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A Randomized Controlled Trial of Intensive Sleep Retraining (ISR): A Brief Conditioning Treatment for Chronic Insomnia

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Study Objective: To investigate the effectiveness of intensive sleep retraining in comparison and combination with traditional behavioral intervention for chronic primary insomnia.

Participants: Seventy-nine volunteers with chronic sleep-onset insomnia (with or without sleep maintenance difficulties) were randomly assigned either to intensive sleep retraining (ISR), stimulus control therapy (SCT), ISR plus SCT, or the control (sleep hygiene) treatment condition.

Intervention: ISR treatment consisted of 50 sleep onset trials over a 25-h sleep deprivation period.

Measurements and Results: Treatment response was assessed with sleep diary, activity monitoring, and questionnaire measures. The active treatment groups (ISR, SCT, ISR+SCT) all resulted in significant improvements in sleep onset latency and sleep efficiency, with moderate to large effect sizes from pre- to post-treatment. Wake time after sleep onset decreased significantly in the SCT and ISR+SCT groups. Total sleep time increased significantly in the ISR and ISR+SCT treatment groups. Participants receiving ISR (ISR, ISR+SCT) experienced rapidly improved SOL and TST during treatment, suggesting an advantage of rapid improvements in sleep in response to ISR. Although there were few statistically significant differences between groups on individual variables, ISR+SCT resulted in consistently larger effect sizes of change than other treatments, including questionnaire measures of sleep quality, sleep self-efficacy, and daytime functioning. The combination treatment group (ISR+SCT) showed trends to outperform other active treatment groups with fewer treatment dropouts, and a greater proportion of treatment responders with 61% reaching "good sleeper" status. Treatment gains achieved at post-treatment in the active treatment groups were largely maintained throughout follow-up periods to 6 months.

Conclusion: This 25-hour intensive conditioning treatment for chronic insomnia can produce rapid improvements in sleep, daytime functioning, and psychological variables. Adding ISR to traditional interventions seems to result in a superior treatment response.

Keywords: Chronic insomnia, classical conditioning, behavioral treatment, intensive sleep retraining

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INTRODUCTION

Chronic insomnia is a pervasive, unrelenting sleep disorder, and is associated with considerable consequences for sufferers. Various conceptual models contribute an understanding of the development and maintenance of insomnia, in addition to helping inform treatment choice. Of those, a conditioning model offers a theoretical basis for the efficacy of behavioral intervention. This model suggests that the wakeful state associated with insomnia may be learned through a process of conditioning.[1] According to this model, transient sleep disturbance may be triggered by acute periods of stress or heightened arousal. With repeated episodes of wakefulness and distress in bed, an individual may rapidly associate the bedroom, bedtime, and other associated cues with anxiety and sleeplessness. Thus both temporal and contextual stimuli may become cues for apprehension, worries, and fear of being unable to sleep, in addition to the sleeplessness itself. Research suggests that once insomnia is established, it may persist over many years.[2,3]

Although a significant body of research supports cognitive behavioral treatments for insomnia, this common sleep disorder often remains untreated, or treated only with pharmacotherapy.[4] Of the behavioral interventions, stimulus control therapy (SCT) is the most widely studied and endorsed single component treatment method. Indeed, SCT is currently recommended with the highest standard of support by the American Academy of Sleep Medicine.[5] SCT is considered effective for both sleep onset and sleep maintenance insomnia symptoms.[6,7] The assumed mechanism involved in successful SCT implementation is the eventual conditioning of a rapid sleep onset, countering the learned psychophysiologic arousal (or absence of de-arousal[8]) associated with the insomnia response.

Nevertheless, the administration of behavioral treatment for insomnia is typically associated with a lag in treatment response, some early treatment sleepiness and/or fatigue, and some difficulties with treatment compliance. Consequently, the rapid treatment response associated with intensive sleep retraining (ISR)[9] may improve the response to and compliance with non-drug interventions. ISR is a brief 25-h conditioning treatment, involving the use of acute sleep deprivation to facilitate a series of rapid sleep onsets in an effort to counteract the conditioned insomnia response. In a case series study, this brief conditioning treatment has shown rapid and sustained improvements in sleep variables.[9]

The current study aims to compare the treatment response to both ISR and SCT treatments alone, in addition to evaluating a combination of the 2 treatment methods in comparison to a control (sleep hygiene intervention only) group.

A commentary on this article appears in this issue on page 11.

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Table 1—Sample demographic and clinical characteristics, mean (standard deviation)

Variable	ISR (*N* = 19)	SCT (*N* = 20)	ISR+SCT (*N* = 20)	Control (*N* = 20)	All (*N* = 79)
Age (SD)	38.53 (13.3)	42.2 (13.1)	36.9 (11.5)	43.8 (13.8)	40.9 (13.8)
Gender (M:F)	8:11	5:15	3:17	7:13	23:56
Length of insomnia (yr)	16.8 (12.8)	12.1 (10.7)	17.9 (13.2)	16.2 (15.4)	16.0 (13.6)
SOL at screening (min)	74.5 (31.4)	82.6 (53.5)	67.7 (24.5)	92.1 (63.8)	81.7 (50.3)
Pre-treatment WASO (min)	83.4 (58.2)	74.3 (47.2)	85.7 (52.4)	94.6 (69.9)	84.5 (56.9)
Insomnia Severity (Mild: Severe)	(9:10)	(10:10)	(10:10)	(10:10)	(39:40)
Body Mass Index (BMI)	25.2 (5.4)	24.2 (2.5)	25.9 (5.2)	25.8 (6.2)	25.6 (5.5)
Respiratory Disturbance Index (RDI)	4.89 (4.5)	6.27 (4.9)	6.33 (3.3)	6.77 (4.9)	6.17 (4.4)
Mean BDI-II Depression score (adjusted)	8.63 (6.2)	9.05 (7.2)	9.65 (7.2)	7.47 (5.7)	8.55 (6.5)

MATERIALS AND METHODS

Participants

Potential participants were recruited from the community, primarily via newspaper advertisements, media releases, and pamphlet distribution to general medical practices. Figure 1 illustrates participant flow through screening and treatment. Following an initial telephone interview, 142 respondents were mailed and returned a one-week sleep-wake diary and comprehensive sleep questionnaire. Of these, 45 were excluded and 97 underwent screening overnight polysomnography.

