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Intensive Sleep Retraining treatment for chronic primary insomnia: a preliminary investigation

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SUMMARY The aim of this study was to assess the effectiveness of Intensive Sleep Retraining, a novel, short duration behavioural therapy in treating chronic primary insomnia. Seventeen consecutive volunteers from the general public (mean age = 39.1 years), meeting selection criteria for chronic primary insomnia participated in the treatment study. The study was performed as a case replication series. Assessment involved sleep diary, actigraph and questionnaire measures of sleep and daytime functioning for a period of 2 weeks prior to, immediately after, and 6 weeks following the treatment. Treatment involved a single night of sleep deprivation, facilitating short sleep latencies (mean: 6.9 min) to a series of 50 brief nap opportunities. Following treatment, Sleep Onset Latency significantly decreased by a mean of 30.5 min (SD = 28.3), Wake Time after Sleep Onset significantly decreased by a mean of 28 min (SD = 34.0), and Total Sleep Time significantly increased by 64.6 min (SD = 45.5). Significant improvements were also seen in the daytime functioning and psychological measures of fatigue and vigour, cognitive sleep anticipatory anxiety and self-efficacy for sleep. This brief therapy was effective in improving sleep and some daytime functioning and psychological questionnaire measures. These improvements were maintained up to 2 months following the treatment weekend. Further exploration of this brief therapy is needed, with larger, randomized, placebo-controlled trials over longer follow-up periods, and in comparison to other traditional therapies for insomnia.

KEYWORDS behavioural therapy, brief therapy, intensive sleep retraining, sleep onset insomnia

INTRODUCTION

With prevalence rates of 9–15% (Ancoli-Israel and Roth, 1999; Ohayon, 2002), chronic insomnia is the most common of sleep disorders. Chronic insomnia is associated with significant fatigue, irritability, decreased concentration and memory, increased risk of serious accidents/injuries and increased healthcare use (Balter and Uhlenhuth, 1992; Chesson *et al.*, 2000; Kapur *et al.*, 2002), resulting in both significantly impaired quality of life and economic burden to society (Walsh and Engelhardt, 1999). The latest figures suggest that, in Australia, insomnia results in AUD$30.4 m annually in GP costs alone, with pharmaceutical costs of managing sleep disturbances exceeding AUD$10 m (Access Economics, 2004).

Pharmacotherapy remains the most common treatment for insomnia (Ancoli-Israel and Roth, 1999). However, research suggests that cognitive behavioural therapies (CBT) for insomnia produce significantly greater benefits than pharmacotherapy (Jacobs *et al.*, 2004) and better long-term outcomes (Morin *et al.*, 1994; Morin *et al.*, 1999a). Although benefits gained from pharmacological treatment are immediate, these agents provide symptomatic relief that is not sustained beyond the cessation of treatment (Edinger *et al.*, 2001; Langer *et al.*, 1999). The treatment benefits in response to CBT, in contrast, remain beyond the termination of treatment (National Institutes of Health, 2005). But despite significant experimental support for non-drug therapies, there is some suggestion that

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they need to provide comparatively rapid improvement in sleep if they are to be an appealing alternative to drug therapy (Guilleminault *et al*., 1995).

The non-pharmacological insomnia treatment literature suggests that the most notable improvements in insomnia occur in response to Stimulus Control Therapy (SCT; Bootzin, 1972) (Edinger *et al*., 2001). The goals of SCT are to strengthen the bed, bedroom, bedtime activities and the sleep attempt as cues for sleep, and to extinguish them as cues for wakefulness by curtailing any sleep-incompatible activities (Bootzin and Rider, 2000). This is accomplished by: going to bed only when sleepy, getting out of bed if unable to initiate sleep quickly, arising at the same time each morning, avoiding using the bedroom for activities other than sleep (and sexual activity), and avoiding daytime naps (Bootzin *et al*. 1991). SCT is the most validated behavioural insomnia treatment with the highest level of supporting empirical evidence and is recommended as a 'standard' treatment by the American Academy of Sleep Medicine (Chesson *et al*., 1999).

