How does sleep restriction therapy for insomnia work? A systematic review of mechanistic evidence and the introduction of the *Triple-R* model

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Conflict of Interest

Colin Espie 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. This review is in no way connected to Big Health Ltd. All other investigators report no potential conflicts of interest.

Summary

For over 30 years sleep restriction therapy (SRT) has been used to treat insomnia but we know very little about how this therapy exerts its effects. When SRT was first described, it was hypothesised to treat insomnia by addressing four key factors: strengthening homeostatic sleep pressure, inhibiting perpetuating practices (excessive time in bed), attenuating hyperarousal and tightening regulatory control of sleep by the endogenous circadian pacemaker. We conducted a systematic literature review in search of evidence for these putative mechanisms-of-action. A total of 15 randomised and non-randomised studies investigating SRT met inclusion criteria. For each study, we extracted all variables associated with the proposed mechanisms and assessed study quality using a structured appraisal tool. The extracted variables were: time in bed (TIB), napping, variability in sleep, markers of circadian rhythmicity, measurements of sleep pressure/sleepiness, and assessments of arousal. Overall study quality was poor as indicated by a mean quality score of 17 (out of a possible range of 0 to 31). No study indicated, or indeed was designed to test, whether changes in the proposed mechanisms act as *mediators* of treatment outcomes. Of all reviewed studies, most reported a reduction in TIB (10/10) and/or revealed a decrease in sleep onset latency (10/14), indexing increased sleep pressure. However, such changes were most often reported at the end of treatment, reflecting an outcome and not a mechanism of SRT per se. Evidence for reduction in arousal (4/4) and night-to-night sleep variability (2/2) was found in only a small number of uncontrolled studies while there was no evidence for change in circadian phase or periodicity (0/1). Our review suggests that SRT targets some of the hypothesised processes but specifically-designed mechanistic evaluations are needed. We introduce a new testable model of SRT mechanism-of-action (*Triple-R*) and set out a research agenda aimed at stimulating prospective investigations.

Keywords

Insomnia, Sleep restriction therapy, Mechanisms, Mediators, Cognitive behavioural therapy, Treatment, Psychological intervention, Review, Sleep

Abbreviations

CBT-I	Cognitive-behavioural therapy for insomnia	SE	Sleep efficiency
CT	controlled trial	SOL	Sleep onset latency
DISS	Daytime Insomnia Symptom Scale	SRT	Sleep restriction therapy
EEG	Electroencephalography	SSS	Stanford sleepiness scale
ESS	Epworth sleepiness scale	SWA	Slow wave activity
GSES	Glasgow sleep effort scale	TIB	Time in bed
MSLT	Multiple sleep latency test	TST	Total sleep time
NREM	non-rapid eye movement	TWT	Total wake time
PSG	Polysomnography	UCT	Uncontrolled trial
RCT	Randomised controlled trial	WASO	Wake-time after sleep onset
RT	Reaction time		

Introduction

Arthur Spielman first formulated the concept of sleep restriction therapy (SRT) in 1983 [1]. His approach was guided by the idea that addressing factors that perpetuate chronic insomnia is essential for therapeutic success [2]. People with insomnia may extend time in bed (TIB) to compensate for long sleep onset latencies or disrupted sleep [3]. Extra TIB will occasionally yield more sleep, but can also lead to increased wakefulness, fragmented sleep, and variability in its timing [4, 5]. Through restriction of TIB*, SRT aims to improve the consolidation of sleep and constrains its occurrence to a specific time. According to the original sleep restriction intervention [6], patients are asked to fill out a sleep diary for two weeks. Subsequently, the therapist prescribes an initial TIB that equals the average reported total sleep time (TST). For example, a patient who spends nine hours in bed, but only reports six of those sleeping, would be assigned a six-hour sleep window. In agreement with the patient, the therapist establishes a rise-time and then sets the time for retiring at night to equal the new prescribed TIB (which is typically no less than five hours). Sleep efficiency (SE) is subsequently used as a criterion for making weekly changes to TIB for the duration of the treatment (typically four to six weeks). It is calculated as a percentage by dividing TST/TIB and therefore takes into account wake time that occurs before and after sleep onset. High SE, reflecting less wakefulness during bedtime, is regarded as an important feature of sleep health, indexing consolidated sleep [6, 7]. Hence, only when a patient reports high SE, TIB will be increased (see Table 1). Through a series of weekly adjustments to TIB, an optimal bedtime length is reached, with the aim of maintaining high SE without excessively curtailing the length of sleep, while delivering improvements in daytime functions.

*Arthur Spielman originally referred to ‘SRT’ as a treatment that involves restricting available sleep time and making changes in TIB contingent upon the patient's clinical response ([6] p. 46, 7-8). Therefore, strictly speaking, the treatment is considered a ‘time in bed restriction’, but is known today as ‘sleep restriction therapy’. As this is the accepted term, it will also be used in the present review.

However, variability in the implementation of SRT is present at every level (sleep window generation, minimum TIB, SE titration criteria, positioning of the sleep window) and may therefore result in different sleep window trajectories. That is, adaptation of the sleep window over time (to improve sleep quality and/or quantity) may vary markedly depending on what SRT criteria are applied [8].

[INSERT TABLE 1 HERE]

Sleep restriction therapy is routinely applied as part of cognitive-behavioural therapy for insomnia (CBT-I) [9-11], but also as an independent, single-component intervention [12, 13]. The efficacy of SRT was recently reviewed by Miller et al. [12]. The authors identified nine studies, four of which met adequate methodological strength. Results revealed moderate-to-large effect sizes for sleep diary measures of sleep onset latency (SOL), wake-time after sleep onset (WASO), and SE, as well as a small effect size for increased TST. While effective, the implementation of SRT may also be associated with short-term side effects. It can lead to transient increases in somnolence, fatigue, and impaired vigilance [14].