Participants reported sleep onset insomnia, with or without concurrent difficulty maintaining sleep. Selection criteria, based on screening stages included (i) age between 18-65 years, (ii) mean sleep diary sleep onset latency > 30 min, for ≥ 3 nights per week, (iii) insomnia symptoms for ≥ 6 months, and (iv) reported daytime consequences of poor sleep. Exclusion criteria were other sleep disorders (e.g., obstructive sleep apnea, delayed sleep phase disorder, periodic limb movement disorder), other medical or psychiatric disorders are likely to affect sleep and daytime functioning (e.g., chronic pain, bipolar disorder, chronic fatigue syndrome), pregnancy, being a heavy smoker, or taking medications likely to affect sleep. Those with significant depression were excluded on the basis of an adjusted Beck Depression Inventory (BDI-II) score > 28. The adjusted scoring method involved excluding items 15, 16, and 20 from the total score—items that reflect sleep disturbance, fatigue, and loss of energy—which are also symptoms of insomnia. Of note, individuals taking daily antidepressant medication (stable dosage for 3 months) were considered suitable for inclusion, provided they were willing to continue on the same dose for the period of the study, and met inclusion criteria on the BDI-II. Participants using sleep medication were considered for inclusion following a withdrawal and wash-out period of ≥ 3 weeks (8-10 of the participants in each group).

A total of 79 participants (23 males, 56 females, mean age 40.9 years [SD 13.8], mean length of insomnia 16.0 years [SD 13.6]) were considered appropriate for inclusion and were accepted into the treatment study.

Suitable participants were stratified according to the severity of their insomnia into a severe (average sleep onset latency [SOL] > 60 min), or mild to moderate classification (SOL ≤ 60 min), on their one-week screening sleep diary. Participants were randomly allocated to the 4 treatment conditions using a computer generated randomization program, with allocations stratified by severity. The demographic and clinical characteristics of the treatment groups are presented in Table 1. There were no significant differences between treatment groups on age, length of insomnia, BMI, RDI, SOL at screening, pre-treatment wake time after sleep onset (WASO), or BDI-II adjusted score.

All participants signed informed consent forms prior to research participation. Ethical approval was obtained from the Social and Behavioural Research Ethics Committee at Flinders University of South Australia.

Apparatus and Materials

Sleep-wake diary

The current study employed a chart version of a sleep-wake diary to provide the primary outcome measures of subjective sleep variables. The sleep-wake diary facilitates a graphical daily recording of lights out time, sleep onset time, estimated periods of sleep overnight, final wake-up time, and out of bed time, as well as estimates of SOL and WASO. Across each 24-h period, food, caffeine, daytime naps, and alcohol were recorded by means of symbols at the appropriate times. Sleep-wake diaries were completed daily for the 2-week baseline period, throughout treatment, and during follow-up assessments.

Actigraphy

Given the discrepancy between objective and subjective sleep that has been reported in insomnia, it was considered important to include actigraphy in the assessment process, in order to provide a more objective estimate of nightly sleep. Participants wore an Actiwatch AW-64 (Mini Mitter Co. Inc., Bend, Oregon) on their non-dominant wrist throughout the day and night. Event markers provided the times of retiring to bed, and out of bed times. Total sleep time (TST), WASO, and sleep efficiency (SE) were estimated using the Actiware Sleep Software version 3.4.

Although actigraphy has been validated in insomniacs, sleep onset latency estimates derived from this measure have been significantly underestimated.[10] Accordingly, two methods were used in an attempt to provide a clinically useful measure in the current study. Firstly, all Actiwatch recordings were scored using a manually adjusted sensitivity setting, derived for each individual by equating actigraphy-scored TST to that of the overnight screening PSG-scored TST on the same night. Secondly, the Actiwatch SOL was manually estimated as the start of the first period ≥ 20 consecutive minutes with no more than one epoch of movement.