Stimulus Control Therapy is supported theoretically by the rationale that psychophysiological, or conditioned, insomnia results when the bed and bedroom environment have lost their discriminative ability to evoke sleepiness, and become instead a conditioned stimulus for arousal, thereby inhibiting sleep onset. SCT can then reintroduce the sleep environment as a cue for sleepiness, utilizing a build up of sleep pressure over the early stages of the therapy to assist in the production of sleepiness, resulting in more rapid sleep onsets. Sleep Restriction Therapy (Spielman *et al*., 1987) similarly uses mild sleep deprivation induced homeostatic sleep pressure to promote improvements in sleep (Morin *et al*., 1999b). However, effective behavioural treatment typically takes between 4 and 8 weekly therapy sessions (Smith *et al*., 2002), and considerable investment of time and effort may be required for optimum response to these strategies (Edinger and Wohlgemuth, 1999). These early stages of the therapy may involve truncated sleep, increased sleepiness and fatigue, and resulting problems with compliance.

The various cognitive behavioural treatments for insomnia are a treatment option that takes longer than drugs to be effective, and require considerable motivation to comply with the often-difficult recommendations. Indeed, a lack of compliance to recommendations, a lack of understanding of the procedures or rationale, experience of previous treatment failure, and lack of partner support may all detrimentally impact on the treatment response (Chambers, 1992). As a result, it is desirable for research in this area to aim to provide novel treatment approaches, with the aim of trying to improve upon or complement those currently offered (Harvey and Tang, 2003).

Intensive Sleep Retraining (ISR), essentially a condensed version of a behavioural conditioning treatment, may prove to be one such therapy. A pilot study with four participants, involved a treatment period of a single weekend of sleep deprivation to facilitate the experience of multiple short sleep onset latencies at half hourly opportunities. This study demonstrated an immediate improvement in sleep that lasted for 2 months following therapy (Lack and Baraniec, 2002). The current research aims to further explore this potentially useful new therapy.

MATERIALS AND METHOD

Participants

Requests for volunteers for the treatment study were made to the general public on radio, television and in local newspapers for people aged between 18 and 60 years, to participate in a study involving assessment and treatment. An initial telephone interview identified potential participants with a complaint of sleep onset insomnia or a disorder of initiating and maintaining sleep of at least 6 months duration. Following the initial interview, potential participants were sent a 1-week sleep diary and a sleep history questionnaire with a symptom checklist. Inclusion criteria, based on returned questionnaires and diaries included (i) sleep latency of >30 min, three times in the week, (ii) Total Sleep Time (TST; average) of <7 h and (iii) daytime consequences of poor sleep, including reports of tiredness/fatigue.

Exclusion criteria included any other reported ill health, severe depression [for example, scores of 20 or greater on the Depression, Anxiety and Stress Scale (DASS-21, short form) questionnaire] (Lovibond and Lovibond, 1995), excessive alcohol or caffeine use, regular tobacco use, the use of hypnotic medication or other medications known to affect sleep, and clinical indications of delayed sleep phase syndrome, obstructive sleep apnoea, restless legs syndrome or periodic limb movement disorder. Altogether, 29 applicants were excluded from participation following screening due to depression (four), pain (three), OSA symptoms (two), sleep maintenance insomnia only (four), regular use of sleep medication (two), DPSP symptoms (six), insufficient SOL (five), restless legs symptoms (one) and other non-hypnotic medication use (two). These selection criteria were designed to recruit chronic primary insomniacs with a principal difficulty of initiating sleep, with or without awakenings during the night. In total, 17 consecutive primary insomniacs who met criteria (five males, 12 females) with a mean age of 39.1 years (SD = 12.41) participated in the research. Participants presented with an average subjective pretreatment mean Sleep Onset Latency (SOL) of 69.94 min, TST of 5 h, 17 min and Sleep Efficiency (SE) of 62.44%. Fifty-nine percent of participants also reported sleep maintenance concerns, awake for an average of >60 min overnight. Participants reported suffering from insomnia for an average of 12.5 (SD = 8.75) years prior to study entry. The 24% who had previously used hypnotic medication ceased use at least 1 month prior to treatment. Previous non-drug treatments reported included hypnosis (one), meditation (two), psychological intervention (one). Seventy-seven percent of participants had not previously tried any non-drug treatment methods.

All participants signed informed consent forms prior to commencing the pretreatment assessment period. Ethics approval was obtained from the Social and Behavioral Research Ethics Committee at Flinders University of South Australia.

