# Isolating the role of time in bed restriction in the treatment of insomnia: a randomised, controlled, dismantling trial comparing sleep restriction therapy with time in bed regularisation

Leonie Franziska Maurer[1], Colin Alexander Espie[1], Ximena Omlin[1], Matthew James Reid[1], Rachel Sharman[1], Dimitri Gavriloff[1], Richard Emsley[2], Simon David Kyle[1*]

1. Sleep and Circadian Neuroscience Institute, Nuffield Department of Clinical Neurosciences, University of Oxford, UK
2. Department of Biostatistics and Health Informatics, Institute of Psychiatry, Psychology & Neuroscience, King's College London, London, UK

**Address of correspondence:**

*Simon D. Kyle, PhD

Sleep and Circadian Neuroscience Institute, Nuffield Department of Clinical Neurosciences, University of Oxford

Dunn School of Pathology, South Parks Road,

Oxford, OX1 3RE

simon.kyle@ndcn.ox.ac.uk

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## Abstract

**Study Objectives**: Sleep restriction therapy (SRT) is one of the most effective treatments for insomnia. Restriction of time in bed (TIB) is assumed to be the central mechanism through which SRT improves sleep consolidation and reduces insomnia symptoms. This hypothesis has never been directly tested. We designed a randomised, controlled, dismantling trial in order to isolate the role of TIB restriction in driving both clinical and polysomnographic sleep outcomes.

**Methods:** Participants aged 25-55 who met DSM-5 diagnostic criteria for insomnia disorder were block-randomised (1:1) to four weeks of SRT or time in bed regularisation (TBR), a treatment that involves prescription of a regular but not reduced time in bed. The primary outcome was assessed with the insomnia severity index (ISI) at baseline, 4-, and 12-weeks post-randomisation. Secondary outcomes included sleep continuity (assessed via polysomnography, actigraphy, and diary) and quality of life. We performed intention-to treat analyses using linear mixed models.

**Results:** Fifty-six participants (39 females, mean age=40.78±9.08) were assigned to SRT (n=27) or TBR (n=29). Daily monitoring of sleep via diaries and actigraphy confirmed large group differences in TIB (*d* range = 1.63-1.98). At 4-weeks post-randomisation the adjusted mean difference for the ISI was -4.49 (*d*=-1.40) and -4.35 at 12-weeks (*d*=-1.36), indicating that the SRT group reported reduced insomnia severity relative to TBR. Robust treatment effects in favour of SRT were also found for objective and self-reported sleep continuity variables (*d* range=0.40-0.92) and sleep-related quality of life (*d*=1.29).

**Conclusions:** For the first time we demonstrate that time in bed restriction is superior to the regularisation of time in bed on its own. Our results underscore the centrality of the restriction component in reducing insomnia symptoms and consolidating sleep.


**Keywords:** Insomnia, SRT, behavioural treatment, mechanisms

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**Clinical Trial:** The MARTINI trial is registered with ISRCTN (ISRCTN10974094) at: https://doi.org/10.1186/ISRCTN10974094

**Statement of Significance**

We sought to investigate the mechanistic foundation of one of the most effective treatments for insomnia – sleep restriction therapy (SRT). This behavioural treatment, introduced in the 1980s, involves restricting and regularising a patient's time in bed in order to prime and strengthen sleep-wake biology. We designed a study in order to isolate the role of the restriction element relative to consistent bed- and rise-times in driving both clinical and polysomnography-based sleep outcomes; a hitherto unanswered but key question. In a randomised controlled trial, we show that participants allocated to SRT demonstrate greater improvements in sleep and quality of life relative to those who receive a control treatment involving the implementation of a regular – but not restricted – time in bed.

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## Introduction

Insomnia is one of the most prevalent sleep and psychiatric disorders world-wide, affecting approximately 10% of the adult population[1-3]. This burdensome condition degrades quality of life[4] and confers risk for the development of depression, anxiety and substance misuse[5]. Sleep restriction therapy (SRT) is an effective treatment for insomnia, as both standalone therapy and as part of cognitive-behavioural treatment[6,7]; however, its mechanisms are not well understood[8]. SRT involves prescribing a restricted time in bed (TIB) that matches patient-reported sleep duration[9]. While total sleep time is not directly targeted, the prescribed TIB often engenders mild sleep deprivation[10] because patients 1) tend to underestimate sleep time at baseline[11], and 2) may continue to experience difficulties with sleep initiation and maintenance during early implementation. Over the course of treatment, TIB is systematically expanded conferring increased sleep opportunity[9].

When Spielman, Saskin and Thorpy first described and tested the implementation of SRT in 1987 they hypothesised that the initial sleep deprivation, caused by the restriction component, is essential because of its effects on sleep consolidation and arousal[12]. This proposition is supported by models of sleep-wake regulation which demonstrate that sleep loss strengthens homeostatic sleep drive[13,14]. Enhanced sleep drive may help diminish cognitive and physiological arousal[15], obviate sleep effort[16] and enhance the consolidation and predictability of sleep[8,17]. While many studies have shown that TIB[8] and sleep time[10,18] are reduced during SRT, only two correlational studies have demonstrated that sleep loss (indexed by increased self-reported sleepiness) is associated with sleep improvements[19,20].

Importantly, Spielman and colleagues also emphasised that the regularisation of bed and rise times, intrinsic to SRT, may be important for therapeutic effects[12,17]. During the intervention, patients are asked to keep a fixed bed and rise time seven nights a week with only small

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variations over successive weeks of treatment. Consequently, studies have revealed that the implementation of SRT is associated with reduced TIB variability[21,22]. Moreover, Riedel and Lichstein showed that greater consistency of time spent in bed and a more consistent rise time, *but not the degree of bedtime reduction*, were correlated with better treatment outcome[22]. This suggests that regularity and not restriction of sleep opportunity *per se* might be the key factor. Indeed studies in the student population have shown that a) frequency of irregular bedtimes is associated with worse sleep quality[23] and b) regularisation of bed times can reduce sleep onset latency, daytime sleepiness and increase sleep efficiency[24].

While theoretical models emphasise restriction of TIB, no causal test has been conducted despite important clinical and mechanistic implications. To test the centrality of the restriction component, we conducted a parallel, randomised, controlled trial comparing SRT (+ sleep hygiene) with a dismantled version of SRT, referred to as Time in Bed Regularisation (TBR; + sleep hygiene).

The primary and secondary clinical hypotheses for the trial were:

1. SRT reduces self-reported insomnia severity relative to TBR (assessed at 4- and 12-weeks)
2. SRT reduces wake-time after sleep onset (WASO) and sleep onset latency (SOL), and increases sleep efficiency (SE) during the treatment phase (week 1-4) relative to TBR as indicated by sleep diary, actigraphy and polysomnography (PSG; assessed at the end of week 1 and 4)
3. SRT improves sleep-related quality of life relative to TBR (assessed at 4- and 12-weeks)
4. SRT reduces self-reported symptoms of depression and anxiety relative to TBR (assessed at 4- and 12-weeks)

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5. SRT reduces self-reported cognitive complaints (assessed at 4- and 12-weeks) relative to TBR.