Theoretical support for SRT and review aims

The ‘3P model’ (also known as ‘Diathesis-stress model’) [15] has served as the conceptual basis for the application of SRT. It proposes that predisposing factors precede the onset of sleep difficulties and increase individuals' vulnerability to insomnia; precipitating factors typically involve acute stressors that trigger the onset of insomnia; and perpetuating factors maintain symptoms by mismatching sleep opportunity and sleep ability. When Spielman, Saskin and Thorpy first fully articulated SRT in 1987 [6], they stated that SRT therapeutically addresses one of the key factors that they believe perpetuates insomnia: the tendency to remain in bed while awake. By restricting TIB, sleep opportunity is curtailed, resulting in partial sleep deprivation that may be crucial for therapeutic effects. Results of the seminal SRT study revealed that patients experienced an increase in TST as well as improvements in SOL, SE, and subjective insomnia symptoms after eight weeks of treatment. When discussing the results of this first trial, the authors proposed several mechanisms that may have led to sleep improvements:

> *‘The mild sleep loss produced at the beginning of sleep restriction therapy may be crucial for its effectiveness. The partial sleep deprivation may have consolidated sleep directly, produced daytime*

> *fatigue that dampened the insomniacs' chronic state of hyperarousal, or reduced maladaptive conditioning because less time was spent lying awake in bed […]. Getting into bed early, staying in bed late, and napping are short-sighted strategies that foster the fluctuations in the distribution and amounts of sleep and waking that are characteristic of the sleep of insomniacs. The present approach of restricting TIB stabilizes sleep.'* ([6] pages 53-54)

In 2011, Spielman, Yang and Glovinsky similarly emphasised that SRT may work through strengthening homeostatic sleep drive, inhibiting perpetuating practices (excessive TIB and napping), attenuating hyperarousal and tightening regulatory control of sleep by the endogenous circadian pacemaker [16]. We are not aware of any published review to date that has examined these mechanisms by systematically synthesising observations from trials of SRT. Each of these pathways can be indexed via commonly employed measurements in sleep research (see Table 2) permitting appraisal of mechanistic change. Building upon the importance of understanding mechanisms-of-action of psychological treatments [17], we conducted a systematic review of the available literature with the aim of identifying mechanistic variables associated with SRT outcomes. Finally, we conclude with a new expanded and testable model of SRT mechanism of action.

[INSERT TABLE 2 HERE]

Systematic literature review

Search strategy

The systematic review was undertaken in compliance with PRISMA guidelines for the reporting of systematic reviews [18] to evaluate evidence regarding mechanisms involved in SRT. We therefore searched for both randomised and non-randomised single-component SRT studies which implemented therapy guidelines consistent with the approach of Spielman et al. [6]. To identify relevant articles, we conducted a thorough search of Web of Knowledge, PubMed, and Scopus. Databases were accessed via the University of Oxford Ovid and EBSCO online system, with a final search date of February 2018. Additional records were identified by accessing journals focused on sleep research, namely: 'Journal of Sleep Research', 'Sleep', 'Sleep Medicine' and 'Behavioral Sleep Medicine'. We applied the same subject and text word strategy as Miller et al. [12], with

'insomnia' and' sleep restriction' or 'sleep compression' as the primary search terms. Sleep compression was included as a primary search term to capture articles that might have used a form of sleep restriction that is similar to SRT or used SRT as a control group. If the titles included 'insomnia', 'behavior/ behaviour' or 'treatment' the online article was accessed, and the abstract examined. We included randomised controlled (RCT), controlled (CT) and uncontrolled trials (UCT) as long as SRT was applied in adults with insomnia. We included studies that combined SRT with sleep hygiene because there is currently no evidence that sleep hygiene is an active single-component treatment [13, 19, 20].

Inclusion criteria

a) Adults (≥ 18 years)
b) Whole sample met criteria for insomnia disorder or insomnia symptoms, as verified by diagnostic criteria; self-report; or objective measures in the sleep laboratory
c) Investigated a standalone intervention (or combination with sleep hygiene) involving restriction of TIB for the treatment of insomnia, consistent with the approach of Spielman et al. [6]
d) Examined sleep-wake outcomes before and after treatment
e) Peer reviewed paper published in English between 1987 and 2018
f) Reported data on at least one variable associated with one of four proposed mechanisms (homeostatic sleep drive, circadian sleep-wake cycle, perpetuating practices, arousal; see table 2)

Exclusion criteria

a) Conference abstract, dissertation, letter, case study, review or case series
b) Applied sleep compression as the intervention

Quality assessment

The Downs and Black checklist [21] was used to assess the methodological quality of all included studies by the first author (LFM). It is considered one of the 'best' tools for evaluating randomised and non-randomised intervention studies [22]. The checklist includes 27 items assessing study reporting, external and internal validity, and has good inter-rater reliability [21]. Item 14, regarding participant blinding to intervention was removed given

the nature of behavioural treatment. Thus, the possible score ranged from 0 to 31, with higher scores indicating better methodological quality.

Data extraction

Articles were screened based on title, abstract and full text by the first author (LFM). Search results were exported to a reference management software (EndNote X7) and duplicates were removed. Eligibility of the papers was finalised following review and discussion between authors (SDK). We extracted information from each study in relation to the four mechanistic pathways proposed by Spielman and colleagues. We then organised review results by these four pathways and measurements used to index them (see Table 2).

Results

Records identified through database searching with keywords yielded 1671 papers (WEB OF KNOWLEDGE = 786, PubMed = 600, SCOPUS = 284). An additional 582 records were identified through journal specific search. Duplicates were removed, and 1620 titles were screened; of which 358 abstracts were acquired. From these, 27 studies were deemed potentially eligible and a full version of the article was therefore accessed. Of these 27 studies, 12 were excluded as they did not meet the inclusion/exclusion criteria. The selection process is shown in Figure 1.