Apparatus and materials

Sleep-wake diary

The sleep-wake diary is an important index of a successful treatment method (Langer *et al*., 1999). The diary used was a graphical version of a sleep log, and required the recording of lights-out-time, estimated SOL, number and length of awakenings during the night, periods of sleep overnight and final out-of-bedtime. Mean SOL, Wake Time after Sleep Onset (WASO), TST and SE (Total Sleep Time/Time in Bed × 100) were calculated for each 2-week assessment period.

Questionnaires

Although few studies have determined the effects of behavioural therapies on daytime functioning (Edinger and Wohlgemuth, 1999), it is important to assess this aspect of the insomnia complaint. The DASS-21 was used as an indicator of depression, anxiety and stress. The DASS-21 consists of three 7-item self-report scales taken from the full version of the DASS, both versions of which have been demonstrated to be a valid, reliable measure of these constructs in clinical and non-clinical groups (Antony *et al*., 1998). Items include 'I was unable to become enthusiastic about anything', 'I felt scared without any good reason' and 'I tended to over-react to situations', with scores added and doubled for each subscale.

The Dysfunctional Beliefs and Attitudes About Sleep (DBAS) is a frequently used 30-item questionnaire, utilized to assess cognitions about sleep, including sleep loss, sleep need and causes and sequelae of insomnia (Morin *et al*., 1993). The 65-item Profile of Mood States (POMS) was administered in order to obtain the Fatigue and Vigor subscale scores that address these particular daytime functioning aspects (McNair *et al*., 1971). Items included in the list are 'listless', 'lively' and 'worn out', with responses ranging from 0 = 'Not at all' to 4 = 'Extremely' over the past week. The Sleep Anticipatory Anxiety Questionnaire (SAAQ) was utilized as a measure of presleep anxiety, including both cognitive and somatic manifestations of anxiety related to the attempt to initiate sleep. The SAAQ shows high reliability, and significantly differentiates insomniacs from the non-insomniac population (Bootzin *et al*., 1994).

The sleep Self Efficacy Scale (SES) is a 9-item scale to assess an individual's confidence in the ability to influence their own sleep behaviour, with possible scores ranging from 9 to 45 (Lacks, 1987). Responders are required to indicate their confidence in being able to perform sleep-related behaviours, for example, 'Fall asleep at night in under 30 min', on a 5-point Likert scale, ranging from 1 (Not at all confident) to 5 (Very confident). Higher scores on this scale indicate a higher degree of self-efficacy for sleep. Research indicates that changes on this measure reflect treatment-related improvement in sleep (Spielman *et al*., 1987). The questionnaire was only introduced half way through the study, and was thus administered to only nine participants. The Stanford Sleepiness Scale (SSS) was administered prior to each trial over the treatment procedure as a measure of current subjective sleepiness (Hoddes *et al*., 1973). This is a frequently used measure of sleepiness, consisting of a choice from a range of descriptives from 1 = 'Feeling active and vital; alert; wide awake' to 7 = 'Lost struggle to remain awake'.

Actigraphy

Participants wore an ActiTrac activity monitor (IM Systems, Baltimore, MD, USA) on their non-dominant wrist during assessment periods. Activity counts were averaged for each 30-s epoch, recorded and downloaded for analysis. Actigraphy sleep onset time was estimated as the start of the first period of at least 15 consecutive minutes of no wrist activity after lights out time (Jean-Louis *et al*., 1996). Amount of WASO was estimated from an algorithm using the number of epochs with movement from initial sleep onset to final wake up time (as indicated by the subject's movement, corroborated by sleep diary). Automatic analyses were utilized to produce scores for TST across the period in bed, determined from the time of estimated sleep onset to the time of final wake up, minus WASO. Sleep Efficiency was calculated by dividing the TST by the time in bed, multiplying it by 100. Actigraphic sleep estimates have been demonstrated to be moderately related to polysomnography measures of sleep in insomnia sufferers, and as such it has been suggested that actigraphy can be a reliable indicator of objective changes in sleep over time within participants with insomnia undergoing behavioural therapy, with the advantage of inexpensively providing data across multiple nights (Ancoli-Israel *et al*., 2003).

