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BRIEF CLINICAL REPORT

Sleep restriction therapy may be effective for people with insomnia and depressive complaints: evidence from a case series

J. Lancee[1,*], M. Maric[2] and J.H. Kamphuis[1]

[1]Department of Clinical Psychology, University of Amsterdam, Amsterdam, The Netherlands and [2]Department of Developmental Psychology, University of Amsterdam, Amsterdam, The Netherlands
*Corresponding author. Email: j.lancee@uva.nl

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Abstract

Background: Insomnia and depression are clearly interrelated. Several studies showed that treating insomnia with cognitive behavioural therapy positively affects concurrent depressive symptoms. However, it is unclear what treatment component is responsible for the change in depressive symptoms.
Aims: To develop the evidence base we employed a case series design in which we administered a single component sleep restriction treatment to individuals with both insomnia and depressive symptoms.
Method: Seven patients were included, of whom six completed the intervention. Patients completed weekly assessments during the 4-week wait period and 6-week treatment phase.
Results: At the post-assessment, two out of six patients showed clinical improvement in depressive symptoms. All six patients showed clinically meaningful improvement at the 3-month follow-up assessment, and two patients had maintained gains at 6-month follow-up.
Conclusions: This case series study shows that, especially at 3-month follow-up, sleep restriction therapy is associated with clinically relevant treatment gains in patients with both insomnia and depressive symptoms.

Keywords: case series; depression; insomnia; sleep restriction treatment

Introduction

With 60–80% of individuals with unipolar depression also having insomnia complaints, these two disorders are clearly inter-related (Ohayon, 2002). It is thought that insomnia plays an important role in the development and maintenance of depression complaints. In line with this, cognitive behavioural treatment for insomnia (CBTI) was also found to be efficacious for depressive symptoms (e.g. van der Zweerde *et al.*, 2019). However, it is unclear what treatment components of the CBTI protocol drive these changes. Traditionally, CBT protocols for depression focus mostly on daily functioning and much less on sleep. It stands to reason that the efficacy of depression treatments may be enhanced if specific and brief sleep treatments can be integrated with conventional CBT protocols for depression.

Accordingly, the principal behavioural ingredient of CBTI, which directly targets sleep/bedtimes, appears to be the most promising aspect of CBTI protocols. We hold that sleep restriction therapy (SRT) may be the best, clearly demarcated, candidate treatment component. The goal of SRT is to increase the homeostatic sleep drive by restricting time in bed and strengthening circadian patterning of sleep–wake through regular bedtime and rising

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time. Importantly, SRT is presumed to be one of the most active treatment components for insomnia (Kyle *et al*., 2015).

Here, we report on a proof of principle study aimed at evaluating SRT for individuals with both insomnia and depressive complaints. In a first step, and to help prepare for more definitive trial designs in the future, we decided to conduct a case series. We used an AB case series design where participants first completed a waiting-period before the commencement of SRT. We formulated the following hypotheses:

(a) relative to the waiting-period, SRT reduces depressive symptoms and insomnia severity, and improves the sleep diary parameters, and
(b) the presumed beneficial effects of SRT would be maintained at 3- and 6-month follow-up assessments.

Method

For a more complete description of the study method and results, please see the online supplementary material.

Inclusion and exclusion criteria

Inclusion criteria were: (1) 18 years or older, (2) a clinical insomnia diagnosis based on SCID-5-RV, (3) Insomnia Severity Index (ISI) score $\geq$ 10, (4) sleep efficiency score $\leq$ 85, (5) Patient Health Questionnaire Depression Scale (PHQ9-D) $\geq$ 10, (6) Beck Depression Inventory II (BDI-II) $\geq$ 14, (7) availability for ten consecutive weeks. Exclusion criteria were: (1) previous CBTI (lifetime), (2) start of psychotherapy <6 months ago, (3) prescribed psychotropic medication, (4) pregnancy/breastfeeding, (5) shift work, (6) severe depressive complaints (BDI-II score $\geq$ 29), (7) probable sleep apnoea (screener), (8) alcohol or marijuana misuse, (9) concrete suicidal ideation (i.e. concrete, specific plans, beyond passive suicidal ideation), (10) lifetime hypomanic/manic period (SCID), (11) current alcohol or drug abuse (SCID section), and (12) current psychosis/schizophrenia (screener/SCID). Other psychiatric or somatic comorbidities were allowed.

Patients

The final sample consisted of four women and three men, ranging in age from 28 to 60 years (mean 45.7). All patients met criteria for a current diagnosis of insomnia and none had a current depressive episode or any other current comorbid axis I disorder. Consistent with the inclusion criteria, the depression scores were in the clinical range (PHQ-9, mean = 12.2; $SD = 2.32$). None of the patients had received prior psychological treatment. All were born and raised in The Netherlands. Please see supplemental Figure 1 for a flowchart of the study.

Procedure

Patients were recruited online via study adverts on Facebook. After inclusion, patients underwent a 4-week waiting period after which the treatment phase, consisting of 6 weeks of single component SRT, began. The post-test, which included a face-to-face assessment, was administered 1 week after completing the SRT protocol. At 3- and 6-month follow-up, assessments included telephone-administered SCID interviews for current depression and insomnia. The study was approved by the University of Amsterdam Ethics Review Board (2017-CP-8305) and was registered at the Netherlands Trial Register (NTR6867).