[INSERT FIGURE 1 HERE]

Study characteristics

Fifteen studies met all criteria and were included in the synthesis. This comprised seven controlled [23-29] and eight uncontrolled trials [6, 14, 30-35]. Studies took place at various sites worldwide: seven in the USA, four in Great Britain, two in Australia, one in New Zealand, and one in China. Seven studies had less than 18 participants, four yielded between 32 and 46 participants, and four reported over 70 participants. The total number of participants included in the analyses was 598. The average age of participants ranged from 36 to 69 years. All studies included mixed-gender groups, however there was a greater proportion of women reported in the descriptions of the study population (66.21%). Six studies implemented SRT in combination with sleep hygiene [24, 26, 27, 29, 30, 36]. Assessment of study quality by the *Black and Downs* questionnaire revealed that overall

study quality was poor, scoring between 13 and 21 (mean score 17.13 ± 2.2) out of a possible 31. More than half of the studies did not employ a randomised-controlled design and therefore lacked internal validity. Reports of side effects, treatment delivery (therapist, facilities) and detailed descriptions of the recruitment processes were typically missing, resulting in low scores on external validity.

Narrative Synthesis

No study reported, or was designed to report, whether changes in the proposed mechanisms act as mediators of treatment outcomes. Nevertheless, in line with the inclusion criteria, all studies reported information on at least one of the proposed mechanisms. Hence, 10 studies reported information regarding perpetuating practices (i.e. TIB and napping), four on arousal (physiological and cognitive measurements), 14 on markers of homeostatic sleep drive (mainly SOL) and three on the circadian sleep-wake cycle (variability in sleep parameters and markers of circadian rhythmicity). However, changes in variables were most often reported at the end of treatment, reflecting an outcome and not necessarily a mechanism of SRT. In fact, only five studies (33%; mostly uncontrolled) obtained measurements of hypothesised mechanisms *during* the course of SRT, in addition to measurements after treatment completion. An overview of all included studies and their mechanistic findings can be found in Table 3.

[INSERT TABLE 3 HERE]

Perpetuating practices

All studies [6, 24, 26, 27, 29, 30, 32, 33, 35, 37] (3 RCTs, 7 UCTs) that reported on perpetuating practices found that participants spent less TIB at the end of treatment. Only four studies (all uncontrolled) revealed a reduction of TIB during the course of SRT [26, 32, 35, 37] and only two studies (1 RCT, 1 UCT) reported data on napping [6, 27]. Time in bed decreased by an average of 94.42 (± 17.83) minutes from baseline to post-treatment and about 107 (± 30.15) minutes during treatment, as indicated by sleep diary analysis. In addition, actigraphy data from two controlled studies showed a reduction of 78.64 (± 30.67) minutes in TIB after SRT [24, 30].

Friedman et al. [27] described a reduction in TIB from baseline to post-treatment by both actigraphy (~73 min) and polysomnography (PSG, ~ 56 min). However, those reductions were not reported to be significant, likely due to the low sample size (N=8). No detailed description of how PSG was conducted or how long

participants were asked to stay in bed was provided. Nap data from the same study revealed that six participants in the SRT group stopped napping entirely during treatment, eight remained non-nappers and two remained nappers (reported napping $\geq$ 6 min/day), indicating an overall reduction in napping consistent with treatment instructions.

Additionally, Spielman, Saskin and Thorpy [6] reported that 10 participants napped at baseline (for a mean time of 26.5 minutes) compared with only two (for an average of 13.3 minutes) on completion of treatment. The authors also analysed whether mild relapses in sleep at follow-up could be accounted for by an increase in TIB. Results showed that change in TIB from the end of treatment to follow-up was positively correlated with change in total wake time and sleep latency, suggesting that the degree to which TIB is increased following treatment is related to the degree to which sleep deteriorates.

Overall, TIB seems to be reliably decreased from pre- to post-treatment, across both controlled and uncontrolled studies. Data on TIB reductions during treatment and changes in napping are limited and have not been investigated as mediators of treatment outcome.

Hyperarousal/Conditioned arousal

Four uncontrolled studies reported data on arousal measurements [14, 32-34] but all investigated different aspects of arousal. In the study of Miller et al. [32] participants completed the ‘Daytime insomnia symptom scale‘ (DISS) [38] at four assessment points per day (rise-time, 12:00 hours, 18:00 hours and bedtime) for one week before-, and for three weeks during the intervention. Alert cognition is one of the DISS sub-scales, comprised of five items (Forgetful, Clear-headed, Concentrate, Effort, Alert). Compared with baseline, alert cognition decreased during the first week of SRT. At week three of SRT, alert cognition was significantly increased at rise-time but significantly decreased at bedtime, resembling those of good sleeping controls. The authors suggest that modification to alert cognition may reflect a re-structuring of cognitive arousal by SRT; inhibiting it before bedtime and restoring it in the morning. These changes could reveal a potential mechanism by which SRT exerts its therapeutic effect. However, the interpretation of the results is limited by the small sample size (N=9) and the lack of a control group.

Vallières, Ceklic, Bastien and Espie [34] investigated physiological mechanisms of action of SRT. In this study, five participants underwent a treatment protocol with daily sleep measurements, actigraphy, PSG and daily cortisol assays. The authors conducted power spectral analysis on PSG nights across baseline and treatment focussing on change in high frequency patterns. There was an overall decrease across the three nights in beta-1 power during stages two and three, as well as total non-rapid eye movement sleep (NREM) among responders (N=3). For beta-2, significant reductions from baseline to treatment night one and two were observed during NREM sleep. Furthermore, *visual inspection* of morning and evening cortisol levels suggested that by the second week of treatment levels were lower than at baseline. The authors concluded that these results demonstrate a potential action of SRT on cortisol levels although it should be noted that cortisol levels showed a tendency to increase after treatment. While interesting, this study is preliminary given the small sample size and absence of a control group.