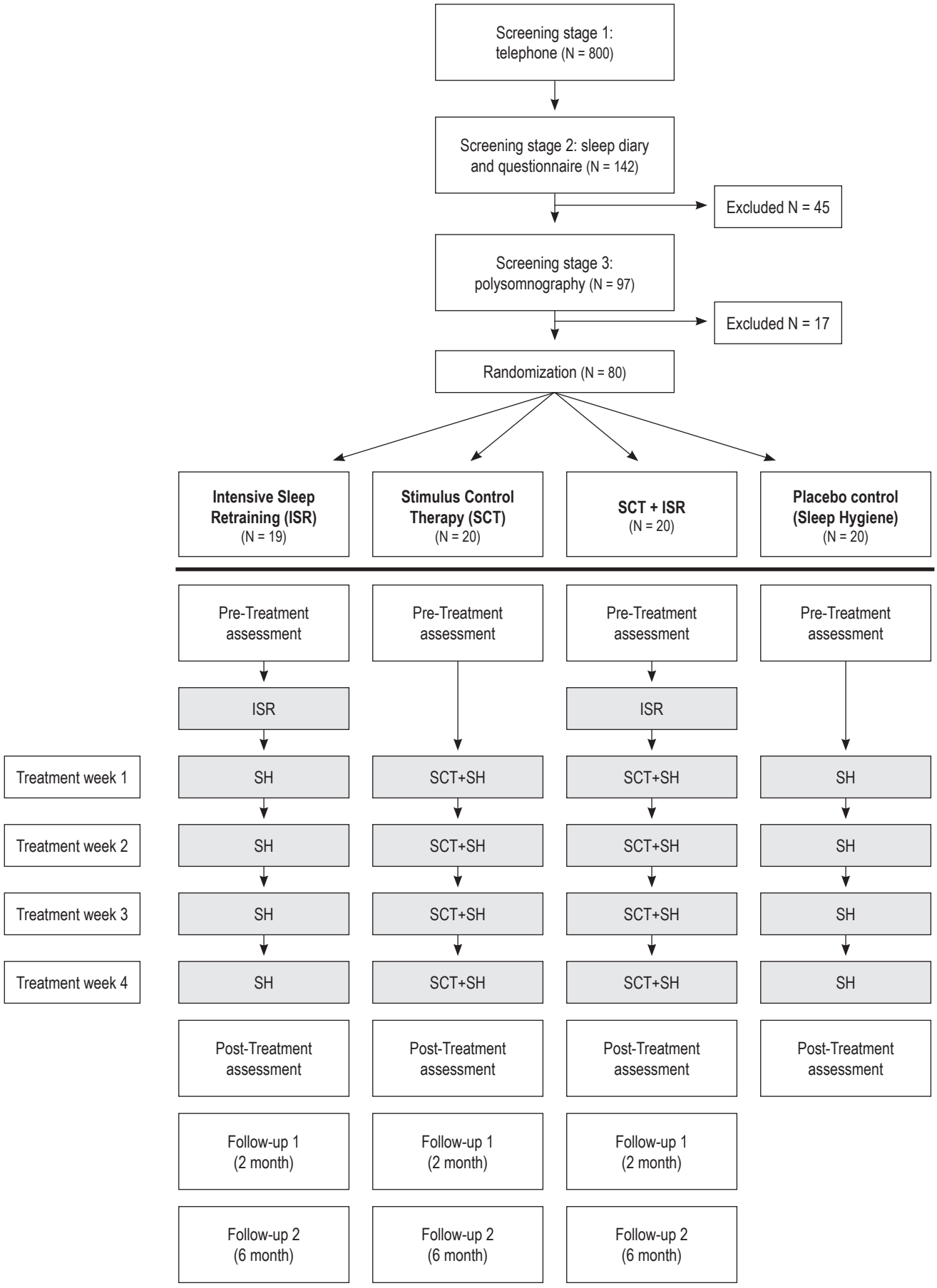

Figure 1—Research design and participant flow.

Questionnaires

Subjects completed sleep, cognition, and daytime functioning questionnaires at the completion of each 2-week assessment period. Measures included the Pittsburgh Sleep Quality Index (PSQI),[11] a 19-item questionnaire assessing sleep quality. The overall "global" score ranges from 0 to 21, with higher scores being indicative of poorer sleep quality. A score > 5 has been used to identify significant sleep disturbance in insomnia studies.[12] The sleep Self-Efficacy Scale (SES), a 9-item scale, was used to assess perceived self-efficacy for sleep-related behaviors.[13] Responses were scored on a 5-point scale from 1 ("Not confident at all") to 5 ("Very confident"). The Dysfunctional Beliefs and Attitudes about Sleep (DBAS) scale, a 30-item questionnaire, was completed to assess cognitions about sleep, including sleep loss, sleep need, and the causes and sequelae of insomnia.[14] In addition, the Daytime Feeling and Functioning Scale (DFFS) was completed, a 12-item scale that assesses the frequency of specific daytime deficits commonly reported in insomnia (e.g. "felt lethargic," "been unable to concentrate"). Each item is scored on a 0-3 scale, where 0 indicates "never or seldom" and 3 indicates "frequently or almost all the time," resulting in a total score between 0 and 36. The DFFS shows good internal consistency in insomnia samples ($\alpha = 0.89$-0.94), reliably discriminates between good and poor sleepers, and reflects treatment-related improvements in sleep variables in insomniacs.[15]

Sleep Onset Determination

In-laboratory polysomnography (PSG) was utilized for participants undergoing the ISR procedure. These were conducted with a Compumedics P-Series (Melbourne, Australia), configured to record 2 EEG channels (C3-O1, C4-A1), 2 EOG channels, EMG, and light level in order to calculate lights out time (LOT) and SOL for each sleep trial. EEG1 utilized a standard bipolar recording method, with C3 and O1 sites referenced to an A2 reference site. A referential recording was utilized between C4 and A1 sites.

PSG was utilized to determine sleep onset, in accordance with Rechtschaffen and Kales[16] sleep scoring criteria. The online display was also the method by which wakefulness between sleep trials was monitored and recorded.

Procedure

Pretreatment

Following screening, participants were scheduled to attend an orientation and education visit to the sleep laboratory approximately 2 weeks prior to their scheduled treatment start date. At this session, participants were provided with the paperwork and equipment required to complete their pre-treatment assessments, and an appointment schedule for all appointments throughout the study program.