## Methods

### *Study Design*

The MARTINI (MechAnisms of sleep Restriction Therapy IN Insomnia) trial was a randomised, controlled, mechanistic evaluation of SRT (+ sleep hygiene) versus TBR (+ sleep hygiene). The trial was based on the principles of a dismantling study design, which involves comparing a full treatment protocol to a *dismantled* comparator, whereby one or more aspects of the treatment protocol that are thought to be active change agent(s) are removed[25]. All other aspects of the study design and implementation were held constant across groups, and attempts were made to ensure equivalence across other aspects of care delivery such as therapist time and attention. The study was conducted in Oxfordshire, UK and approved by the Central University Research Ethics Committee of the University of Oxford (CUREC R51331/RE005).

### *Participants*

Participants were recruited between July 2017 and January 2019 in Oxford (UK) and its surrounding area through community advertisement (Facebook, posters, newspaper, and radio). General inclusion criteria were: aged 25-55 years, meeting criteria for DSM-5 chronic insomnia disorder (assessed by the Sleep condition indicator[26] and telephone interview), and mobile phone and internet use. Exclusion criteria were: psychiatric diagnoses other than insomnia; anxiety 'caseness' [scoring >10 on the anxiety sub-scale of the Hospital anxiety and depression scale; HADS[27]]; depression 'caseness' [Patient health questionnaire $\geq$10; PHQ-9[28]]; current alcohol misuse [assessed by items from the Alcohol use disorders identification test, AUDIT[29]]; additional sleep disorders other than insomnia [assessed via the

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sleep disorders screen[30]]; sleep-disruptive medical comorbidity or conditions contraindicated for potential sleep deprivation (e.g. epilepsy); current prescription of medication or psychotherapy for sleep; overnight-, evening-, or rotating shift work; pregnancy; and not living in the area of Oxford. Participants were reimbursed £20 for each completed study phase (baseline, week 1, week 4 and week 12).

***Procedures***

Eligibility assessment was a two-part process, whereby an initial online questionnaire (Qualtrics, Provo, UT) was followed by a semi-structured telephone interview (based on Morin and Espie[31]) to determine inclusion/exclusion. Once deemed eligible, participants were invited for a study induction at the Sleep and Circadian Neuroscience institute (SCNi) to discuss study procedures, obtain written consent and commence the baseline assessment for two weeks (baseline phase). Upon completion of all baseline measurements, participants were randomised and invited for their first treatment session (see below). All participants were instructed to follow their new sleep schedule for four weeks and asked to complete assessments throughout the acute intervention phase and at post-treatment. At twelve weeks post-randomisation, participants received a link to an online-follow-up questionnaire (Qualtrics, Provo, UT).

***Randomisation and Masking***

Participants were randomly assigned (1:1) to either SRT or TBR using an online computer-generated randomisation sequence (www.sealedenvelope.com), stratified by age (25-40 vs 41-55) and sex (female/male), with varying block size (2-6) to maintain concealment of future allocations. The randomisation sequence was kept by a departmental colleague who had no formal involvement with the MARTINI study and no interaction with participants.

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***Interventions***

All participants were informed that they would be randomised to one of two behavioural interventions, both of which are believed to be beneficial for sleep but have never been compared in one study before. For the purposes of trial reporting we refer to the control condition as TBR (time in bed regularisation) but participants were introduced to the name "bedtime consistency therapy" to account for the possibility that they might search for the intervention on the internet and find plausible results. Both interventions were delivered through four weekly 1:1 sessions (see Table 1). During session 1 (~60 mins), the treatment rationale was introduced, and bed and rise-times were selected. Sessions two to four (15 mins each) served to review adherence, side-effects and problems with implementation, and to adjust the sleep window if required. Both interventions, SRT and TBR, followed a set structure for each session, required a similar amount of time with the therapist and were matched as closely as possible with respect to rationale, positioning of side-effects and expectation of benefit.

[INSERT TABLE 1]

Upon completion of all baseline assessments, and following randomisation, participants were invited for their first individual face-to-face treatment session at the SCNi. Treatment content for both groups was delivered by a doctoral researcher (LFM) trained in CBT-I, and under the supervision of experts in behavioural sleep medicine (CAE and SDK). Treatment was delivered according to a manualised protocol, supported by workbooks and personalised treatment slides (PowerPoint, 2013). Both interventions were referred to as 'sleep scheduling' and started with a brief introduction to standard sleep hygiene guidelines, covering caffeine, alcohol, diet, exercise, nicotine, noise, light, comfort, temperature and air quality. Sleep hygiene advice is commonly delivered as part of usual care for insomnia[32] but has not been shown to be an effective monotherapy[33,34]. It was incorporated in order to ensure that both

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groups had equivalent awareness of the effects of lifestyle and bedroom factors on sleep. After setting goals to improve sleep hygiene, the key principles of sleep scheduling were explained and new bed and rise times were derived according to standardised criteria (see Table 2).

[INSERT TABLE 2]

Diaries and sleep efficiency grids were used to calculate and track sleep efficiency on a weekly basis in the SRT group. Similarly, participants in the TBR group were introduced to a bed-time consistency checklist, where they were asked to check each day if their bed and rise time was within 10 minutes of the agreed prescription. During the first week of treatment, participants in both arms received daily check-in text messages to remind them about the agreed sleep schedule and support adherence. One week after the first session, a home visit (session 2) took place to review diaries and sleep efficiency/consistency for the previous week. In the following weeks (2-4), both groups were contacted weekly by telephone (sessions 3 and 4; see Table 2).

To evaluate treatment expectations, all participants completed the credibility/expectancy questionnaire (CEQ)[35,36] at the end of treatment session one, during which the chief therapeutic ingredients were explained. We also audio-recorded the first treatment session for all participants to permit evaluation of treatment fidelity. A random sample of recordings (10% in each arm) was selected for fidelity appraisal by an independent clinical psychologist specialising in behavioural sleep medicine (DG).

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***Measures***

*Primary outcome*

Insomnia severity was assessed with the insomnia severity index (ISI)[37]. The ISI comprises seven items that are rated on a 0–4 scale. The total score ranges from 0 to 28 with a higher score indicating more severe insomnia and a cut-off score of 10 for detecting insomnia cases. The ISI has good internal consistency ($\alpha \geq 0.90$)[38].

*Secondary outcomes*

*Sleep*: Participants completed a sleep diary based on the international consensus format[39]. This was completed each morning and evening for 14 days during baseline and 28 days during the treatment phase. Participants provided estimates for SOL, WASO, SE and total sleep time (TST). To compare sleep variables across a similar number of days, the treatment phase (28 days) was split into two 14-day segments, reflecting early (weeks 1-2) and late treatment (weeks 3-4).

Concurrently, participants wore an actigraph watch (MotionWatch 8, Camntech Ltd.) for six weeks throughout the study. The watch was set to record activity in tri-axial mode and light over 60 second epochs, comparable to recording settings in other sleep studies[40]. Participants were instructed to press a marker button on the watch when initiating sleep. These event markers were used to define sleep periods. In the absence of event markers, a decision was made based on bed and rise times from the sleep diary or by indicators from light and movement measurements. All sleep variables of interest were calculated by the validated in-build algorithm of the MotionWare software 1.1.26[41] and were averaged across 14-day periods.