Another study investigated physiological markers of arousal during SRT [33]. Overnight measures of temperature and cortisol in six participants were collected at baseline and at 6 weeks (post SRT). Results revealed that core body temperature decreased significantly by 0.09°C, suggesting that SRT may reduce physiological arousal in insomnia after six weeks of treatment. Contrary to the hypothesis, cortisol concentrations were found to be higher in the morning at post-treatment. The authors suggest that this might represent normalization of the cortisol awakening response. However, the reliability of these results is limited by the quality of the study (N=8, [6 with complete overnight data], no control group).

Kyle et al. [14] conducted a mixed methods study focussing on the patient experience of SRT. The questionnaire battery included the ‘Glasgow sleep effort scale’ (GSES, [39]), which purports to measure sleep effort, a construct thought to be closely linked to cognitive and emotional arousal [40]. Results revealed that the mean GSES score decreased significantly with SRT from baseline to post-treatment. The pattern remained robust at 3-month follow-up. However, reduction in sleep effort was not investigated as a predictor of clinical improvement, and the study is limited by the absence of a control group.

In summary, there is no strong evidence for a reduction in cognitive or physiological arousal. The only studies reporting data on change in measures of arousal with SRT are uncontrolled with sample sizes of $n \leq 18$ and therefore no reliable conclusions can be drawn.

Homeostatic sleep drive

All studies were reviewed for subjective and objective markers of homeostatic sleep drive. The finding most consistently reported was a reduction in SOL. Fourteen studies reported SOL at baseline and post-treatment. Ten (5 RCTs) of them indicated a significant decrease in SOL of 23.93min ($\pm$ 6.54) as obtained by sleep diaries. In addition, Vallières et al. [34] reported a reduction in SOL of 38.8 minutes during the first two days of treatment and a reduction of 37 minutes at post-treatment in treatment responders (N=3), as measured by PSG. Non-responders (N=2) did not show a decrease in SOL during the first two days of treatment (+ 0.9min), but a reduction of 11.8 minutes at the end of treatment. Given the small sample size these results were not tested for statistical significance. Reductions in SOL were also reported by six studies presenting follow-up data [6, 14, 23, 26, 37, 41], indicating that SRT may lead to stable change in SOL. All other markers of sleep pressure were obtained by different measurements across studies and are summarised below.

Vallières et al. [34] measured SWA at five time-points in five participants (2 at baseline, 3 during treatment). Participants were also asked to monitor their sleepiness and alertness levels in the morning and evening using a '0 to 4 Likert scale' as part of the sleep diary. *Visual inspection* of PSG recordings did not indicate a greater homeostatic response during SRT, but analysis of subjective sleepiness indicated that greater sleepiness at night was associated with higher SE and shorter WASO during the subsequent night. While both measurements provide an opportunity to test an association between sleepiness and treatment outcome, a stronger study design is needed to investigate if sleep pressure as indicated by SWA or sleepiness is a) reliably observed in SRT versus an appropriate control group; and b) a mediator of clinical treatment effects.

Friedman et al. [27] compared three groups (SRT, nap-modified SRT, sleep hygiene) on subjective sleepiness using the Stanford sleepiness scale (SSS) and objective sleep propensity using the Multiple sleep latency test (MSLT). For the SSS, participants were asked to rate their sleepiness every two hours during days in which the actigraph was worn (4 consecutive days at baseline, treatment and follow-up). Analysis revealed an

overall significant treatment x time interaction on SSS scores. The authors interpreted this result as evidence of a treatment-related decrease in sleepiness for both the SRT and sleep hygiene groups but not the nap modified SRT group. In contrast, data from the MSLT indicated that participants across all groups were somewhat sleepier at the end of treatment, although statistical significance was not reported. Again, associations between sleepiness and treatment outcome were not tested and results are clearly limited by the small sample size (N=6 in the SRT group).

Kyle et al. [14] assessed sleep-restriction related side effects finding 94% (18 participants) reported 'extreme sleepiness' during the first week of treatment. In addition, exploratory correlation analysis revealed that higher frequency of side-effects was associated with a greater magnitude of change in symptom severity and SE. However, this relationship was not specifically tested for sleepiness.

In a follow-up study by Kyle et al. [37], the psychomotor vigilance task (PVT) and the 'Epworth sleepiness scale' (ESS) [42] were assessed at baseline, during treatment (week 1 to 4), and after three months follow-up. Compared to baseline, results showed that the ESS score increased significantly during treatment (from baseline to week 1, week 2 and week 3) but was not different at the end of treatment [week 4 (post-treatment) and week 12 (follow-up)]. A similar pattern was observed for reaction times (RT) obtained by the PVT. Analysis revealed that participants were slower during the acute treatment, returning to baseline levels at follow-up. The aim of the study was to investigate whether implementation of SRT was associated with increased daytime somnolence and impaired vigilance; therefore, no analyses were conducted to link sleepiness with improvements in insomnia. Furthermore, the results are limited by the small sample size (N=16) and the lack of a control group.

Conversely, Whittall et al. [35] did not observe increased sleepiness during SRT. Sixteen participants underwent a brief SRT phase (2 weeks) and completed the ESS and a Go/NoGo task at pre-, mid- (i.e. after one week of SRT) and post-SRT (after two weeks of SRT). The authors found no significant changes in ESS scores, RTs or accuracy in the Go/NoGo task. Yet, the interpretation of these results is limited by several factors. The implementation of SRT was very brief and modified to prevent extreme daytime sleepiness, TST did not decrease significantly during the two weeks of treatment, there was no control group and the sample size was small (N=16).

In a small, uncontrolled study (N=9) Miller et al. [47] tested the hypothesis that daytime symptoms would deteriorate during SRT. Daily sleepiness was measured by the 'sleepiness/fatigue' sub-scale of the DISS (containing adjectives; Sleepy, Fatigued, and Exhausted) and compared between baseline, week one and week three of SRT. Consistent with the hypothesis, sleepiness increased in the beginning of the treatment (week 1) but decreased towards the end of the treatment (week 3). A time of day × week interaction revealed that sleepiness/fatigue was increased at bedtime. The authors suggest that this result is indicative of heightened sleep pressure, which may help to overcome cognitive arousal to facilitate the initiation of sleep. The interpretation of results is limited by the uncontrolled study design and small sample size (N=9, no control group), and because *fatigue* and *sleepiness* were subsumed within one domain of functioning.