Treatments

Intensive Sleep Retraining (ISR): On the night prior to attending the laboratory, participants undergoing ISR treatment were required to restrict their bed period to a total of 5 h, with the aid of an alarm clock, in an attempt to increase homeostatic sleep drive for the treatment period. Treatment then started with an arrival to the sleep laboratory on night 1 at 21:00. Following an explanation, the signing of an informed consent form, electrode application, and a quiet settling period, treatment began at 22:30. Treatment trials were conducted every half hour, finishing after 23:00 on night 2. Thereby, the ISR treatment routine allowed a series of 50 half-hourly sleep onset opportunities. Prior to the beginning of each trial, participants completed the Stanford Sleepiness Scale (SSS)[17] in order to gauge subjective sleepiness throughout treatment. Within each treatment trial, the opportunity for sleep onset was limited to a 20-min period, with the trial stopping if sleep onset had not occurred by this time. For those trials in which sleep was initiated, 3 consecutive minutes of sleep were permitted, prior to being awoken. Upon awakening, treatment participants first rated their perception of whether sleep onset had occurred (on a Likert scale of 1 "No, definitely not" to 7 "Yes, definitely"). Following this response, participants were provided with information as to whether sleep onset had or had not occurred.

Participants then arose from bed following each treatment trial, to maintain quiet alertness in a bedside chair, undertaking activities such as reading or watching DVD movies. At the culmination of the treatment session, participants were provided with detailed treatment feedback regarding the number of treatment trials, their sleep status for each trial, perceived sleep attainment, sleep onset latencies, and total sleep time for each trial. Participants then traveled home via a taxi, in order to have a recovery night's sleep (with a maximum of 8 h in bed). Following this weekend component of treatment, participants were booked to attend a series of 5 further treatment sessions, involving sleep hygiene alone or in combination with SCT. In order to create a degree of experimental treatment blindness, the SCT/sleep hygiene therapist was blind to the presence or absence of the ISR treatment component.

Stimulus Control Therapy (SCT): Stimulus control therapy was implemented and monitored over a series of 5 weekly appointments with a clinical psychologist experienced in the treatment of sleep disorders. Although improvements have been demonstrated in as little as one week in some patients,[18,19] an average of 5 weeks for psychological therapies have been reported in meta-analyses.[6] SCT was administered concurrently with sleep hygiene treatment.

Treatment sessions lasted for approximately 30 min each. Session one was devoted to providing treatment instructions and explaining the rationale behind SCT. Written therapy instructions were provided at this session. Sessions 2 to 4 were devoted to treatment support and encouragement, a review of the implementation of treatment instructions, and troubleshooting barriers to effective implementation of treatment. Session 5 was devoted to relapse prevention, with recommendations to reapply SCT instructions as required following treatment.

ISR plus SCT: The combination ISR and SCT group received treatment in the same manner that each were provided separately. The SCT (plus sleep hygiene) component of treatment began on the day following ISR treatment, after the recovery sleep period.

Control (Sleep Hygiene Only): The minimal treatment control utilized in the current study was sleep hygiene (SH) treatment only. Given that there is not sufficient evidence to suggest that SH is effective as a stand-alone treatment,[20] pre-

vious investigators have also used SH treatment as a control condition.[5,21-24] The SH control group was chosen over the alternative of a wait-list control due to its ability to control for variance in outcomes explained by the measurement process (i.e., monitoring via sleep diary) and nonspecific factors such as therapist attention.[25]

Sleep hygiene instructions involved education regarding healthy sleep behaviors and sleep-conducive environmental conditions, and are applied under the assumption that poor sleep, at least in part, results from a violation of these rules.[24] The SH treatment in the current study consisted of instructions addressing caffeine, nicotine, alcohol, eating behaviors, exercise, pre-bed "wind down" periods, and addressing environmental considerations of noise, light and temperature in the bedroom. These basic instructions were similar to those recommended by Morin[26] for treatment, in addition to incorporating some relaxing, resting time prior to retiring to bed. Sleep hygiene treatment was administered and monitored over a series of 5 weekly appointments of approximately 30 min in length. For those receiving SH treatment in the control condition, participation continued until the completion of the Post-treatment assessment, after which time all participants were offered further treatment.

Treatment Response Determination

Each assessment period involved 2 consecutive weeks of sleep diary and activity monitoring, with questionnaires completed at the completion of each assessment period. Assessment periods were scheduled for 2 weeks immediately prior to treatment start (Pre-treatment), immediately following treatment (Post-treatment), 6 weeks after treatment completion (Follow-up 1), and at a 6-month follow-up (Follow-up 2). Treatment response was assessed via change on both measures of sleep (sleep diary and actigraphy) for the variables of SOL, TST, WASO, and SE. Questionnaires scores also provided an important index of various psychological and subjective daytime functioning aspects of treatment response.

Treatment Fidelity

The SCT and sleep hygiene treatment components were provided by experienced clinical psychologists. The therapists were provided with written instructions as to how to explain the therapeutic rationale and each treatment recommendation. Furthermore, each client was provided with written treatment instructions to ensure they were aware of all treatment components.

Therapy sessions were audio recorded by the psychologist using a small portable digital recording device. Of these files, a randomly selected subset of 6 recordings of each of the 5 treatment sessions was selected for scrutiny as a check on treatment fidelity. Reviews suggested that all treatment items were addressed in treatment implementation and review for each treatment.

Furthermore, this process allowed a check on treatment sessions length. The mean session lengths were as follows: session 1, 35:05 minutes (SD 13:49); session 2, 21:40 minutes (SD 10:49); session 3, 20:13 minutes (SD 8:01); session 4, 19:48 minutes (SD 10:42); session 5, 17:20 minutes (SD 8:12). One-way ANOVAs indicated that there were no significant differences between all treatment groups on overall mean session length (at $P > 0.05$).