Objective sleep was recorded with the portable PSG system, SOMNO HD (Somnomedics GmbH, Germany), at participants' homes. The set-up was conducted and recorded according

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to the recommended montage of the American Academy of Sleep Medicine (AASM)[42]. This included six scalp electrodes (F3, F4, C3, C4, O3, O4), one ground electrode (forehead), one scalp reference electrode (CZ) and two reference electrodes placed on each mastoid process (A1, A2). Due to the ambulatory nature of this study, participants indicated sleep intention with a biomarker, five left-right eye movements, to mark lights out and scoring start. Recordings were blinded and scored according to AASM 2017 guidelines[43]. Reliability of sleep scoring was confirmed by an experienced scorer (RS), who double-scored 10% of all recordings (agreement=91.5%). We derived the same sleep continuity variables as described above.

*Quality of life*: Sleep-related quality of life was measured with the Glasgow sleep impact index (GSII)[44]. Three participant generated areas of impairment are ranked in order of concern (1–3; i.e. 1=the most concerning impairment), and then rated on a 0-100 VAS in relation to impact in the past two weeks (0=very bothered, 100=not bothered). Participants re-rated these specified areas of impairment at post-treatment and follow-up. We calculated the average across the three areas of concern with lower scores indicating poorer sleep-related quality of life. The GSII has shown sensitivity to CBT-I interventions[44,45].

*Emotional distress*: We measured symptoms of depression and anxiety with the HADS[46]. The questionnaire consists of 14 items (7 covering depressive symptoms; 7 covering anxiety symptoms), scored 0-3, to give a range between 0-21 with higher scores indicating greater severity. The questionnaire reveals acceptable internal consistencies for both the anxiety ($\alpha$=0.68-0.93) and the depression scale ($\alpha$=0.67-0.90)[27].

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*Cognitive impairment*: Self-reported cognitive complaints were measured by the British Colombia cognitive complaints inventory (BC-CCI)[47]. The questionnaire comprises six questions where each is scored between 0-3, and the total score ranges from 0-18 where higher scores indicate higher cognitive impairment. The measurement reveals good internal consistency ($\alpha \geq 0.82$)[47].

*Treatment expectations*

The credibility/expectancy questionnaire (CEQ) is a scale for measuring treatment expectancy and rationale credibility for use in clinical outcome studies[36]. The questionnaire contains six items (three indexing expectancy, three indexing credibility) divided into two sections that differ in their instructions. The first section of the questionnaire (first four items) is answered in terms of what the participant thinks about the treatment, the second section is answered in terms of what the participant feels about the treatment. The CEQ utilises two different response formats: items 1, 2, 3 and 5, are scored on a 1-9 scale, while items 4 and 6 are scored on 0–100% scale. For analysis, all items were standardised on the same 1-9 scale in order to derive sum scores for expectancy (items 1-3) and credibility (items 4-6) with a possible range from 3 to 27, respectively.

*Treatment fidelity and TIB manipulation check*

We developed a content checklist for both treatments, whereby a maximum score of two was given if the content was fully covered; a score of one if the content was partially covered; and a score of 0 if it was not covered at all. This resulted in a possible range of 0-154 for SRT and 0-138 for TBR. To investigate if participants followed their new sleep schedule as prescribed, we compared the groups on self-reported and actigraphy-derived TIB during the four weeks of intervention. To assess whether both treatment arms implemented more consistent sleep times we calculated within-subject variability (root mean square of the successive

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differences; RMSSD) for diary-reported bed- and rise times, and assessed within-group change from baseline.

*Adverse events measures*

Serious adverse events were defined as death, suicide attempt, admission to hospital, and formal complaints about the intervention and were recorded when a member of the research team was made aware of their occurrence.

We also assessed pre-defined adverse effects using a standardised questionnaire. The side-effects checklist[20] is a 14-item measure which asks participants to answer yes/no questions regarding whether implementing the new sleep schedule resulted in any of the 14 listed side-effects. The principal outcome is number of endorsed symptoms per participant. The scale was administered weekly throughout treatment; participants filled out the paper copy of the checklist in weeks 1 and 4, while they completed it over the phone at weeks 2 and 3.

***Statistical analyses***

Based on meta-analysis of placebo-controlled trials of behavioural insomnia treatment[48], the study was powered to detect a minimum standardised effect size of 0.80 between-groups on the primary outcome (ISI) at post-treatment (week 4), at 5% level of significance with 80% power. Accounting for 10% attrition we sought to recruit 56 participants (28 in each group).

Consistent with CONSORT guidelines on the reporting of RCTs[49], all data from all randomised participants were analysed using the *intention to treat* principle[50]. Summary statistics are presented by intervention group. All analysis was conducted in SPSS.25 (IBM).

A linear mixed effects regression model was fitted for the primary outcome with fixed effects of group and time point. The outcomes at 4- and 12-weeks were included as the response. Following the guidelines from Landau and Everitt[51], we included the centred baseline score

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as a covariate and a participant-specific random intercept to account for repeated measures. The covariance structure for random effects, here participant, was set to variance components (VC) as this is the default for a mixed model with one random effect[52]. An interaction between time point and randomised group was included to estimate treatment effects separately at each time point. Cohen's *d* statistics were calculated as the adjusted treatment effect divided by the baseline standard deviation of the outcome for the combined treatment groups[53]. Primary and secondary outcomes were analysed in the same way. For continuous sleep diary variables, responses were entered as averages of weeks 1-2 (early treatment) and weeks 3-4 (late treatment). Unadjusted means and SDs are also reported. For side-effects, linear mixed models were fitted with number of symptoms at week 1, 2, 3 and 4 included as the response, while effects sizes were calculated as $ES=Diff/((SD^1+SD^2)/2$[53]. Differences in treatment credibility and expectations were analysed using independent t-tests.

## Results

### *Participant characteristics*

Five-hundred-twenty-nine individuals were screened online, 58 were deemed eligible, and 56 were randomised. Main reasons for exclusion were other psychiatric symptoms, additional sleep disorders and not meeting insomnia criteria. See Figure 1 for the CONSORT flow diagram. Session attendance was high in both arms, with just one participant in the SRT arm withdrawing from the trial due to lack of benefit. Hence, 55 participants completed all post-treatment and follow-up assessments.

[INSERT FIGURE 1]

The sample was predominantly middle-aged (mean=40.78±9.19 yrs.; range 26-55), comprised of females (70%), and was university-educated (79%). Consistent with inclusion criteria, ISI scores were in the clinical range (mean=14.90±3.21). Table 3 provides the

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baseline characteristics of the participants recruited into each of the treatment arms. The two groups were well-matched on demographic and clinical outcomes at baseline.