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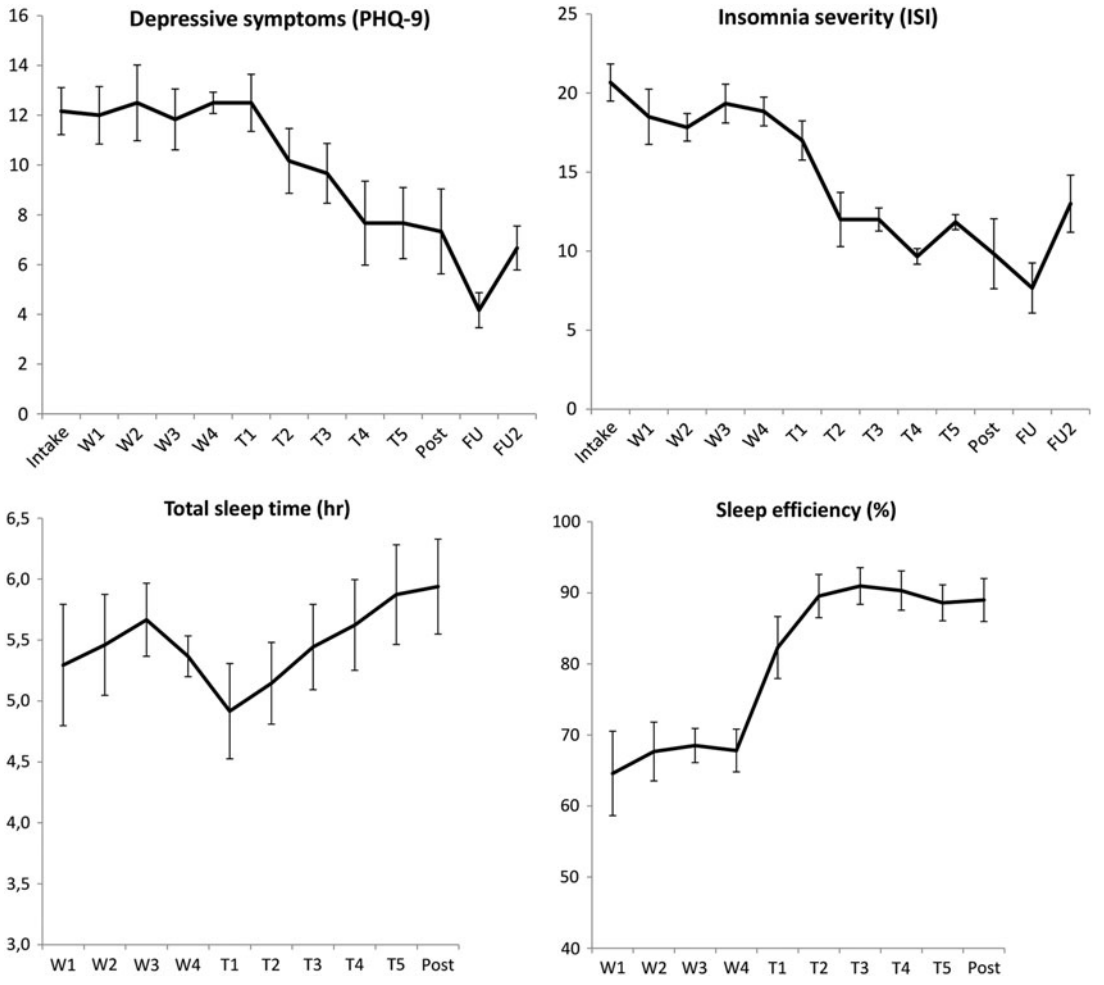

Figure 1. Depressive symptoms, insomnia symptoms, total sleep time and subjective sleep efficiency at a group level ($n = 6$) over time. Please see online supplementary material for a more detailed report at the group level.

Treatment

Patients received 6 weeks of SRT including a sleep diary and weekly assessments (Kyle *et al.*, 2015). In SRT, time spent in bed is restricted to the average reported sleep time in the last week (with a 5-hour minimum). Time in bed is increased when efficiency is ≥85%. SRT started with a face-to-face session explaining the rationale. Each week for the subsequent 5 weeks (one face-to-face and the other via telephone) time in bed was titrated. Per participant, face-to-face therapist time was approximately 90 minutes and 40–60 minutes by telephone.

Assessment occasions and outcome measures

PHQ-9, ISI and BADS-F (not reported here) were measured weekly until post-test and at the follow-up assessments. BDI-II and SCID were administered at baseline, mid-treatment, post-treatment and follow-up assessments. Our primary measure was the PHQ-9 for depressive symptoms. Secondary outcomes were the BDI-II, ISI and the consensus sleep diary (but omitting number of awakenings and terminal wakefulness). We also collected sleep actigraphy data (reported in supplementary material).