Polysomnography

Participants were monitored throughout the ISR sleep latency treatment trials utilising electroencephalography (EEG; CZ-OZ bipolar placement) and electro-oculography (EOG). EEG was used to determine sleep onset during treatment using LABVIEW5 (a modified version of the Laboratory Virtual Instrument Engineering Workbench; National Instruments Corporation, Texas, USA), a computer software program used to graphically quantify raw EEG power within different frequency bandwidths. This method has been validated against standard raw EEG defined SOL on the Multiple Sleep Latency Test ($r = +0.90$; Lack *et al*., 2003). Sleep onset was determined in accordance with conventional criteria as described in Rechtschaffen and Kales (1968). Sleep onset was deemed to occur when the alpha power reduced to less than one half of the average relaxed wakefulness baseline for three consecutive epochs. This three consecutive epoch criterion is thought to

identify unequivocal sleep, and is advocated for experimental use (Carskadon, 1986).

Procedure

Pretreatment

After a full explanation of the study and a tour of the Sleep Laboratory, participants completed the DASS questionnaire. After this final screening questionnaire and familiarisation with the Sleep Laboratory, participants completed a 2-week baseline assessment period using sleep/wake diaries and actigraphy.

Treatment

Participants arrived at the Sleep Laboratory at 18:00 hours, allowing time for EEG and EOG placement, equipment calibrations and participant settling time (approximately 30 min). Participants were required to spend the treatment period in bed, with the exception of bathroom visits. The treatment, scheduled to begin at approximately 2 h prior to habitual bedtime (range: 19:30–21:00 hours), involved a series of 'sleep trials'. Subjective sleepiness was monitored immediately prior to each trial by the SSS. For each sleep trial participants were provided instructions to 'lie down, relax and let yourself fall asleep'. Lights were then extinguished, the bedroom door was closed, and the experimenter retired to an adjoining monitoring room. Participants were then monitored via EEG for sleep onset, then being allowed to sleep for at least 1.5 min, up to 4 min (Average: 3.1 min, SD = 1.2). The duration of sleep allowed in each trial was limited in order to maintain homeostatic sleep drive and short sleep latencies. If sleep was not achieved in 25 min the trial was terminated. The following trial began 30 min following the initiation of the previous trial. Thus, sleep onset trials were conducted at each half hour time point throughout the treatment period. Each participant completed a minimum of 50 sleep onset trials during the therapy period (range: 50–56). Treatment ceased with sufficient time to travel home prior to habitual bedtime.

In between trials, participants remained in semi-supine or supine position in bed, awake and engaging in minimal activity such as reading or watching video films. If a participant appeared to have difficulty maintaining wakefulness during the inter-trial wake periods, quiet conversations initiated by the experimenter avoided inadvertent sleep onsets. No time cues were provided during the routine, and no feedback was provided as to whether the participants met criteria for sleep onset during the trials. The ambient temperature was kept constant at 22 °C and room illumination was 50 lux, measured at the head of the bed. Small portions of food were provided every 2 h to satiate hunger, but to avoid the soporific effects of large meals on sleepiness. Following the therapy procedure, participants were allowed to return home to obtain a recovery sleep (with an allotted bed period maximum of 8 h), beginning at a time as close as possible to the participants' habitual bedtimes.

Post-treatment

The 2-week post-treatment monitoring was initiated following two nights of recovery sleep after the treatment routine, implemented to exclude effects of acute sleep deprivation on sleep variables (Bonnet, 1985). A further 2-week follow-up period was undertaken beginning at 6 weeks after treatment. Questionnaires were completed at the end of every 2-week assessment period. Following the final assessment, participants were paid a nominal reimbursement fee to contribute towards accumulated travel costs.

RESULTS

Treatment protocol

Figure 1 illustrates the mean SOL for participants, averaged at each half hourly time point over the treatment period. As participants started treatment approximately 2 h prior to habitual bedtime, some initial and final trial times include fewer than 17 participants. The maximum of 17 participants were included from 21:00 hours on night 1 until 21:00 hours on night 2 of treatment. The sleep onset criteria were satisfied in 93% of the sleep trials, indicating the success of the treatment protocol in allowing the repetitive experience of falling asleep. The mean SOL for all trials was 6.9 min (SD = 3.6). The