[INSERT TABLE 3]

***Primary outcome***

*Insomnia severity.* Our primary hypothesis was to assess if SRT reduces self-reported insomnia severity relative to TBR. As shown in Table 4 and Figure 2, there were large between-group effects at 4- and 12-weeks post-randomisation. At 4-weeks post randomisation, the estimated adjusted mean difference in outcome was -4.49 (95% CI: -6.17, -2.81; $p$<.001, $d$=-1.40), indicating that participants in the SRT group reported a lower ISI total score than those in the TBR group. Treatment effects were maintained at 12-weeks, illustrated by an adjusted mean difference of -4.35 (95% CI: -6.03, -2.67; $p$<.001, $d$=-1.36).

[INSERT FIGURE 2]

***Secondary outcomes***

*Self-reported sleep continuity.* Between-group comparisons revealed group differences for SOL, WASO and SE at both time points (early and late treatment, see Table 4). Medium effect sizes were found for between-group comparisons of SOL ($p_{\text{early treatment}}$=.007, $d$=-.46; $p_{\text{late treatment}}$=.001, $d$=-.57), favouring the SRT group. Medium effect sizes were observed for WASO at early (WASO: $p$<.001, $d$=-.62) and late treatment (WASO: $p$<.001, $d$=-.69), showing lower WASO in the SRT group. These results were also reflected in group differences for SE ($p_{\text{early treatment}}$<.001, $d$=.60; $p_{\text{late treatment}}$<.001, $d$=.92), revealing higher SE for the SRT group.

*Actigraphy-defined sleep continuity*. Comparable to sleep diary, between-group comparisons revealed group differences for actigraphy-derived SOL, WASO and SE at both time points

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for (early and late treatment, see Table 4). Medium-to-large effect sizes were found for between-group comparisons of SOL ($p_{\text{early treatment}}$=.003, $d$=-.78; $p_{\text{late treatment}}$=.001, $d$=-.87), favouring the SRT group. For WASO, the treatment effects showed medium effect sizes ($p_{\text{early treatment}}$<.001, $d$=-.57; $p_{\text{late treatment}}$<.001, $d$=-.58) in favour of the SRT group. Again, these results were also reflected in group differences for SE ($p_{\text{early treatment}}$=.003, $d$=.40; $p_{\text{late treatment}}$<.001, $d$=.48), revealing higher SE for the SRT group.

*Polysomnographically-defined sleep continuity.* Due to data loss two recordings from two different participants were excluded from analysis of WASO and SE (one baseline [TBR] and one week-1 recording [TBR]). Between-group comparisons (see Table 4) revealed medium-to-large effects for SOL at week 1 ($p$=.041, $d$=-.74) and week 4 ($p$=.091, $d$=-.61), although the latter was not statistically significant. Medium-to-large effects were also found for WASO at week 1 ($p$<.001, $d$=-.79) and week 4 ($p$<.001, $d$=-.68) in favour of SRT. Similar results were found for SE at week 1 ($p$=.002, $d$=.56) and week 4 ($p$=.002, $d$=.56), revealing higher SE for the SRT group.

[INSERT TABLE 4]

*Sleep-related quality of life.* There were no between-group effects on the GSII at post-treatment. However, at follow-up (12-weeks post-randomisation), participants in the SRT group reported reduced impairment to sleep-related quality of life relative to the TBR group ($p$=.001, $d$=1.29; see Table 5). Similar results were found when we analysed group differences for impairment ratings separated by rank (see supplementary Table S1).

*Symptoms of depression and anxiety.* There were no between-group effects for symptoms of depression or anxiety at post-treatment or follow-up ($p$s≥.093; see Table 5).

*Cognitive complaints.* There were no between-group effects for cognitive complaints at post-treatment ($p$=.864) or follow-up ($p$=.071; see Table 5).

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[INSERT TABLE 5]

***Treatment expectations, fidelity and TIB manipulation check***

There were group differences for ratings of treatment credibility (SRT=22.44±2.38 vs TBR=20.83±3.28; $t_{1,54}$=2.10, $p$=.041, $d$=0.57). Group differences for expectancy were of similar magnitude ($d$=0.52) but did not reach statistical significance (SRT=18.68±4.64 vs TBR= 16.18±5.0; $t_{1,54}$=1.62, $p$=.058). Treatment fidelity ratings revealed high content coverage for both interventions (SRT=99%; TBR=97%).

To investigate if participants followed their new sleep schedule as prescribed, we compared the groups on self-reported and actigraphy-derived TIB during the four weeks of intervention (see Figure 3). Self-reported average TIB was significantly lower in the SRT group during early (SRT=421.82±54.26 min vs TBR=497.14±45.10 min; $p$<.001, $d$=-1.98) and late treatment (SRT=432.84±50.41 min vs TBR=501.25±39.29 min; $p$<.001, $d$=-1.83). Consistent with this, actigraphy-derived TIB revealed a significant reduction of TIB in the SRT group during early treatment (SRT=413.51±39.60 min vs TBR=482.96±42.31 min; $p$<.001, $d$=-1.98) and late treatment (SRT=427.03±45.12 vs TBR=483.58±40.36; $p$<.001, $d$=-1.63). Reductions in TIB were in line with average *prescribed* TIB across early (SRT=411.35±67.75 min; TBR=495.60±39.98 min) and late treatment (SRT=414.02±43.54 min; TBR=492.07±38.81 min), indicating that participants adhered to their new sleep schedule. Indeed, comparing average bed and rise-times from the sleep diary with prescribed bed and rise-time showed that both groups closely followed the instructions (average deviation was between 0 to 18 minutes for SRT and between 5 and 14 minutes for TBR; see supplementary Table S2).

[INSERT FIGURE 3]

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To assess whether both treatment arms implemented more regular sleep times, consistent with therapeutic instructions, we examined variability (RMSSD) of diary reported bed and rise-time at baseline, early treatment and late treatment. Within-group comparisons revealed that both groups reported less variability in their bed- and rise times at early ($ps \leq .002$) and late treatment ($ps \leq .0.044$) when compared to baseline (see supplementary Table S3).

Finally, to analyse the extent to which the reduction of TIB was reflected in reduced total sleep time, we performed between-group analysis on sleep diary, actigraphy and PSG-derived TST (see Table 4). For sleep diary, results from linear mixed models revealed a significant reduction at early ($p<.001$, $d=-0.90$) and late treatment ($p<.001$, $d=-0.46$), indicating that participants in the SRT group had a shorter TST. Similarly, results for actigraphy revealed a significant TST reduction in the SRT group at early ($p<.001$, $d=-1.33$) and late treatment ($p<.001$, $d=-1.01$), when compared to TBR. Reductions in TST were also observed for PSG-defined sleep at week 1 ($p<.001$, $d=-1.01$) but not at week 4 ($p=.129$).

***Treatment side-effects***

No serious adverse events were reported to the trial team. There were between-group differences in total number of symptoms reported at week 2, indicating that the SRT group reported more side-effects ($p=.041$). A similar, though non-significant pattern was found at week 3 ($p=.064$). There were no clear group differences at week 1 ($p=.231$) or week 4 ($p=.485$; see supplementary Table S4).