Participant Attrition

Of the 79 participants randomized to a treatment condition, 71 (89.9%) were classified as treatment completers, having attended all 5 treatment sessions. The proportion of treatment completers for each treatment group were as follows: ISR (84%), SCT (84%), ISR+SCT (100%), and control (90%). Following treatment, the proportions of participants who completed the Follow-up 1 were as follows: ISR (84%), SCT (75%), ISR+SCT (95%). The reasons for dropout included illness, unwillingness due to time commitments, an inability/unwillingness to follow treatment recommendations, and medication changes.

RESULTS

Statistical Overview

All analyses were conducted using SPSS version 14.0 (2005). The degree of change in outcome variables in response to treatment was assessed for each treatment condition separately via the use of one-way repeated measures ANOVAs and effect sizes. The effect size calculation utilized was a Cohen's d index (d = [M1-M2]/SDpooled) where M1 = Pre-treatment mean, M2 = Post-treatment mean, and SDpooled = pooled standard deviation, obtained by pooling the Pre-treatment SD across groups.

Between-group comparisons on outcome variables were undertaken between the 3 active treatment groups and the control group via the use of maximum likelihood linear mixed model analyses, hereafter referred to as linear mixed model analyses (these analyses are also known by the terms mixed effects models or random effects models). Linear mixed models are considered to be flexible and robust with respect to handling missing data in repeated measures research, and are considered superior to traditional ANOVA analyses for repeated measures designs.[27-29]

For each dependent variable, 2 linear mixed model analyses were undertaken, a preferred statistical approach for longitudinal clinical trials.[30] Firstly, all 4 treatment groups, including the SH control, were compared throughout treatment (sleep variables only) and the immediate Post-treatment assessment, using their baseline mean values as a covariate control. The 3 active treatment groups were then compared throughout Post-treatment and Follow-up periods, again controlling for baseline differences. Planned comparisons within these analyses were conducted between treatment groups at both Week 1 and at Post-treatment, in order to make a comparison of both immediate treatment response and final treatment outcome. Power calculations indicated that with the current sample size of approximately 20 per group, large effect sizes of $d = 0.80$ with a probability of above 0.80 ($P < 0.05$) might be detected. Smaller differences between active treatment groups were unlikely to be detected in the current study. Therefore, analyses were primarily aimed to detect differences between each of the active treatment groups with the control.

Intensive Sleep Retraining Treatment Application

In-laboratory ISR was successfully applied, with data demonstrating an increased and sustained objective sleepiness throughout the procedure, as indicated by repetitive rapid sleep attainment throughout the procedure. Figure 2 plots the mean subjective

(Stanford Sleepiness Scale) and objective (SOL) values throughout the 25-h procedure. After the first 2-3 h, the majority of participants obtained short sleep onset latencies (< 5 min).

Sleep Variables

Pre- to post-treatment sleep variables

The average sleep parameters of SOL, WASO, TST, and SE were calculated from the sleep diaries and activity monitor data. The averages are from 2-week periods for each assessment period, and a 1-week average for treatment weeks. Table 2 presents mean diary and actigraphy sleep values at Pre-treatment and Post-treatment (from ANOVA analyses), and the associated effect sizes (*d*). For the sleep diary measures all active treatments resulted in a significant reduction in SOL and increased sleep efficiency, compared to non-significant changes in the control group. Treatment groups undergoing ISR (ISR, ISR+SCT) resulted in increased TST time from Pre-treatment, whereas those treated with SCT (SCT, ISR +SCT) and the control group all showed significant reductions in WASO. The combined ISR+SCT group showed trends towards greater response as indicated by effect sizes. Actigraphy showed some minor trends towards improved sleep (i.e., greater SE and reduced SOL in active treatment groups), however, failed to indicate any statistically significant treatment-related changes in sleep variables other than a decreased TST in the SCT and control groups.

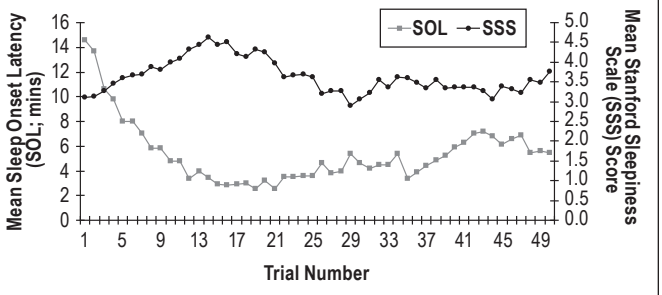

Figure 2—Mean sleep onset latency (SOL) and Stanford Sleepiness Scale (SSS) score by trial number during the intensive sleep retraining (ISR) treatment procedure.

Sleep onset latency

Figure 3 illustrates changes in sleep diary SOL throughout and following treatment in the various treatment groups. Using linear mixed model analyses and controlling for the log transformed baseline SOL, between-group comparisons were carried out on SOL values (with a log transformed sleep diary SOL variable) during treatment (Weeks 1-4) and Post-treatment. There was a main effect for treatment group ($F_{3,71.4} = 7.33$, $P < 0.001$), with a marginally significant interaction ($F_{12,68.6} = 1.82$, $P = 0.06$) and main effect for time ($F_{4,68.0} = 2.42$, $P = 0.06$). Paired comparisons between groups indicated ISR+SCT reported a decreased SOL during the treatment weeks and Post-treatment period in comparison to both the control group ($P \leq 0.001$), and the SCT group ($P < 0.05$). ISR alone also demonstrated a consistently shorter SOL than the control group across this period ($P < 0.01$). The SCT group did not differ significantly from the control group.