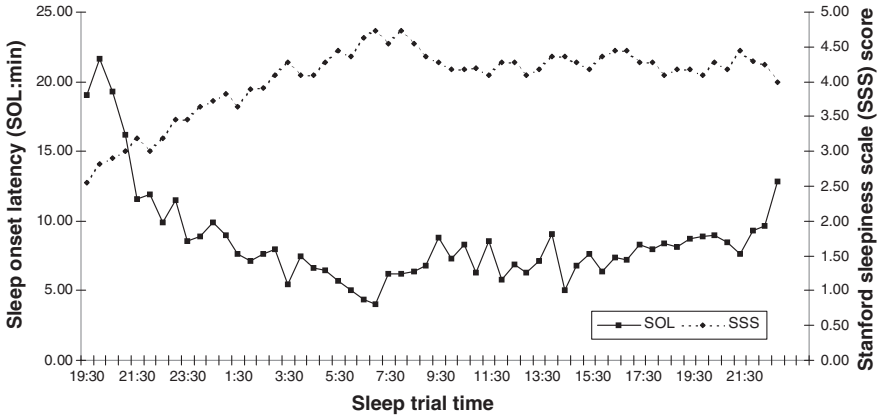

Figure 1. Mean Sleep Onset Latency and Stanford Sleepiness Scale scores at each sleep trial time throughout the treatment procedure.

average TST within nap opportunities was 3.1 min (SD = 1.2). Those few trials that were terminated after 25 min without achieving sleep onset were almost entirely restricted to the first few hours of the treatment period. The figure also demonstrates the usefulness of the routine in providing a large number of rapid sleep onsets. On average, 32.0 trials per participant contained a SOL value of < 5 min in length.

The figure demonstrates that the mean SSS for each trial increased over the procedure, indicating a subjective increase in sleepiness, in addition to the increased objective sleepiness (decreased sleep onset latencies).

Treatment response

Response to treatment in subjective (sleep diary) and objective (actigraphic) estimates of sleep parameters, and questionnaire variables was determined by examining the change from baseline (pretreatment) values (see Table 1) using repeated measures ANOVAS. The assumptions of ANOVA were largely met, with Q-Q plots confirming the normality of the residuals for each variable. For those variables that violated sphericity assumptions, a Greenhouse-Geisser adjustment has been utilised. *Post hoc* analyses examined the significance of the difference between pretreatment and both post-treatment and 6-week follow up.

Sleep Onset Latency

There was a significant change over time in mean subjective SOL (see Fig. 2). Mean SOL decreased significantly by 30.4 min from the pretreatment mean value of 69.9 to 39.5 min post-treatment. At the 6-week follow up the improvement in SOL remained significant. Objective mean SOL decreased with marginal statistical significance over time, from 47.8 min pretreatment to 34.6 min at post-treatment. *Post hoc* analyses indicated that the mean objective SOL value at follow up (38.1 min) was no longer significantly different from baseline (Table 1).

Wake Time after Sleep Onset

A significant change over time was also demonstrated in subjective WASO figures (see Table 1). The mean subjective WASO was significantly reduced by 28 min at post-treatment, and 34.5 min at follow up. Although actigraphy measured

Table 1 ANOVA and *post hoc* analyses for treatment-related changes on sleep parameters

	Pretreatment		*Post-treatment*		*Follow up*				
Measure	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*F*	*d.f.*	*P*-value
Sleep Onset Latency (min)									
SOL (sleep diary)	69.94	(35.38)	39.46****	(18.71)	47.05**	(26.36)	14.84	1.4	0.000
SOL (actigraph)	47.80	(31.33)	34.60*	(25.47)	38.06	(32.06)	4.15	10	0.049
Total Sleep Time (min)									
TST (sleep diary)	317.20	(80.19)	381.76****	(68.90)	359.82**	(93.11)	16.46	15	0.000
TST (actigraph)	356.08	(70.46)	384.00*	(55.11)	361.78	(64.15)	2.92	10	0.100
Wake After Sleep Onset (min)									
WASO (sleep diary)	87.49	(63.03)	59.46**	(53.88)	53.02**	(56.02)	6.18	15	0.011
WASO (actigraph)	104.43	(75.15)	102.67	(68.36)	90.58*	(61.89)	3.5	10	0.073
Sleep Efficiency (%)									
SE (sleep diary)	62.44	(15.85)	75.71****	(13.64)	73.70****	(14.95)	22.66	15	0.000
SE (actigraph)	69.85	(14.71)	72.81*	(15.57)	74.57***	(13.51)	10.50	10	0.003

Asterisks indicate *post hoc* significant changes from pretreatment: *$P < 0.05$, **$P \leq 0.01$, ***$P \leq 0.001$, ****$P < 0.0001$.