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## Discussion

We designed a randomised, controlled trial to compare SRT (+ sleep hygiene) with its dismantled version (TBR; + sleep hygiene) in order to isolate the role of TIB restriction and test its contribution to the treatment of insomnia. It was hypothesised that full SRT would be superior to TBR across measurements of insomnia, sleep and daytime functioning. The superiority of SRT was demonstrated with large group differences in our primary outcome, insomnia severity, at both 4- and 12-weeks post-randomisation. In support of our hypotheses, the SRT arm reported reduced WASO and SOL, which manifested in increased SE during treatment, relative to TBR. Importantly, these diary findings were accompanied by medium-to-large effect sizes for most actigraphy- and polysomnography-derived sleep variables (*d* range=.40 to .87).

Our data show that SRT improves sleep-related quality of life relative to TBR at 12-, but not at 4-weeks post-randomisation. These results are potentially indicative of a lag between improvements in sleep and daytime functioning, whereby restricted sleep time – and the associated consequences - may mask beneficial treatment effects on daytime variables during the acute phase[8,20]. Related to this, we note that participants in the SRT group reported more treatment-related side-effects during week 2 of the intervention.

We found no group differences for self-reported cognitive complaints, anxiety or depression. These null findings may reflect true effects or could be due to our strict exclusion criteria for comorbid 'caseness' of anxiety and depression, limiting the potential for change. Moreover, given the sample size, the study was not powered to detect small-to-medium effect sizes which are commonly reported for self-reported cognitive complaints[54], anxiety[55] and depression[56]. Indeed, effect sizes for depressive symptoms and cognitive complaints were

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small-to-medium in strength and in favour of SRT (12 weeks), with corresponding *p* values <.1.

The main strength of this work lies in our study design. Choosing a control group that is matched for all key factors minus the component of interest (TIB restriction) is consistent with recent calls for investigators to carefully select and justify control conditions in psychotherapy research[57,58]; and enables us to draw strong conclusions about the specificity of TIB reduction. We note, also, that high-resolution measurement of sleep via diary, actigraphy and polysomnography – with convergent findings across methods – lends support to the robustness of our conclusions. While guidelines recommend that both self-reported and objective assessments of sleep are included in insomnia treatment studies[59], this rarely occurs. A recent meta-analysis found that only a handful of studies reported on PSG, revealing no clear group effect at post-treatment[60]. This is the first randomised-controlled trial to report on the beneficial effects of SRT on sleep continuity as assessed by the gold-standard measure of sleep, polysomnography.

Limitations of this work should be considered. Our sample was recruited in the restricted area of Oxford, UK, and required to meet strict eligibility criteria. Participants in this study were between the age of 26 and 55, highly educated (29% with doctoral degree), and hence, likely to have a high socio-economic status. Consequently, the sample we recruited might not be representative of the general insomnia population in the UK[61]. It is important to note, however, that this study did not seek to test effectiveness (or generalisability) but instead aimed to efficacy test the role of TIB restriction. While we assessed for and excluded additional sleep disorders using standardised questionnaire and interview methods, these assessments have not undergone formal validation. Both treatment arms incorporated sleep hygiene advice. We took this approach because sleep hygiene is often treatment as usual in

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the UK, particularly in primary care[32], but the content may vary. We wanted to eliminate between-subject and between-group variability by standardising exposure to the same sleep hygiene guidance. Our decision was also informed by the fact that sleep hygiene is not considered an effective monotherapy for insomnia.

Although both groups rated treatment credibility in the moderate-to-high range, we note that participants in the SRT group gave slightly higher ratings. These differences, however, did not seem to affect 1) treatment participation and engagement (all participants in the TBR arm attended all treatment sessions and implemented therapeutic instructions, reflected in reduced variability in bed- and rise times); or 2) retention at follow-up assessment (which was 100% in the TBR arm). Indeed, a major strength of this study is that both groups closely adhered to treatment instructions (see Figure 3 and supplementary Tables S2 and S3). Another point is that treatment fidelity for both arms was assessed only for the delivery of treatment session one (and not sessions 2-4). These sessions, however, did not readily lend themselves to recording (taking place over the phone or at participants' homes) and were very brief, following a structured and standardised protocol to review weekly progress and adjust the sleep window. A final limitation is that the principal researcher delivered both study interventions and therefore was not blind to allocation. We built-in several design features in order to minimise both participant and therapist bias: we (a) carefully positioned the study in the participant information sheet so that we did not reveal the superiority hypothesis; (b) recorded treatment sessions and demonstrated high levels of fidelity as assessed by independent appraisal; (c) employed standardised procedures for all visits for both treatment arms; (d) objectively verified the implementation of treatment instructions – giving confidence this accounts for group differences; (e) blind-scored objective outcomes where possible (e.g., PSG); and (f) collected long-term follow-up outcomes via online questionnaire (limiting researcher involvement in the collection of clinical outcomes).

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In conclusion our study identifies restricted time in bed as central to clinical gains and reinforces systematic review evidence that SRT is an effective standalone therapy[7]. Future reports from this trial will help characterise the pathways through which TIB restriction drives sleep improvement. Additional manipulation studies are needed to elucidate whether clinical benefit varies in relation to timing, dose and titration of the sleep window[62].

**Disclosure Statement**

This research study was supported by the National Institute for Health Research (NIHR) Oxford Biomedical Research Centre (BRC). The views expressed are those of the authors and not necessarily those of the NHS, the NIHR or the Department of Health. The research was also supported through a DPhil Scholarship (to LFM) from the Dr Mortimer and Theresa Sackler foundation.

Financial disclosure statement*:* CAE is co-founder of and shareholder in Big Health Ltd, a company which specialises in the digital delivery of cognitive behavioural therapy for sleep improvement (Sleepio). DG is a salaried employee of Big Health Ltd and holds shares in the company. RE reports personal fees from Big Health Ltd., outside the submitted work. All other investigators report no competing interests

Non-financial disclosure statement*:* SDK reports nonfinancial support from Big Health Ltd. in the form of no-cost access to Sleepio for use in clinical trial research. All other investigators report no competing interests.

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**Figure Captions**

**Figure 1.** Participant recruitment CONSORT-style flow diagram.

**Figure 2.** Changes in the primary outcome, insomnia severity, across groups and time points. SRT=Sleep restriction therapy; TBR=Time in bed regularisation. Raw means (+/- 1 SE are presented for both groups at each time point. Statistical group differences are derived from linear mixed models and represented by a double asterisk (**=$p$<.01).

**Figure 3.** Prescribed, self-reported (Diary TIB) and actigraphy-derived time in bed (AW TIB) across baseline (week 1 & 2) and treatment weeks (week 3-6) divided by group (left: SRT= Sleep restriction therapy, right: TBR=Time in bed regularisation). Raw means (+/- 1 SE) are presented for both groups at each time point. In the SRT group, TIB decreased from baseline weeks to treatment weeks, while in the TBR group, TIB remained consistent.