Table 2—Mean (SD) sleep diary, actigraphy and questionnaire values from Pre-treatment (Pre-T) to Post-treatment (Post-T), and change effect sizes (*d*)

	ISR			SCT			ISR+SCT			CONTROL		
Variable	**Pre-T**	**Post-T**	***d***	**Pre-T**	**Post-T**	***d***	**Pre-T**	**Post-T**	***d***	**Pre-T**	**Post-T**	***d***
Sleep Diary												
SOL	61.41 (25.21)	38.41** (16.24)	0.61	68.33 (44.04)	38.94*** (29.39)	0.78	60.79 (42.79)	24.70*** (12.83)	0.96	71.87 (37.23)	68.65 (37.72)	0.09
TST	368.59 (62.71)	403.23** (55.37)	0.53	370.65 (54.13)	387.95 (57.36)	0.26	357.41 (65.61)	411.91*** (50.91)	0.83	348.76 (78.83)	350.26 (76.76)	0.02
WASO	75.57 (56.00)	60.71 (59.01)	0.26	72.34 (44.47)	42.66** (37.53)	0.52	87.24 (53.30)	42.42*** (25.32)	0.79	99.25 (70.48)	80.31* (57.38)	0.33
SE	70.02 (11.08)	79.85*** (8.84)	0.65	70.64 (9.40)	81.57*** (7.34)	0.91	68.08 (11.89)	84.20*** (5.68)	1.34	65.96 (14.89)	68.24 (14.14)	0.24
Actigraphy												
SOL	38.53 (17.18)	28.05 (15.29)	0.35	34.17 (23.73)	28.36 (15.60)	0.19	40.53 (26.46)	31.89 (32.04)	0.29	47.54 (45.70)	43.01 (42.64)	0.15
TST	386.61 (69.92)	392.38 (62.54)	0.09	375.86 (55.32)	365.12* (65.98)	0.17	388.84 (58.22)	378.24 (71.41)	0.17	379.40 (73.84)	368.50* (72.74)	0.17
WASO	90.04 (66.90)	90.31 (69.04)	0.01	93.85 (49.46)	92.47 (44.00)	0.03	85.37 (50.48)	84.20 (50.90)	0.02	99.47 (43.11)	100.19 (46.49)	0.01
SE	75.32 (12.27)	76.57 (11.91)	0.11	74.33 (10.96)	75.61 (9.82)	0.11	75.55 (10.03)	77.59 (11.63)	0.18	72.26 (12.58)	71.92 (11.91)	0.03

SOL, sleep onset latency; TST, total sleep time; WASO, total wake time overnight after sleep onset; SE, sleep efficiency (total sleep time/time in bed × 100). Data is derived from participants within each condition with available data at both assessment points. ANOVA (P) statistics for Pre-Treatment to Post-Treatment change, *** < 0.001, ** < 0.01, * < 0.05.

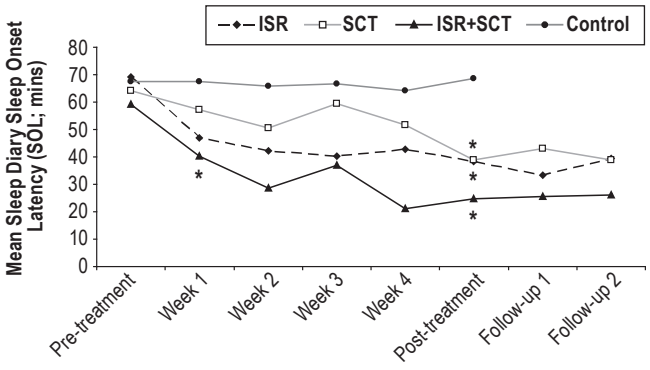

Figure 3—Mean sleep diary sleep onset latencies (SOL) prior to, during, and following treatment. * Mean values are significantly different (P < 0.05) from control.

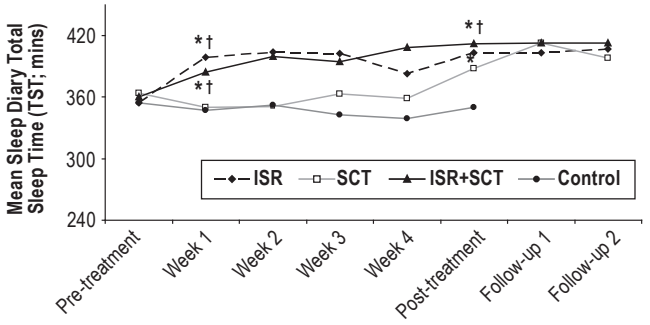

Figure 4—Mean sleep diary total sleep time (TST) prior to, during, and following treatment. *Mean values are significantly different (P < 0.05) from control, †Mean values are significantly different (P < 0.05) from SCT.

Planned paired comparisons were performed between the control group and active treatment groups in Week 1 and at Post-treatment. In Week 1, only the ISR+SCT group reported a mean SOL that was significantly shorter than the control group (P < 0.01). No significant differences between any of the active treatment groups were apparent at this stage. By Post-treatment, all active treatment groups reported a significantly reduced SOL in comparison to the control group (ISR: P < 0.05; SCT: P < 0.01; ISR+SCT: P < 0.001). Again, there were no significant differences between active treatment groups.