Overall, while most studies obtained some indicator of homeostatic sleep pressure, there is little standardisation in measurement, studies are of low quality (small sample size, no control group) and only one study assessed EEG markers of sleep pressure.

Circadian sleep-wake cycle

In this systematic review, only three studies reported results on circadian markers and sleep variability estimates. Spielman, Saskin and Thorpy [6] measured change in night-to-night variability of self-reported SOL, total wake time (TWT), TST and SE, between baseline and end of treatment. All variables showed a decrease from baseline to the end of treatment, indicating stabilised sleep parameters.

Second, the study of Friedman et al. [26] compared TIB variability between a relaxation and a sleep restriction arm in 22 elderly participants. Only participants in the sleep restriction group showed a significant change in TIB variability, reporting a 22-minute reduction in TIB in the final two weeks of treatment relative to baseline. However, there was no treatment x time interaction reported and the results are limited by the small sample size (N=10 in the SRT group).

Lastly, Miller et al [33] assessed changes in core body temperature from pre to post six-week SRT in a small sample of patients. However, there was no change in estimates of circadian phase or periodicity as indicated by model parameters for the fitted overnight data. The lack of findings could be explained by the small sample size (N=6) and the absence of a control group.

In sum, only two studies reported a decrease in sleep or TIB variability even though SRT is proposed to stabilise sleep. As sleep diaries (and actigraphy) are standard measurements in clinical sleep research, data on sleep variability were likely collected in most of the studies described in this review but not reported.

Discussion

Summary

The primary objective of this review was to systematically examine empirical evidence for four proposed mechanistic pathways involved in SRT treatment response [6, 43]. One of the most consistent observations across studies was that TIB was reliably decreased both during and at the end of SRT, in comparison to baseline values and relative to control groups. Spielman et al. proposed that reduced TIB increases sleep pressure and subsequently inhibits arousal. However, we did not find research evidence for this temporal pathway, principally because no study sought to investigate it directly. Instead, reviewed studies showed that SOL was reliably decreased from baseline to post-treatment, as well as from baseline to the first weeks of SRT implementation, potentially reflecting increased sleep pressure. In addition, five studies reported increased subjective sleepiness during treatment but only one study assessed homeostatic sleep pressure through quantitative EEG. In this small study (N=5), participants showed a reduction in high-frequencies but no change in SWA. Limited assessment of homeostatic sleep pressure is surprising given that 1) sleep homeostasis may be dysfunctional in insomnia [44]; and 2) partial sleep deprivation has well-described effects on sleep homeostatic processes [45, 46]. Equally surprising is the lack of investigation of arousal, which is considered a key maintenance factor in the expression of persistent insomnia [47, 48]. Physiological arousal was investigated by two uncontrolled studies, indexed by core body temperature [33] and cortisol levels [33, 34]. While core body temperature significantly decreased, there was no reliable change in evening cortisol, and an increase in morning cortisol levels. Evidence for decreased cognitive arousal was limited. Only three small, uncontrolled studies reported results for a decrease in sleep effort, alert cognition and cortical arousal. Finally, while a regular TIB is proposed to stabilise sleep-wake timing, only two studies reported changes in sleep or TIB variability, indicating less variability at the end of treatment. Importantly, no study showed change in objective measures of circadian rhythmicity, but only one study included such measures.

In summary, then, the principal conclusion from this review is that the field lacks high quality evidence that SRT achieves therapeutic success through its proposed mechanistic pathways. That is, despite a strong

evidence-base for single component SRT we do not know which mechanisms account for change in key insomnia outcomes.

Methodological considerations

To the authors' knowledge this is the first systematic review to investigate the mechanisms of SRT by focusing on its theoretical underpinnings. A systematic review applying selective criteria for identification and screening of relevant literature was chosen as the main methodological approach. Studies using SRT as a partial treatment (e.g. within CBT-I) were excluded, ensuring that we were able to examine the effects of SRT in the absence of possible influence from other evidence-based components of CBT-I, such as stimulus control therapy or relaxation.

There are several limitations of this review. First, uncontrolled studies and secondary analyses were included to increase the quantity rather than the quality of studies. Heterogeneity in both study design and measurement of proposed mediator variables precluded meta-analysis. A second limitation is that only published studies were included, and so our analysis may be influenced by potential publication bias. Third, this review did not consider adherence to the SRT protocol or protocol variability. We hypothesise that these factors could significantly influence measurements associated with mechanisms and contribute to variability in findings across studies (e.g. sleepiness). Fourth, we pre-defined possible mechanisms consistent with the seminal SRT literature and screened research articles accordingly. It is possible that other putative treatment mechanisms have been investigated in the SRT field. However, given the small size of the SRT literature, and the fact that we sought and read all published SRT studies, we consider this highly unlikely.

The identification of variables associated with mechanisms was hindered by overlapping constructs and the lack of distinction between mediators, indicators of adherence, and outcome variables. For example, reduction of TIB is not just part of SRT, it indexes adherence and functions as a mechanism. Increased homeostatic sleep pressure is hypothesised to be central to treatment effects and manifests as increased sleepiness before sleep and increased SWA during sleep. However subjective sleepiness is also a symptom of insomnia and expected to be improved by successful treatment. Therefore, sleepiness would be expected to increase during treatment (particularly in the evening), but decrease at the end of treatment as a sign of good quality, restorative sleep

(particularly in the morning). Moreover, reviewed variables can often be associated with more than one mechanism, for example core body temperature and cortisol can reflect measurements of arousal as well as measurements of circadian rhythmicity [49, 50]. Without conducting appropriately powered and hypothesis-driven mediational analyses in RCTs, it becomes difficult to assess the temporal order of these relationships, especially across studies with variable treatment implementation periods (e.g. treatment outcome after 2 vs. 4 weeks of SRT) [51].