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Results

Below, the findings on an individual level are reported. In Fig. 1 the findings at group level are summarized. At the individual level, we determined clinical improvement for the PHQ-9 (50% decrease) and the ISI (change of 8 or more). For a comprehensive description of the results, we refer the reader to the supplementary material. Please note that one patient dropped out from the study, and is therefore not included in the subsequent analyses.

Clinical improvement and diagnosis

Of the six patients who completed the PHQ-9, two demonstrated clinically relevant change at post-test; all six patients reported this improvement at 3-month follow-up, two of which maintained the improvement at 6-month follow-up. For the ISI, the number of patients showing a clinically relevant change were: post-test, $n = 4$; 3-month follow-up, $n = 5$; 6-month follow-up, $n = 2$.

At intake, all six patients met criteria for a DSM-5 diagnosis of insomnia. At 3-month follow-up, none of the patients met criteria for a clinical diagnosis of insomnia, but two patients did meet criteria for the insomnia diagnosis at 6-month follow-up. None of the patients had a clinical diagnosis of depression at intake or at any of the follow-up assessments.

Subjective sleep diary

At post-test, all five patients who kept a sleep diary (SRT25 diary was not admissible) showed more than 10% sleep efficiency increase and more than 60 minutes of decrease in total wake time. Three out of five patients showed $\geq$30 minutes increase in total sleep time. Based on the multi-level estimates, all patients improved on all measures, with only one exception (TST change in SRT37).

Discussion

In this study we set out to investigate whether depressive complaints could be ameliorated by a single-component sleep restriction protocol. To this aim we employed a case series design in which we included seven patients who underwent a 4-week waiting-period followed by a 6-week sleep restriction protocol. Over the waiting-period, all patients remained stable in terms of their symptom severity level. Of the six patients who completed the protocol, two showed a clinically relevant change at post-test on (PHQ-9) depressive symptoms, and all patients showed this change 3 months later. Four patients showed clinically relevant change in insomnia symptoms at post-test, whereas five patients reported this change 3 months later. The efficacy of SRT is further supported by the subjective sleep diary data. Patients reported improvements on these sleep indices with an average 21% increase of sleep efficiency and a decrease in total wake time of 110 minutes.

The observed effects were more pronounced at 3-month follow-up than at the immediate post-test. This finding may well be explained by patients maintaining the sleep restriction protocol beyond the post-test and that they may still have experienced side-effects associated with SRT (Kyle *et al.*, 2014). It is also consistent with the Triple-R model where daytime functioning takes longer to evolve and is at the end of the cascade of factors that SRT is affecting (Maurer *et al.*, 2018).

Of note is that only two participants showed a clinical response on depressive symptoms and insomnia complaints at the 6-month follow-up. In part, this may be explained by two patients who reported a stressful life event at follow-up which may have influenced their results. However, it could also be that the isolated behavioural treatment is not sufficient to implement the strict bedtime changes in the long run. This would be consistent with the observation of Harvey

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and colleagues (2014) that behavioural treatment showed the most pronounced short-term treatment effects and cognitive therapy the most sustained effects (with the combination of the two most effective overall).

For balance, it should be noted that one patient dropped out from the treatment after 2 weeks of sleep restriction and she was therefore not included in the analyses. This patient reported that she suffered too much from the sleep loss associated with the protocol. Even though an adherence rate of 86% (six out of seven) is high for psychological treatments, and all other patients improved, it is important to keep in mind that SRT may be a demanding treatment that may be subject to drop-out, especially in the first 1–2 weeks (because complaints appear to increase during that time).

Other limitations also warrant discussion. First, this study followed an AB case series design. Future studies should employ a multiple baseline design, in which different clients are randomized to different lengths of the baseline period. Either option falls short of the evidentiary power an adequately powered randomized controlled trial would yield. Also noteworthy are the incomplete actigraphy data, not using the full consensus sleep diary, and the self-report only assessment for the apnoea exclusion criterion.

Another limitation is that no participant met criteria for a clinical diagnosis of major depressive disorder (MDD) at baseline. As we used a single-component sleep restriction therapy for the first time in individuals with depressive symptoms, we excluded severely depressed patients, operationalized as individuals with BDI scores $\geq$29. This may help explain why we collected a sample without clinical diagnoses of MDD. Conversely, a strength of this paper was that we included people with at least moderate levels of depressive symptoms, thus meeting a higher cut-off level than employed in earlier studies (e.g. van der Zweerde *et al.*, 2019).

Taken together, we think this study demonstrates proof-of-principle that single component SRT may help reduce symptoms of depression and insomnia. We hold that this study opens up new avenues for the use of SRT as a vehicle for studying the relationship between insomnia and depression. Finally, we recommend empirical testing of SRT's potential as an add-on or preliminary intervention to depression treatment.

Supplementary material. To view supplementary material for this article, please visit: https://doi.org/10.1017/S1352465819000705

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Ethical statements. The authors abided by the Ethical Principles of Psychologists and Code of Conduct as set out by the APA (http://www.apa.org/ethics/code/). The study was approved by the University of Amsterdam Ethics Review Board (2017-CP-8305) and was registered at the Netherlands Trial Register (NTR6867).

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