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## Tables

**Table 1**. Contents of treatment per session

| Session | SRT | TBR |
|---|---|---|
| Session 1<br>(60 min) | - Sleep hygiene instructions<br>- Treatment rationale: homeostatic sleep drive & circadian clock<br>- Review average *sleep times*<br>- Set a sleep window and discuss action plan | - Sleep hygiene instructions<br>- Treatment rationale: circadian clock<br>- Review average *bedtimes*<br>- Set a sleep window and discuss action plan |
| Session 2 -4<br>(15 min) | - Review sleep efficiency<br>- Adjust sleep window according to SE criteria<br>- Discuss side effects/challenges to adherence | - Review bedtime consistency<br>- Adjust sleep window if required<br>- Discuss side effects/challenges to adherence |

Abbreviations: SRT=Sleep restriction therapy, TBR=Time in bed regularisation, SE=Sleep efficiency

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**Table 2.** Sleep scheduling criteria

| Criteria | SRT | TBR |
|---|---|---|
| Initial sleep window (TIB) | Average sleep-diary derived TST over the last 2 weeks with a minimum of 5 hrs | Average sleep-diary derived TIB over the last 2 weeks |
| Rise time | Rise time that fits working schedule | |
| Bed time | Bed time was set to equal the new prescribed TIB | |
| Weekly adjustments | a) SE $\geq$ 90% => TIB + 15 min<br>b) SE < 85% => TIB – 15min<br>c) SE 85-89% => No change to TIB | Minimal adjustments ($\pm$15min) if difficulties with work schedule/ changes in circumstances |

Abbreviations: SRT=Sleep restriction therapy, TBR=Time in bed regularisation, TIB=Time in bed, SE=Sleep efficiency (total sleep time/time in bed*100)

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**Table 3.** Participant characteristics by group

| | SRT | | TBR | |
|---|---|---|---|---|
| | n=27 | | n=29 | |
| **Baseline characteristics** | | | | |
| Age, years; M (SD) | 40.63 | 9.13 | 40.93 | 9.24 |
| Female (n; %) | 19 | 70.37 | 20 | 68.97 |
| Marital status (n;%) | | | | |
| Married | 13 | 48.15 | 19 | 65.52 |
| Single | 8 | 29.63 | 7 | 24.14 |
| Divorced | 5 | 18.52 | 3 | 10.34 |
| Separated | 1 | 3.70 | 0 | 0.00 |
| Employment status (n;%) | | | | |
| Employed for wages | 25 | 92.59 | 20 | 68.97 |
| Student | 1 | 3.70 | 4 | 13.79 |
| Self-employed | 0 | 0.00 | 4 | 13.79 |
| Homemaker | 1 | 3.70 | 1 | 3.45 |
| Highest level of qualification (n;%) | | | | |
| GCSE | 1 | 3.70 | 0 | 0.00 |
| A-Levels, diploma or the equivalent | 2 | 7.41 | 0 | 0.00 |
| Some college credit, no degree | 1 | 3.70 | 1 | 3.45 |
| Trade/technical/vocational training | 1 | 3.70 | 2 | 6.90 |
| Associate degree | 0 | 0.00 | 1 | 3.45 |
| Bachelor's degree | 8 | 29.63 | 9 | 31.03 |
| Master's degree | 5 | 18.52 | 6 | 20.69 |
| Professional degree | 3 | 11.11 | 0 | 0.00 |
| Doctoral degree | 6 | 22.22 | 10 | 34.48 |
| **Health characteristics** | | | | |
| BMI, M (SD) | 25.3 | 4.19 | 25.23 | 5.43 |
| Sleep problem duration, years, M (SD) | 12.5 | 8.00 | 9.09 | 8.14 |
| **Outcomes at baseline** | | | | |
| Insomnia severity index, M (SD) | 15.63 | 2.76 | 14.17 | 3.66 |
| GSII, M (SD) | 31.12 | 12.38 | 34.05 | 13.40 |
| HADS-A, M (SD) | 3.92 | 3.30 | 4.97 | 3.72 |
| HADS-D, M (SD) | 6.15 | 3.04 | 6.72 | 3.54 |
| BC-CCI, M (SD) | 7.74 | 3.37 | 8.03 | 4.08 |
| ***Sleep Diary*** | | | | |
| Sleep onset latency, min, M (SD) | 25.96 | 14.96 | 22.81 | 22.33 |
| Wake after sleep onset, min, M (SD) | 62.87 | 31.57 | 76.29 | 51.70 |
| Sleep efficiency, %, M (SD) | 79.12 | 8.13 | 79.25 | 10.23 |
| Total sleep time, min, M (SD) | 396.38 | 43.29 | 388.52 | 54.38 |
| ***Actigraphy*** | | | | |
| Sleep onset latency, min, M (SD) | 8.44 | 9.21 | 10.39 | 8.63 |
| Wake after sleep onset, min, M (SD) | 72.74 | 28.35 | 71.74 | 22.93 |
| Sleep efficiency, %, M (SD) | 82.37 | 6.27 | 82.14 | 5.30 |
| Total sleep time, min, M (SD) | 404.93 | 36.72 | 398.93 | 39.90 |
| ***Polysomnography*** | | | | |
| Sleep onset latency, min, M (SD) | 8.67 | 7.55 | 8.43 | 6.97 |
| Wake after sleep onset, min, M (SD) | 64.58 | 55.95 | 42.88 | 25.82 |
| Sleep efficiency, %, M (SD) | 87.03 | 9.81 | 90.05 | 6.23 |
| Total sleep time, min, M (SD) | 478.60 | 51.11 | 464.04 | 53.98 |

Abbreviations: SRT=Sleep restriction therapy, TBR=Time in bed regularisation, GSII=Glasgow sleep impact index, HADS=Hospital anxiety and depression scale (A=Anxiety scale/D=Depression scale), BC-CCI=British Columbia cognitive complaints inventory

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**Table 4.** Effects of SRT versus TBR on insomnia severity and sleep outcomes