Towards a mechanistic model of SRT

When SRT was first developed it was principally based on the 3-P account of insomnia development and maintenance. This pragmatic (and generic) model has provided a very useful 'wide-angle lens' approach to understanding insomnia chronicity and the factors that require modification (e.g., excessive TIB) to achieve therapeutic success [6, 15]. We believe that the field is now ripe for a more fine-grained analysis of SRT (a 'zoom lens' approach), to fully elucidate mechanisms and drive treatment refinement and innovation. Below we outline a testable model, specific to SRT, which integrates Spielman's key observations but also introduces new hypotheses based on contemporary understanding of insomnia and basic sleep-wake regulation.

The Triple-R model of SRT: Restricting, Regularising, and Re-conditioning

Insomnia is a psychobiological disorder that can be examined across a number of levels of brain and behaviour. Psychological processes (thoughts, emotions, behaviours) serve to dysregulate sleep biology and in turn, modifications to sleep can feed-back onto key cognitive-behavioural and physiological processes. This interaction maintains insomnia over time. We propose that SRT effectively treats insomnia because it influences cognitive-behavioural and physiological pathways simultaneously and reciprocally. However, it primarily exerts its effects by doing three key things:

1. Restricting time in bed awake,
2. Regularising timing of sleep and wake, and consequently
3. Re-conditioning the association between bedroom factors and sleep.

Targeted modification of these key processes subsequently drives a cascade of change in subsidiary physiological and cognitive-behavioural components that serves to weaken insomnia maintenance factors and restore normal sleep (see Figure 2). The immediate and subsequent effects of the three R's are described below, with respect to both physiological and cognitive-behavioural pathways.

[INSERT FIGURE 2 HERE]

Physiological pathway

During SRT, participants are asked to keep a fixed bed and rise time seven nights a week with only small variations over successive weeks of treatment. Hence, a regular TIB will tighten the regulatory control of sleep by the endogenous circadian pacemaker and standardise exposure to other zeitgebers (i.e. light, meal times). It is recognised that the circadian pacemaker is in permanent interaction with homeostatic sleep pressure [52] and thus a regular TIB will make sleep more predictable and stabilise the circadian sleep-wake cycle [16, 53]. The restricted TIB prescription is based on subjective estimates of participants' TST and will, therefore, result in mild sleep deprivation since people with insomnia tend to underestimate sleep time [54]. Moreover, even if the patient accurately judges TST they would not be expected to sleep 100% of their TIB because good sleepers also spend some time awake during the night [6, 15, 55]. Accordingly, sleep pressure (measured, for example, by SWA) will increase, reflecting a strengthened homeostatic response to accumulated sleep loss [56, 57], which will concurrently feed-back onto the circadian drive [58]. During sleep, homeostatic pressure for sleep dissipates in synchrony with decreasing circadian pressure for wakefulness, possibly leading to a greater alignment between the processes [59, 60]. Consequently, there is little remaining wake pressure throughout the night, which results in a consolidated period of sleep [61]. On a neurobiological level, strong inputs generated by the gradual build-up of the circadian and homeostatic pressures may lead to changes in the *imbalance* between sleep-promoting areas (i.e., ventrolateral preoptic nucleus; VLPO) and arousal-promoting neurons in the lateral hypothalamus [62, 63]. Thus, circadian and homeostatic drives, together, enhance the activity of the sleep-promoting areas and simultaneously inhibit the arousal system, favouring the sleep state [64]. As a result, cortical (e.g., high-frequency EEG) and cognitive arousal (e.g. 'racing mind') prior to sleep and throughout the sleep period will diminish [40, 59, 65]. This strengthened homeostatic sleep drive may suppress *conscious awareness* during NREM sleep

through activity reductions in the left frontoparietal and posterior cingulate, regions that have been associated with cognition and self-referential processes [47, 66]. In healthy participants it is known that sleep restriction principally reduces time spent in stage 2 and REM sleep [67, 68]; sleep stages which have been linked to subjective-objective sleep discrepancy in insomnia [69, 70]. Restriction and consolidation of these sleep stages may, therefore, drive reduction in subjective-objective sleep discrepancies [47, 71]. We assume that SRT takes advantage of relatively intact, though perhaps poorly co-ordinated, homeostatic and circadian processes. In sum, strengthened homeostatic sleep drive, regularised circadian sleep-wake timing and reduced arousal will lead to more consolidated and predictable sleep, and ultimately improved perceptions of sleep.

Cognitive-behavioural pathway

Sleep restriction therapy has parallel effects on cognitive-behavioural processes. Initial restriction of TIB aims to reduce time spent in bed awake which, if adhered to, ensures that bed and sleepiness are paired more consistently than bed and wakefulness, promoting counterconditioning against the typical wake-bed association presumed to characterise insomnia [72]. Over time, SRT changes the conditioning history and therefore increases the probability that sleep-related stimuli will trigger the desired response of sleepiness and sleep [72-74]. Extending TIB or taking a nap to compensate for poor sleep are considered counterproductive safety behaviours that reinforce erroneous beliefs about sleep and, together with sleep related worries, exacerbate insomnia (e.g. ‘I have to stay in bed to get more sleep, I need to nap to cope during the day’) [75, 76]. However, blockade of TIB extension provides the opportunity to directly challenge the need for such behaviour, or specific notions that one needs to sleep eight hours a night. That is, during SRT, sleep can be short but deep and more restorative. Furthermore, partial sleep deprivation caused by decreased TIB potentiates homeostatic sleep pressure to a point whereby sleep will occur inevitably, without attention, intention, or effort; processes which typically serve to dysregulate sleep in insomnia [40, 64, 77, 78]. The accumulation and discharge of sleep pressure also helps demonstrate that the sleep system is not irreversibly damaged and challenges the belief that sleep is entirely uncontrollable [79, 80]. In this way SRT may be considered a behavioural experiment, helping to test out and challenge beliefs presumed to underlie insomnia and related distress. We postulate that targeting such core beliefs, in synchrony with reduced night-time arousal and improved sleep consolidation (direct physiological effects of

restricted TIB), will feedback onto daytime cognitive processes (e.g., sleep-related monitoring) and safety behaviours, and help improve perceptions of daytime functioning.