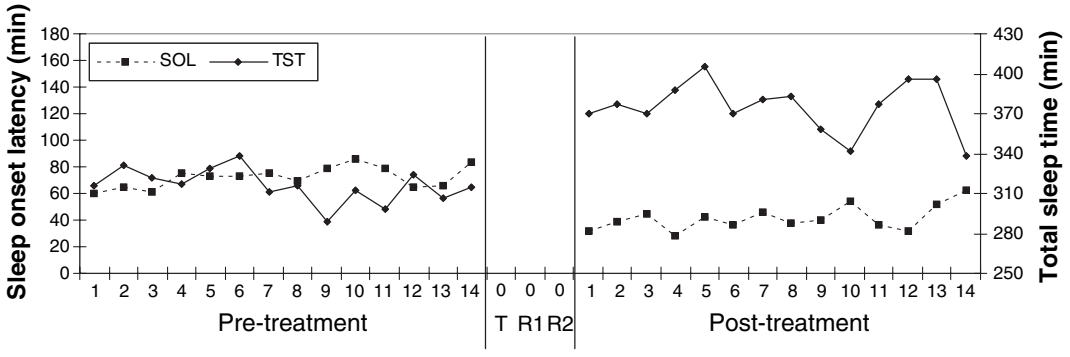

Figure 2. Mean daily Sleep Onset Latency and Total Sleep Time immediately prior to, and following T (treatment), R1 (recovery night 1) and R2 (recovery night 2).

WASO showed a reduction by the follow-up period (see Table 1) it was of minimal clinical importance (13.8 min).

Total Sleep Time

Subjective TST showed a statistically significant change over time (see Fig. 2). Mean TST significantly increased from a pretreatment mean value of 5 h, 17 min to a post-treatment value of 6 h and 21 min, an average increase of 65 min. This improvement from baseline was maintained at follow up, with an average increased TST by 42.6 min. The 28-min increase in TST as indicated by actigraphy at post-treatment, was no longer significantly different from baseline by follow up.

Sleep Efficiency

A significant improvement over time was observed for subjective SE, improving by 13% to post-treatment, with an improvement from baseline of 11% maintained at follow up (see Table 1). Objective SE improved only slightly from pretreatment by a mean of only 2.9% to post-treatment, and 4.7% to follow up. No significant changes over time were found in either the objective or subjective time in bed component of SE.

Immediate treatment response

In order to assess and highlight the immediacy of response following treatment, Fig. 2 shows average daily SOL and TST variables both before treatment and immediately following the recovery nights. The graph indicates that an immediate change in sleep variables following treatment is maintained throughout this post-treatment assessment period.

Profile of Mood States

Analyses indicated that the POMS Fatigue subscale scores reduced following treatment, with only the immediate post-treatment change being significantly reduced from baseline (see Table 2). Marginally significant changes in the POMS Vigour subscale scores indicated that vigour increased following treatment and was maintained at follow up.

Sleep Anticipatory Anxiety

The overall change in the total SAAQ score was only approaching statistical significance (see Table 2). However, as previous research has indicated that cognitive factors often seem more important in insomnia than somatic factors (Nicassio *et al.*, 1985), the somatic and cognitive items were evaluated separately. Although the somatic items in this scale did not significantly change over time, significant reductions were seen in cognitive sleep anticipatory anxiety from pre-treatment to post-treatment and follow up.

Dysfunctional Beliefs and Attitudes About Sleep

Although no overall significant main effects for time were found in the DBAS variable, *post hoc* analyses indicated trends for improvement following treatment.

Sleep Self Efficacy Scale

The SES measure showed a significant increase in sleep self-efficacy scores over time, indicating improved self-efficacy for sleep following treatment (see Table 2).

