleep restriction therapy (SRT) for insomnia. *Sleep Med.* 2011; 12:735–747. [PubMed: 21907616]
- Levitt H, Wood A, Moul DE, et al. A pilot study of subjective daytime alertness and mood in primary insomnia participants using ecological momentary assessment. *Behav Sleep Med.* 2004; 2:113–131. [PubMed: 15600228]
- Morgenthaler T, Kramer M, Alessi C, et al. Practice parameters for the psychological and behavioral treatment of insomnia: An update. *An American Academy of Sleep Medicine Report.* *Sleep.* 2006; 29:1415–1419. [PubMed: 17162987]
- Morin, CM. *Insomnia: Psychological Assessment and Management.* New York, NY: Guilford Press; 1993.
- Morin CM, Bootzin RR, Buysse DJ, Edinger JD, Espie CA, Lichstein KL. Psychological and behavioral treatment of insomnia: Update of the recent evidence (1998–2004). *Sleep.* 2006; 29:1398–1414. [PubMed: 17162986]
- Morin CM, Daley M, Leblanc M, Gregoire JP, Savard J. The Economic Burden of Insomnia: Direct and Indirect Costs for Individuals with Insomnia Syndrome, Insomnia Symptoms, and Good Sleepers. *Sleep.* 2009; 32:55–64. [PubMed: 19189779]
- Pigeon WR, Perlis ML. Sleep homeostasis in primary insomnia. *Sleep Med Rev.* 2006; 10:247–254. [PubMed: 16563817]
- Rechtschaffen, A.; Kales, A., editors. *A manual of standardized terminology, techniques and scoring system of sleep stages in human subjects.* Los Angeles: Brain Information Service/Brain Research Institute, University of California; 1968.
- Shiffman S, Stone AA, Hufford MR. Ecological momentary assessment. *Annu Rev Clin Psycho.* 2008; 4:1–32.
- Siebern AT, Manber R. Insomnia and Its Effective Non-pharmacologic Treatment. *The Medical clinics of North America.* 2010; 94:581–591. [PubMed: 20451034]
- Smith MT, Perlis ML, Park A, et al. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. *Am J Psychiatry.* 2002; 159:5–11. [PubMed: 11772681]
- Spielman AJ, Saskin P, Thorpy MJ. Treatment of Chronic Insomnia by Restriction of Time in Bed. *Sleep.* 1987; 10:45–56. [PubMed: 3563247]
- Van Dongen HPA, Maislin G, Mullington JM, et al. The cumulative cost of additional wakefulness: dose-response effects on neurobehavioral functions and sleep physiology from chronic sleep restriction and total sleep deprivation. *Sleep.* 2003; 26:117–126. [PubMed: 12683469]
- Varkevisser M, Van Dongen HPA, Van Amsterdam JGC, Kerkhof GA. Chronic insomnia and daytime functioning: an ambulatory assessment. *Behavioral sleep medicine.* 2007; 5:279–296. [PubMed: 17937583]

<i>Pre-Study Period</i>		<i>← EMA Collection Period →</i>				<i>Post Treatment</i>
<i>PSG 1</i>	<i>PSG 2</i>	Baseline Week	Week 1 SRT	Week 2 SRT	Week 3 SRT	Week 4

Figure 1. Ecological momentary assessment data collection period through brief SRT for insomnia

Participants were sampled from a concurrent insomnia study and underwent two nights of polysomnography (PSG 1 & 2), prior to study enrolment. This was followed by a 4 week ecological momentary assessment (EMA) first through an initial baseline data collection week and then with 3 weeks of sleep restriction therapy (SRT). SRT post treatment outcome measures were assessed at week 4.

Figure 2. Time course of daytime symptoms through baseline and 3 weeks of sleep restriction therapy

Graphs of each of the 4 factors of the Daytime Insomnia Symptom Scale (DISS); Alert cognition, Sleepiness / Fatigue, Positive mood, and Negative mood for all (n=9) participants through the four weeks of ecological momentary assessment. Mean visual analogue scores (0–100) are represented on the y-axis. Week of data collection (baseline - week 3) is represented along the x-axis. Each line represents a daytime assessment time point; Risetime (blue), 12:00hr (black), 18:00hr (red), and Bedtime (green), for the specific factor through baseline and weeks 1–3 of Sleep Restriction Therapy (SRT). Error bars represent one standard error of the mean.

Table 1

Mean sleep diary data, Sleep Restriction Adherence Scale (SRAS), and Daytime Insomnia Symptom Scale (DISS) factor scores at baseline and through Sleep Restriction Therapy

The top half of the table describes the mean sleep diary data with standard deviations (minutes) for total sleep time and prescribed time in bed (weeks 1–4 of SRT), the mean and standard deviations of Sleep Restriction Adherence Scale (SRAS) scores (for weeks 1–4 of SRT), and the mean (with 95% CI) for the 4 daytime symptoms variables over the 4 weeks of observation. The bottom half of the table tests for differences in total sleep time, time in bed and SRAS results.

	Mean and Standard Deviations			Mean and 95% Confidence Intervals			
	Prescribed Time in bed (apart from baseline)	Total Sleep Time	SRAS	Alert Cognition	Sleepiness / Fatigue	Negative Mood	Positive Mood
Baseline Week	489 (49.4)	357 (35.8)		53.0 [45.3, 60.8]	54.5 [44.8, 64.2]	26.1 [15.8, 36.4]	55.0 [48.0, 61.9]
Week 1	355 (24.5)	285 (33.1)	26.1 (3.5)	47.7 [39.9, 55.6]	59.2 [49.5, 68.9]	27.4 [17.1, 37.7]	52.0 [45.1, 58.9]
Week 2	350 (21.2)	311 (27.3)	24.0 (3.8)	51.8 [42.2, 61.7]	52.0 [42.2, 61.7]	25.9 [15.6, 36.2]	54.5 [47.6, 61.4]
Week 3	362 (20.5)	323 (27.4)	25.1 (1.3)	53.0 [45.2, 60.8]	48.4 [38.7, 58.1]	22.5 [12.2, 32.8]	57.1 [50.2, 64.1]
Week 4	367 (21.3)	340 (24.8)	23.1 (6.1)				
<i>Paired Differences Testing</i>							
	Mean and Standard Errors			Mean Difference and 95% Confidence Intervals			
Baseline v. Week 1	489 (16.5) v. 355 (8.7) ***	357 (11.9) v. 285 (11.0) ***		5.3 [2.5, 8.0] ***	4.7 [8.1, 1.4] **	1.3 [3.4, 0.8]	3.0 [0.7, 5.2] *
Baseline (Week 1 for SRAS) v. Week 3 (Week 4 for TIB, TST & SRAS)	489 (16.5) v. 367 (7.5) ***	357 (11.9) v. 340 (9.4) ***	26.1 (1.2) v. 23.1 (2.2)	0.0 [2.8, 2.7]	6.1 [2.8, 9.6] ***	3.6 [1.5, 5.7] ***	2.1 [4.5, 0.94]

This also tests our specific hypotheses about a transient worsening of mood in response to SRT at week 1 followed by an overall improvement over the course of treatment by week 3 (* $p < .05$, ** $p < .01$, *** $p < .001$).