Becoming a good sleeper

We see the psychobiological interaction as central to the effects of SRT. Modification of both the cognitive-behavioural and physiological pathways serves to improve sleep consolidation, reduce subjective-objective sleep discrepancy and remove (maladaptive) psychological agency. Consequently, those with insomnia will have a positive sleep experience that enhances sleep-related self-efficacy [81]. A feedback loop could arise, whereby the knowledge provided early in the intervention (i.e. 'this is how sleep is regulated', 'it is inevitable that I will sleep', 'I have the capacity to sleep like a good sleeper') may initially increase feelings of sleep self-efficacy and deliver a sense of sleep normalcy. This subsequently motivates the individual to continue to implement the new (and often challenging) schedule, which then delivers improved sleep and further enhances the patient's confidence that they can make positive changes to sleep and behaviour, encouraging further adherence [82]. These processes initially change sleep but over successive weeks begin to modify daytime symptoms [14]. That is, a lag likely exists between sleep and daytime functioning improvement because of the side effects of restricted sleep time, especially at the beginning of treatment [14, 37]. With continued experience of predictable and restorative sleep, alongside sleep window extension and improved daytime function, the insomnia identity is challenged (and ultimately revised) despite only modest increases to TST [12, 83].

Consistent with Spielman [6], we assume that restricted sleep is central to the rapidity and effectiveness of SRT. While SRT has circumscribed targets, it has generalised effects on a range of cognitive-behavioural and physiological processes. We assume, therefore, that SRT has the capacity to address different phenotypes of insomnia, consistent with recent work showing that behavioural insomnia treatments can drive robust cognitive change, similar in magnitude to targeted cognitive therapy [84].

Summary

This review sought to investigate the mechanisms of SRT. Evidence for mechanistic relations between proposed mediators and treatment outcomes is limited, principally because studies have not been specifically designed to elucidate them. We suggest a number of areas for future research with the aim of advancing understanding of

SRT (see research agenda). The *Triple-R* model, based upon Spielman's seminal work, provides a framework specific to SRT from which mechanisms of action can be systematically interrogated through empirical evaluation.

Practice Points

- Understanding treatment mechanisms has the potential to refine treatment protocols and tailor therapies to presenting phenotype and to maximise efficacy
- Mechanisms underlying the effects of SRT have been proposed in the literature but only rarely investigated
- There is a lack of distinction between mediators and outcome variables in the insomnia (and SRT) literature
- A new testable model (Triple-R) is introduced that proposes that SRT primarily exerts its effects by regularising, restricting and re-conditioning TIB, followed by subsequent changes in physiological and cognitive-behavioural pathways

Research agenda

- Adequately powered and controlled evaluations of single-component SRT are needed which embed mechanistic measurements to determine mediating effects on clinical outcomes
- Accordingly, the following four conditions should be tested and satisfied: a) SRT must be related to therapeutic change (e.g. improvement in insomnia severity); b) SRT must be related to the proposed mediator (e.g. homeostatic sleep drive); c) The proposed mediator must be related to therapeutic change and; d) The relationship between SRT and therapeutic change must be reduced after controlling statistically for the proposed mediator
- High resolution and continuous measurement throughout treatment is needed to track trajectories in sleep and putative mediators
- Key pathways of the proposed Triple-R model of SRT should be interogated through the integration of physiological and cognitive-behavioural measurements

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Figure captions

Figure 1:

Prisma flowchart [18] showing the process of selecting studies included in the review.

Figure 2:

The Triple-R model states that SRT exerts its treatment effects by doing three key things: restricting time in bed awake, regularising timing of sleep and wake, and re-conditioning the association between bedroom factors and sleep. Targeted modification of these key processes subsequently drives a cascade of change in subsidiary physiological (e.g. diminished arousal) and cognitive-behavioural factors (e.g. reduced sleep effort) that serve to weaken insomnia maintenance factors and restore normal sleep. Interaction between cognitive-behavioural and physiological pathways improves sleep consolidation, reduces subjective-objective sleep discrepancy and ensures that sleep-related psychological agency (which in insomnia serves to disrupt sleep) surrenders to the two-process model of sleep-wake regulation. Consequently, those with insomnia will have a positive sleep experience that enhances sleep self-efficacy. With continued experience of predictable and restorative sleep, alongside sleep window extension and improved daytime function, the insomnia identity is challenged and ultimately revised.

Tables

Table 1. Sleep window titration according to Spielman et al. (1987).

Sleep Efficiency	=>	**Sleep Window (Time in Bed)**
Less than 85%	=>	Decrease by 15 minutes
85-90%	=>	Keep sleep window
More than 90%	=>	Increase by 15 minutes

Table 2. Theoretical mechanisms of SRT and proposed mediator measurements.

Mechanism	**Measurements**
Perpetuating practices (excessive TIB and napping)	*Time in Bed/ Napping* during T/ at PT (e.g. sleep diary, actigraphy)
Circadian sleep-wake cycle	*Variability in sleep and bedtime parameters* during T/ at PT (e.g. sleep diary, actigraphy) *Markers of circadian rhythmicity* during T/ at PT (e.g. phase/periodicity/amplitude of body temperature)
Homeostatic sleep drive	*Sleep pressure* during T/ at PT (e.g. SOL, SWA, subjective and objective sleepiness)
Hyperarousal	cognitive/ physiological *Arousal* during T/ at PT (e.g. Pre-Sleep-Arousal Scale, cortisol concentration, high-frequency EEG)

**Note*. T = Treatment. PT = Post-Treatment, PSG = Polysomnography, SOL = Sleep onset latency, SWA = Slow wave activity, TIB = Time in bed, EEG = Electroencephalography

Table 3. Description of mediator variables in SRT trials.