Table 2 ANOVA and *post hoc* analyses for treatment-related changes on questionnaire measures

	Pretreatment		*Post-treatment*		*Follow up*				
Questionnaire	*Mean*	*SD*	*Mean*	*SD*	*Mean*	*SD*	*F*	*d.f.*	P-*value*
Profile of Mood States (POMS)									
Fatigue subscale	14.40	(6.86)	8.17**	(6.28)	10.87	(7.84)	6.457	13	0.011
Vigour subscale	12.067	(6.76)	17.50*	(6.65)	16.87*	(6.82)	3.796	13	0.05
Sleep Anticipatory Anxiety (SAA)									
SAA total score	14.81	(4.94)	12.13	(5.00)	11.56	(5.55)	3.76	1.35	0.056
SAA cognitive score	10.13	(3.56)	7.81**	(2.68)	7.13***	(3.16)	10.78	1.45	0.001
SAA somatic score	5.25	(2.08)	4.31	(2.80)	4.31	(2.98)	0.711	14	0.508
Dysfunctional Beliefs and Attitudes about Sleep (DBAS)									
DBAS total score	79.19	(16.09)	72.44*	(15.41)	72.09	(16.78)	3.09	1.36	0.083
Sleep Self Efficacy (SES)									
SES total score	20.2	(4.93)	28.2**	(5.77)	26.95**	(4.87)	8.128	8	0.012
Depression, Anxiety, Stress Scale (DASS)									
Depression	8.38	(8.33)	5.44	(8.25)	5.38	(9.00)	0.924	14	0.420
Anxiety	4.13	(3.22)	2.63	(3.07)	3.38	(4.99)	1.516	14	0.254
Stress	15.5	(11.33)	10.88	(5.21)	11.63	(7.01)	2.21	1.26	0.15

Asterisks indicate *post hoc* significant changes from pretreatment: *$P \leq 0.05$, **$P \leq 0.01$, ***$P \leq 0.001$, ****$P < 0.0001$.

Depression, Anxiety Stress Scales

Although all of the subscales (depression, anxiety and stress) of the DASS-21 showed mean decreases over time, the changes were not statistically reliable in this study.

DISCUSSION

The current study was designed to investigate a novel behavioural therapy for primary insomnia, ISR. The treatment protocol was successfully applied, resulting in a large number of rapid sleep onsets over the treatment period.

Following this single laboratory treatment session, there were significant improvements in objective and subjective sleep parameters, and some daytime functioning and psychological variables. The improvements of sleep shown in this study were very similar to those in an earlier pilot study ($N = 4$) using the ISR treatment protocol. The subjective sleep variables demonstrated the greatest improvement in sleep, with sleep latency reducing by 30 min, TST increasing by over an hour, and wake time overnight reducing by almost 30 min immediately after the treatment. For the most part, these improvements were maintained up to 2 months following treatment. This degree of improvement was comparable, at least in the short-term, to that obtained from non-drug therapies in the literature. For instance, meta-analyses indicate that diary reported SOL decreases from an average of 60–65 min to 35 min following treatment (Morin *et al.*, 1994, 1999a; Murtagh and Greenwood, 1995). Improvements in subjective TST in response to ISR even exceeded improvements following other therapies, which research indicates tends to be in the range of 30 min (Morin et al., 1999a).

There are a number of advantages to the provision of non-drug insomnia treatment in a rapid manner. The ISR protocol allows treatment implementation in a single sitting, which then counters issues regarding the length and potential difficulty of present non-drug therapies. Although some clinical research trials of SCT have demonstrated an early improvement in SOL for some clients (Turner and Ascher, 1979; Espie *et al.*, 1989), the rapid treatment response in the present study indicated improvement in all sleep variables immediately after treatment without concomitant reductions in bed period. In that respect it is comparable with pharmacotherapy in terms of rapidity of response. The ISR procedure, involving increased sleepiness and frequent awakenings from sleep during treatment, although procedurally onerous, was associated with only occasional reports of side effects (e.g. headache and irritability). The procedure was reasonably well tolerated by participants, and all were able to complete the protocol, indicating the potential for a low drop-out rate from treatment completion. Future systematic reporting of side effects in a larger study would, however, be useful.