| | | SRT | | TBR | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | *M* | SD | *M* | SD | $Diff_{adj}$ | 95% CI | | *p* | ES |
| **Insomnia severity index** | | | | | | | | | | |
| | week 4 | 7.65 | 3.20 | 11.19 | 4.86 | -4.49 | -6.17 | -2.81 | **<.001** | -1.40 |
| | week 12 | 6.50 | 4.01 | 9.90 | 4.72 | -4.35 | -6.03 | -2.67 | **<.001** | -1.36 |
| **Sleep diary** | | | | | | | | | | |
| SOL | week 1-2 | 17.03 | 21.31 | 22.71 | 17.85 | -8.42 | -14.49 | -2.35 | **0.007** | -0.46 |
| (min) | week 3-4 | 13.07 | 8.90 | 21.01 | 17.74 | -10.68 | -16.75 | -4.61 | **0.001** | -0.57 |
| WASO | week 1-2 | 27.27 | 17.72 | 61.75 | 51.48 | -25.64 | -36.57 | -14.71 | **<.001** | -0.62 |
| (min) | week 3-4 | 19.46 | 13.99 | 56.84 | 42.58 | -28.54 | -39.47 | -17.61 | **<.001** | -0.69 |
| SE | week 1-2 | 88.18 | 6.20 | 83.00 | 10.26 | 5.50 | 2.64 | 8.37 | **<.001** | 0.60 |
| (%) | week 3-4 | 90.33 | 4.36 | 82.24 | 10.65 | 8.41 | 5.55 | 11.27 | **<.001** | 0.92 |
| TST | week 1-2 | 362.72 | 45.17 | 399.92 | 46.80 | -43.76 | -56.12 | -31.40 | **<.001** | -0.90 |
| (min) | week 3-4 | 385.62 | 50.25 | 401.50 | 49.81 | -22.44 | -34.81 | -10.08 | **<.001** | -0.46 |
| **Actigraphy** | | | | | | | | | | |
| SOL | week 1-2 | 4.39 | 2.70 | 12.35 | 12.73 | -6.98 | -11.57 | -2.39 | **0.003** | -0.78 |
| (min) | week 3-4 | 4.96 | 3.42 | 13.48 | 16.56 | -7.76 | -12.35 | -3.17 | **0.001** | -0.87 |
| WASO | week 1-2 | 57.96 | 22.19 | 71.14 | 20.59 | -14.52 | -20.87 | -8.18 | **<.001** | -0.57 |
| (min) | week 3-4 | 59.81 | 21.73 | 73.76 | 20.36 | -14.79 | -21.13 | -8.44 | **<.001** | -0.58 |
| SE | week 1-2 | 84.07 | 5.03 | 81.68 | 5.19 | 2.33 | 0.80 | 3.85 | **0.003** | 0.40 |
| (%) | week 3-4 | 84.13 | 4.59 | 81.23 | 5.08 | 2.80 | 1.27 | 4.32 | **<.001** | 0.48 |
| TST | week 1-2 | 345.73 | 32.14 | 391.28 | 37.23 | -50.85 | -62.88 | -38.82 | **<.001** | -1.33 |
| (min) | week 3-4 | 359.46 | 35.37 | 393.59 | 28.41 | -38.59 | -50.62 | -26.56 | **<.001** | -1.01 |
| **Polysomnography** | | | | | | | | | | |
| SOL | week 1 | 5.63 | 4.95 | 10.93 | 13.31 | -5.35 | -10.46 | -0.23 | **0.041** | -0.74 |
| (min) | week 4 | 7.17 | 7.35 | 11.52 | 14.65 | -4.41 | -9.52 | 0.71 | 0.091 | -0.61 |
| WASO | week 1 | 31.90 | 20.48 | 51.18 | 32.84 | -32.34 | -45.76 | -18.92 | **<.001** | -0.79 |

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| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| (min) | week 4 | 37.38 | 35.41 | 52.03 | 34.44 | -27.89 | -41.20 | -14.59 | **<.001** | -0.68 |
| SE | week 1 | 92.26 | 4.41 | 89.46 | 5.91 | 4.51 | 1.65 | 7.37 | **0.002** | 0.56 |
| (%) | week 4 | 91.54 | 7.23 | 88.80 | 7.18 | 4.49 | 1.65 | 7.33 | **0.002** | 0.56 |
| TST | week 1 | 372.42 | 33.44 | 425.71 | 40.92 | -48.01 | -69.71 | -26.31 | **<.001** | -1.01 |
| (min) | week 4 | 394.64 | 49.06 | 415.14 | 50.01 | -16.63 | -38.13 | 4.87 | 0.129 | -0.35 |

Abbreviations: SRT=Sleep restriction therapy, TBR=Time in bed regularisation, ISI=Insomnia severity index, SOL=Sleep onset latency, WASO=Wake-time after sleep onset, TST=Total sleep time. $Diff_{adj}$= Adjusted mean difference derived from linear mixed model, 95%CI=95% Confidence interval of the adjusted mean difference, ES=Effect size (Cohen's d). Significant p-values are displayed in bold. M and SD refer to unadjusted means and standard deviations.

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**Table 5.** Effects of SRT versus TBR for measures of daytime functioning

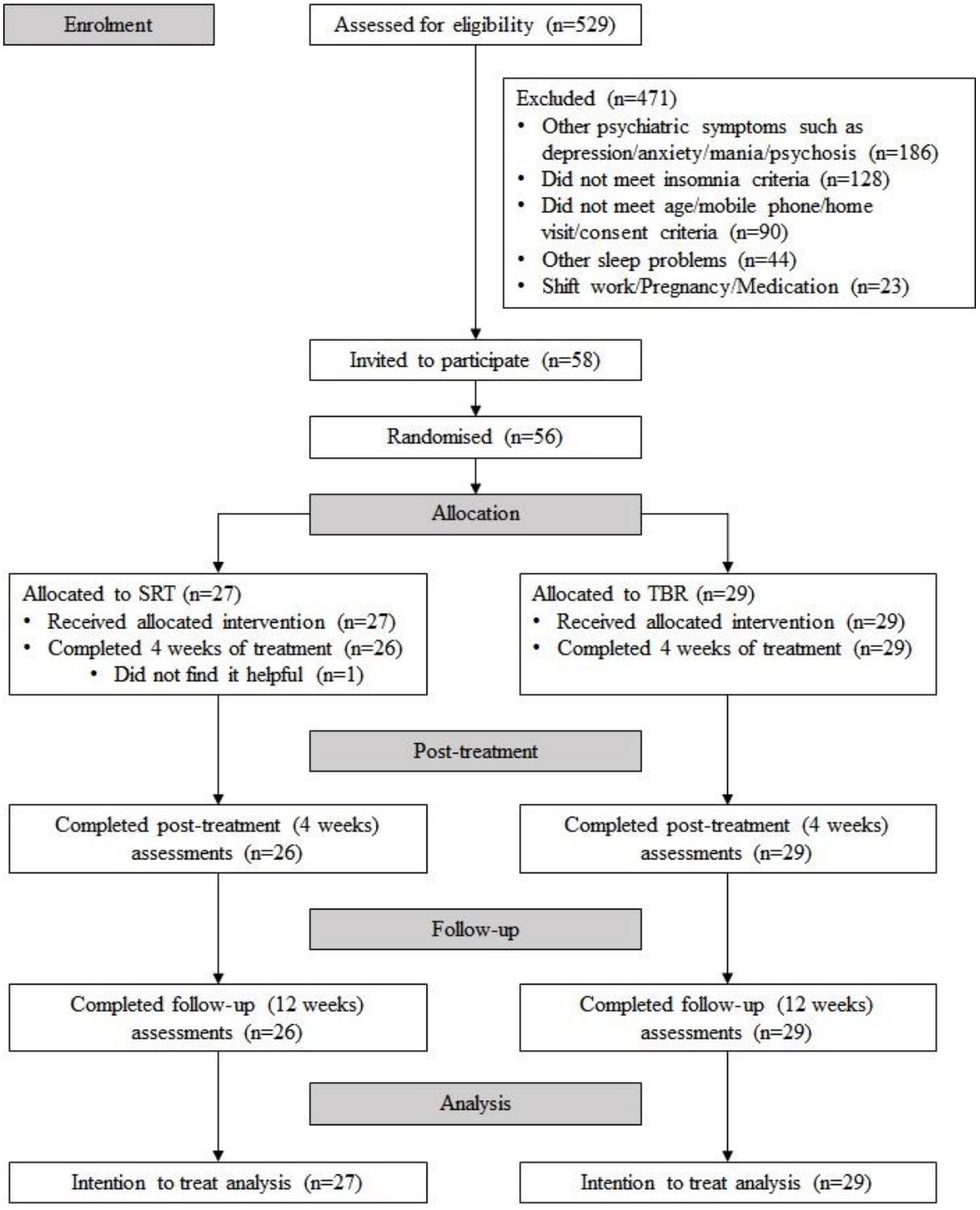
| | | SRT | | TBR | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | *M* | SD | *M* | SD | $Diff_{adj}$ | 95% CI | | *p* | ES |
| GSII | week 4 | 50.62 | 18.80 | 49.27 | 22.74 | 2.52 | -6.84 | 11.89 | 0.595 | 0.20 |
| | week 12 | 57.94 | 21.07 | 42.45 | 23.38 | 16.67 | 7.31 | 26.04 | **0.001** | 1.29 |
| HADS-A | week 4 | 2.54 | 2.27 | 3.79 | 3.41 | -0.54 | -1.46 | 0.38 | 0.249 | -0.15 |
| | week 12 | 2.42 | 2.37 | 3.76 | 3.20 | -0.62 | -1.54 | 0.30 | 0.186 | -0.18 |
| HADS-D | week 4 | 5.15 | 3.15 | 4.97 | 3.26 | 0.60 | -0.66 | 1.86 | 0.218 | 0.18 |
| | week 12 | 4.56 | 2.80 | 6.07 | 4.01 | -1.02 | -2.34 | 0.18 | 0.093 | -0.33 |
| BC-CCI | week 4 | 5.73 | 4.36 | 6.14 | 4.96 | -0.15 | -1.83 | 1.53 | 0.864 | -0.04 |
| | week 12 | 3.23 | 3.00 | 5.03 | 3.95 | -1.54 | -3.22 | 0.14 | 0.071 | -0.41 |