Further analyses were undertaken on the active treatment groups during the Post-Treatment and Follow-up periods. Again, linear mixed model analyses (controlling for Pre-treatment mean SOL values) were performed between the treatment groups during the Post-treatment, Follow-up 1, and Follow-up 2 assessments only. There was a main effect for treatment group ($F_{2,45.3}$ = 3.26, P < 0.05), with ISR+SCT outperforming both SCT (P < 0.05) and ISR alone (P < 0.05). There was no main effect for time ($F_{2,47.1}$ = 1.59, P = 0.22) or interaction effect ($F_{4,47.1}$ = 0.44, P = 0.78), indicating that following the treatment period, all active treatments maintained their response.

Actigraphy measures failed to indicate a significant main effect for treatment group (controlling for baseline differences) for mean SOL either during treatment ($F_{3,68.1}$ = 2.56, P = 0.06) or following treatment ($F_{2,55.9}$ = 2.25, P = 0.09).

Total sleep time

During treatment and Post-treatment, linear mixed model analyses of TST (controlling for baseline values) indicated a main effect for group ($F_{3,73.3}$ = 11.59, P < 0.001), a main effect for time ($F_{4,68.3}$ = 3.47, P < 0.05), and a significant interaction effect ($F_{12,74.8}$ = 2.01, P < 0.05). Between-group significant effects were found between the control group and both ISR+SCT treatment group (P < 0.001) and ISR treatment group (P < 0.001). In addition, both ISR+SCT (P < 0.001) and ISR (P < 0.001) outperformed SCT treatment, producing significantly increased TST throughout treatment, illustrated in Figure 4.

When comparing treatment groups to the control group at Week 1 in planned comparisons, the ISR treatment groups showed rapidly increased TST, with significantly increased TST relative to the control group in the ISR group (P < 0.001) and the ISR+SCT group (P < 0.01) in Week 1. In addition, the ISR and ISR+SCT treatment groups demonstrated a significantly greater TST than those in the SCT group at Week 1 (ISR: P < 0.001; ISR+SCT: P < 0.01). By Post-treatment, participants receiving ISR and ISR+SCT maintained a superior response, with a greater mean TST than the control group (ISR: P < 0.01; ISR+SCT: P < 0.001). Comparisons between active treatment groups at Post-treatment found the only significant difference to be a greater mean TST in the ISR+SCT group than in the SCT treatment group (P < 0.05).

Analyses following treatment were conducted between active treatment groups, indicating no main effect for treatment group ($F_{2,47.5}$ = 1.55, P = 0.224), time ($F_{2,45.1}$ = 0.43, P = 0.66), or interaction effect ($F_{4,45.1}$ = 1.74, P = 0.16). This suggests that following treatment, all groups were comparable on the TST variable.

A main effect for treatment group on actigraph TST ($F_{3,70.4}$ = 3.02, P < 0.05) indicated a greater TST in the ISR group during treatment than the SCT group (P < 0.01). Following treatment, there were no significant between group effects for actigraphically measured TST ($F_{2,47.7}$ = 0.34, P = 0.72).

Wake time after sleep onset

During treatment and Post-treatment, linear mixed model analyses resulted in a main effect for treatment group ($F_{3,72.7}$ = 6.5, P ≤ 0.001) for the WASO variable, illustrated by Figure 5. There were, however, no significant main effects for time ($F_{4,70.5}$ = 1.33, P = 0.267) or interaction effects ($F_{12,76.7}$ = 0.87, P = 0.58). Both the SCT and ISR+SCT treatment groups reported significantly lower mean WASO values than the control group (P ≤ 0.001). In addition, the ISR+SCT group reported a decreased mean WASO compared with ISR group (P < 0.05). Overall, ISR treatment alone was not significantly different from SCT or control groups on the WASO variable.

Planned comparisons indicated that improvements in WASO were rapid in those treatment groups receiving SCT. In Week 1, both ISR+SCT (P < 0.05) and SCT (P < 0.05) showed decreased mean WASO values in comparison to the control group, with no other significant differences between treatment groups. By Post-treatment, ISR+SCT (P < 0.01) and SCT (P < 0.05) groups maintained their gains relative to control. Between active treatment groups, only the ISR+SCT group showed a significantly reduced WASO in comparison to the ISR group (P < 0.05) at Post-treatment.

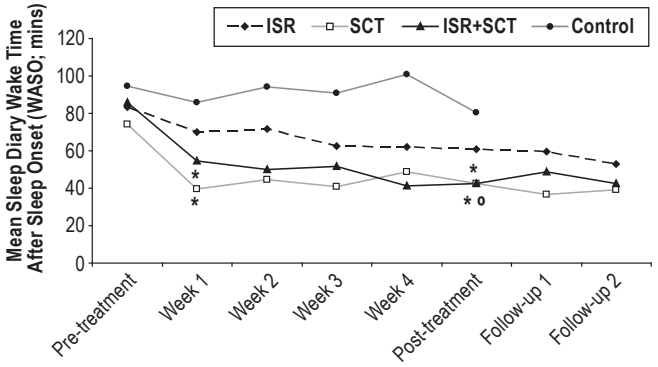

Figure 5—Mean sleep diary wake time after sleep onset (WASO) prior to, during, and following treatment. *Mean values are significantly different (P < 0.05) from control, °Mean values are significantly different (P < 0.05) from ISR.