Nevertheless, despite immediate improvements of a significant magnitude, it must be noted that post-treatment values, on average, leave room for improvement. The implementation of the ISR therapy, in isolation of any other treatment, resulted in a SE improvement of 11% from baseline, up to 74% 2 months after treatment. Although clinically significant improvement has been defined in research as a SE ≥80–85% (Morin et al., 1999a), the lower final SE values may reflect the severity of insomnia symptoms in this group. Absolute improvement compares with the response to multifaceted psychological intervention (e.g. 12.3%; Morin *et al.*, 1994). Suboptimal treatment response from this protocol may have been a result of not providing any recommendations regarding sleep hygiene (Morin et al., 1999a), or further treatment instructions to follow upon completion of the ISR. It is possible that behaviours that had previously precipitated and/or maintained the insomnia were still present after therapy, thereby predisposing to a relapse of poor sleep. However, this design was desired to determine the impact of this rapid conditioning treatment in isolation from the impact of other treatment. Further research may provide data on whether the rapid response seen after a single treatment period can be further enhanced by therapy instructions, such as adjunctive SCT after the implementation of ISR.

The aspects of the treatment protocol that may contribute to the post-treatment improvements are a matter of speculation. The treatment was designed to decondition the insomnia arousal response to the sleep onset attempt that is typically observed in primary insomnia, or correspondingly, to recondition positive cues for sleep onset. This process is presumed to be similar to that hypothesized for SCT or Sleep Restriction Therapy. In SCT, sleep restriction typically occurs in the first weeks of the therapy and increases homeostatic sleep drive, gradually facilitating reduced sleep latencies. This behavioural insomnia treatment typically takes a few weeks to produce a treatment response. The conditioning cues for sleep onset may be such factors as the bedroom environment, temporal and sleepiness cues, and attempts to initiate sleep, including lights being turned off, and closing the eyes. Some of these cues are also present in the Sleep Laboratory environment. Thereby, an expectation of a generalization of the ISR treatment response to the home environment is, perhaps, reasonable. However, future research may be aimed towards providing this kind of rapid treatment within the home environment, allowing a greater specificity in conditioning, as well as a reduced cost of treatment.

Improvements may have been facilitated by perceptions of having fallen asleep at the termination of sleep trials. The average nap length of 3.1 min was greater than the 1.5 min allowed in the original pilot study. Following the nap, the participants may perceive an awakening from sleep thus confirming they had, in fact, fallen asleep. This alone, may be therapeutic. If the degree to which the procedure is therapeutic is dependent upon perception of having fallen asleep, this will have important implications for the conditioning model. For example, if there is no therapeutic benefit for participants who did not perceive sleep onsets despite rapid sleep attainment, the purely behavioural conditioning theoretical basis for treatment may be challenged. Although the perception of sleep was not formally assessed, nor was

confirmatory feedback of sleep onset given, this may be a worthwhile avenue of future research. Further research with larger sample sizes may also attempt to determine the relative therapeutic importance of the number of trials in which sleep is attained, and the rapidity of those sleep onsets.

The near-total sleep deprivation occurring during the treatment also needs consideration for its potential therapeutic benefit. Although this degree of sleep deprivation would be expected to improve sleep for one or two nights, this effect should be temporary. The inclusion of two nights of recovery sleep before the start of the post-treatment assessment should have eliminated the acutely heightened homeostatic sleep drive as a contributor to improved sleep during the 2-week post-treatment assessment period (Bonnet, 1985; Lorenzo *et al*., 1995). There may also be a key component of treatment arising from the experience of sleep deprivation. The sleep deprivation and sleepiness experienced during the treatment period may have been less adverse than participants expected, resulting in a decreased fear of sleeplessness. Future research may examine the potential for improved sleep following total sleep deprivation, without the use of sleep onset trials, thereby testing the specific efficacy of the sleep trials in promoting improved sleep.

The potential for improvement to be due to a placebo effect is unable to be ruled out in the current study. Likewise, the small sample size in this investigation, and the limited length of follow up, restricts the conclusions that can be drawn from the results. Future research should evaluate this treatment with greater participant numbers, utilizing a randomised placebo-controlled design to further explore the utility of ISR.

Further investigation of ISR therapy is warranted. Immediate improvements in sleep and daytime functioning, comparable to other non-drug treatments, were observed after the 25–28 h treatment regime. Indeed, the rapidity of response seen in this study improves upon the speed of response that is generally seen with behavioural therapies. Further research may compare ISR to other individual behavioural therapies, as well as ISR in combination with other behavioural therapies.

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