Study	**Design**	**Insomnia sample**	**Mediator measures (T&PT)**	**Mediator findings within the SRT group**	**Quality rating**
Bliwise et al., 1995 [23]	SRT vs. RLT	32 elderly people with insomnia (community)	Homeostatic sleep pressure (SOL)	SOL decreased significantly from baseline to post-treatment.	17/31
Brooks et al., 1993 [30]	SRT vs. RLT	9 elderly people with insomnia (community)	Perpetuating practices (TIB) & homeostatic sleep pressure (SOL)	TIB decreased significantly from baseline to post-treatment (sleep diary & actigraphy). SOL showed a trend to decrease (sleep diary).	15/31
Epstein et al., 2012 [24]	SCT vs. SRT vs. MCI vs. WLC	179 elderly people with insomnia (community)	Perpetuating practices (TIB) & homeostatic sleep pressure (SOL)	TIB and SOL decreased significantly from baseline to post-treatment (sleep diary).	18/31
Friedman et al., 1991 [26]	SRT vs. RLT	22 elderly people with insomnia (community)	Perpetuating practices (TIB), homeostatic sleep pressure (SOL) & circadian sleep-wake cycle	The SRT group displayed significant reductions in TIB, TIB variability and SOL from baseline to post-treatment (sleep diary).	14/31
Friedman et al., 2000 [27]	SRT vs. NSRT vs. SH	39 elderly people with insomnia (community)	Perpetuating practices (TIB) & homeostatic sleep pressure (SOL & sleepiness)	TIB and SOL decreased significantly from baseline to post-treatment (sleep diary). Subjective sleepiness decreased from baseline to end of treatment and follow-up, while objective sleepiness seemed to be increased after treatment.	18/31
Kyle et al., 2011 [14]	SRT	18 people with insomnia (community)	Homeostatic sleep pressure (SOL & sleepiness) & arousal	SOL (sleep diary) and sleep effort (indicator for arousal) decreased significantly from baseline to post-treatment. Ninety-four percent of the participants reported “extreme sleepiness” during SRT.	20/31

Kyle et al., 2014 [37]	SRT	16 people with insomnia (community)	Perpetuating practices (TIB) & homeostatic sleep pressure (SOL & sleepiness, RT)	TIB decreased significantly during the first week of treatment and increased by 15min over the following three weeks of the treatment. SOL decreased significantly from baseline to post-treatment (sleep diary). Sleepiness (obtained by ESS and RT) increased significantly from baseline to week one, week two and week three of SRT but decreased at post-treatment.	19/31
Miller et al., 2013 [32]	SRT	9 people with insomnia (community)	Perpetuating practices (TIB), homeostatic sleep pressure (SOL & sleepiness) & arousal	TIB and SOL decreased significantly from baseline to post-treatment (sleep diary). Subjective sleepiness (DISS) increased from baseline to week one but decreased from baseline to week three of SRT. Overall alert cognition decreased from baseline to week one. Alert cognition at bedtime decreased from baseline to week three of SRT.	16/31
Miller et al., 2015 [33]	SRT	11 (8) people with insomnia (community)	Perpetuating practices (TIB), homeostatic sleep pressure (SOL), arousal & circadian timing of core body temperature (N=6)	TIB decreased significantly from baseline to post-treatment (sleep diary). SOL displayed a trend to decrease. Core body temperature decreased significantly, especially in the pre-sleep period. No individual changes in phase or periodicity were detected. Cortisol concentrations in the morning increased from baseline to post-treatment. No change was observed in the pre-sleep phase/early night.	16/31

Spielman, Saskin & Thorpy, 1987 [6]	SRT	35 patients with chronic insomnia	Perpetuating practices (TIB), homeostatic sleep pressure (SOL) & circadian sleep-wake cycle	TIB, SOL and variability in sleep parameters decreased significantly from baseline to post-treatment (sleep diary).	13/31
Taylor et al., 2010 [28]	SRT + HW vs. SH	46 people with insomnia (community)	Homeostatic sleep pressure (SOL)	SOL decreased significantly from baseline to post-treatment (sleep diary).	17/31
Vallières et al., 2013 [34]	SRT	5 people with insomnia (community)	Homeostatic sleep pressure (SOL, SWA & sleepiness) & hyperarousal (high-frequency EEG/cortisol)	SOL decreased in responders from baseline to post-treatment and at the beginning of the treatment (PSG). There was no change in SWA. Subjective sleepiness correlated with sleep improvements. High-frequencies in EEG seemed to decrease. Morning and evening cortisol levels seemed to be lower by the 2nd week of SRT.	16/31
Wang et al., 2015 [29]	PASR vs. SRT	71 patients with chronic insomnia	Perpetuating practices (TIB) & homeostatic sleep pressure (SOL)	TIB and SOL decreased significantly from baseline to post-treatment (sleep diary).	21/31
Whittall, Pillion & Gradisar, 2017 [35]	SRT	16 insomnia participants	Perpetuating practices (TIB) & homeostatic sleep pressure (SOL, sleepiness, RT)	TIB and SOL decreased significantly from baseline to post-treatment (sleep diary). No significant changes in ESS and RT scores from baseline to mid-treatment and post-treatment.	18/31

Note. DISS = Daytime insomnia symptom scale; ESS= Epworth sleepiness scale; HW= Hypnotic withdrawal; MCI = Multicomponent intervention; NSRT = Nap-modified SRT; PASR = Physical activity counselling with SRT; PSG = Polysomnography; RLT = Relaxation therapy; RT = Reaction time; SCT = Stimulus control therapy; SE = Sleep efficiency; SH = Sleep hygiene, SRT = Sleep restriction therapy; SOL = Sleep onset latency; TIB = Time in bed; TST = Total sleep time; TWT = Total wake time