Abbreviations: SRT=Sleep restriction therapy, TBR=Time in bed regularisation, GSII=Glasgow sleep impact scale, HADS=Hospital anxiety and depression scale (A=Anxiety scale/D=Depression scale), BC-CCI=British Columbia cognitive complaints inventory, $Diff_{adj}$= Adjusted mean difference derived from linear mixed model, 95%CI=95% confidence interval of the adjusted mean difference, ES=Effect size (Cohen's d). Significant p-values are displayed in bold. M and SD refer to unadjusted means and standard deviations.

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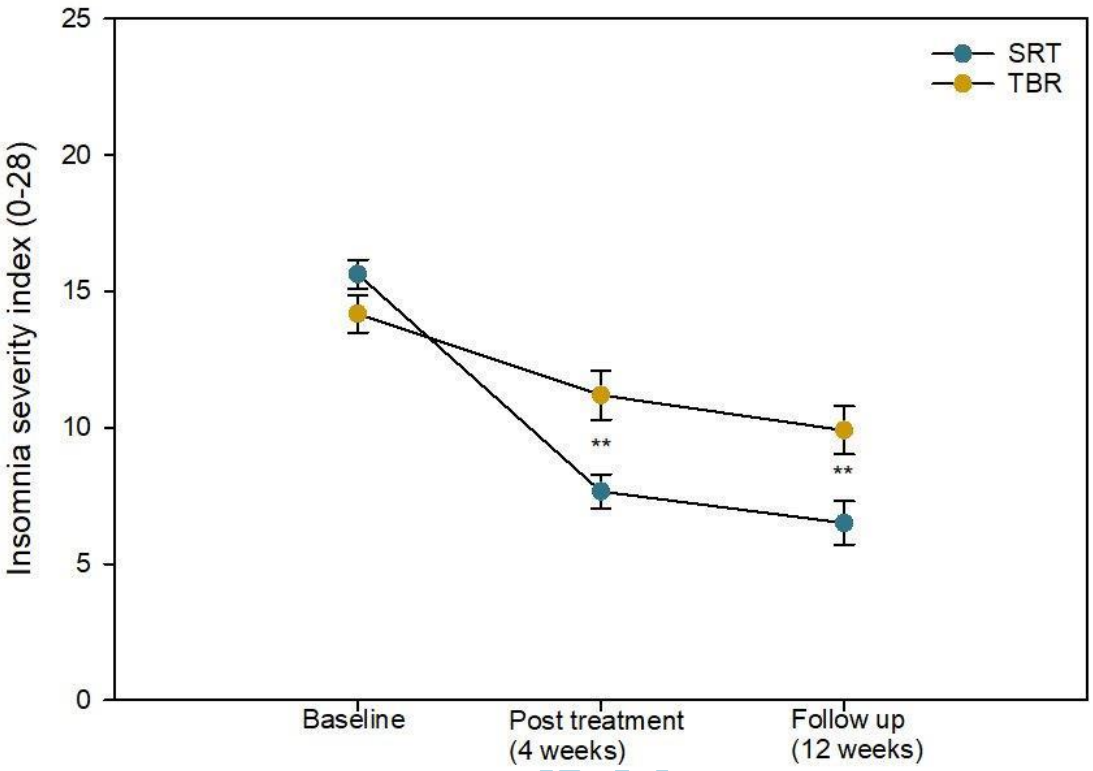
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Figure 1




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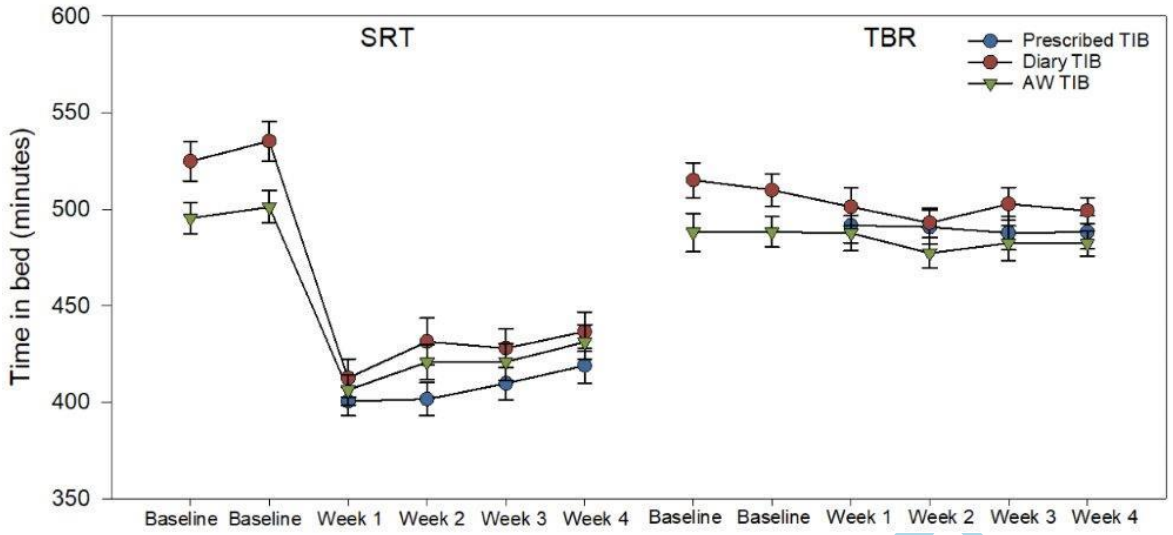
Figure 2




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Figure 3




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