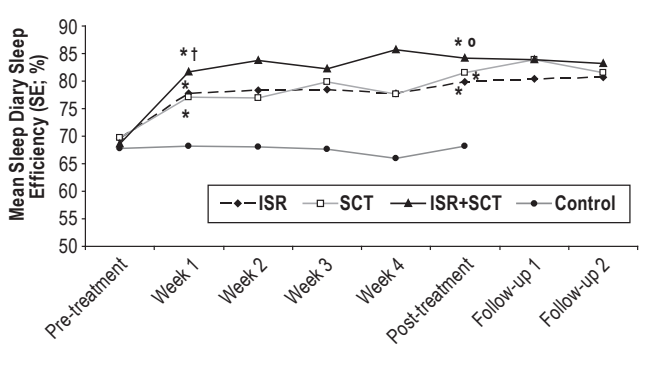

Figure 6—Mean sleep diary sleep efficiency (SE) prior to, during and following treatment. *Mean values are significantly different (P < 0.01) from control. °Mean values are significantly different (P < 0.05) from ISR. †Mean values are significantly different (P < 0.05) from SCT.

Following treatment, there were no main effects for time ($F_{2,45.4} = 0.36$, P = 0.70), treatment group ($F_{2,49.8} = 1.93$, P = 0.16), or interaction effects ($F_{4,45.5} = 0.80$, P = 0.54) on the sleep diary WASO variable. These data indicate no differential response on WASO to treatment or change in response over time following treatment, based on Post-treatment and follow-up assessments.

There were no significant between-group effects for objective WASO as measured by actigraphy either during treatment ($F_{3,71.9} = 1.75$, P = 0.16) or following treatment ($F_{2,50.6} = 0.23$, P = 0.80).

Sleep efficiency

Figure 6 illustrates changes in subjective SE across and following treatment in the various treatment conditions. Across treatment to Post-treatment, linear mixed model analyses indicated a significant main effect for treatment group ($F_{3,72.9} = 18.15$, P < 0.001), with ISR (P < 0.001), SCT (P < 0.001), and ISR+SCT (P < 0.001) outperforming the control group on the SE variable. In addition, the combination ISR+SCT treatment group produced higher overall sleep efficiencies than SCT (P ≤ 0.01) and ISR (P < 0.01). ISR and SCT were not significantly different. There were no significant main effects for time ($F_{4,68.5} = 2.18$, P = 0.08) or interaction effects ($F_{12,68.6} = 0.96$, P = 0.49) up to Post-treatment.

Planned comparisons between treatment groups at the beginning and end of treatment indicated that all active treatment groups showed increased SE in comparison to the control group at both Week 1 and Post-treatment (P < 0.01). ISR+SCT, however, outperformed SCT in Week 1 (P < 0.05), with no other differences between active treatments at this time. By Post-treatment, ISR+SCT was superior to ISR treatment alone (P < 0.05). This data indicates that all treatments were effective in rapidly increasing SE and maintaining those changes throughout treatment, although ISR+SCT outperformed other active treatments.

Following treatment, after controlling for baseline differences, there were no main effects for treatment group ($F_{2,49.2} = 2.69$, P = 0.08), time ($F_{2,44.2} = 0.54$, P = 0.59), or interaction effects ($F_{4,44.2} = 0.96$, P = 0.44). Nevertheless, as was the case during treatment, the ISR+SCT treatment group maintained a significantly higher mean SE than the ISR treatment group following treatment (P < 0.05). These data suggest no significant changes within groups over time following treatment.

Objective actigraph-measured sleep efficiency failed to indicate any main effects for treatment group either during treatment ($F_{3,69.8} = 2.45$, P = 0.07) or following treatment ($F_{2,48.7} = 0.10$, P = 0.91).

Questionnaire Variables

Pittsburgh Sleep Quality Index

Table 3 outlines mean changes (and effect sizes) on questionnaire variables over time. Within groups, using ANOVA analyses, there was a significant change from Pre-treatment to Post-treatment on mean global Pittsburgh Sleep Quality Index scores in all active treatment groups (P < 0.001), with no significant change in the control group. Comparisons between all groups at Post-treatment indicated a main effect for treatment group ($F_{3,63} = 10.66$, P < 0.001) on global PSQI scores, when controlling for baseline values. The combination ISR+SCT treatment group reported lower overall PSQI scores than ISR (P < 0.001), SCT (P < 0.01) and control (P < 0.001) groups. At Post-treatment, SCT also had significantly lower mean PSQI scores than the sleep hygiene control group (P < 0.05).

Linear mixed model analyses between active treatment groups, following treatment, indicated a significant main effect for treatment group ($F_{2,47.91} = 6.66$, P < 0.01) and time ($F_{2,36} = 4.28$, P < 0.05) on Global Sleep Quality. Participants in the ISR+SCT treatment group reported a significantly higher sleep quality across assessments following treatment than those in the SCT only group (P < 0.05) and the ISR group (P < 0.01). Significant changes across time indicated a significantly decreased PSQI score from Post-treatment to Follow-up 1 in the active treatment groups. The interaction effect was non-significant ($F_{4,36} = 1.49$, P = 0.23).

Sleep Self Efficacy Scale

Mean scores on the SES questionnaire variable increased significantly, indicating improved self-efficacy in all active treatment groups (P < 0.001), with little change in the control group. Using linear mixed model analyses, controlling for baseline values, at Post-treatment there was a significant treatment

Table 3—Mean (SD) questionnaire values from Pre-treatment (Pre-T) to Post-treatment (Post-T), Follow-up 1 (6-weeks) (FU1), and Follow-up 2 (6-months) and change effect sizes (*d*) from Pre- to Post-treatment

Variable	Pre-T	Post-T	*d*	FU1	FU2
PSQI					
Control	12.22 (3.02)	11.11 (2.72)	0.48	–	–
ISR	11.13 (2.31)	8.88 (3.05)***	0.97	7.93 (3.65)	8.53 (5.02)
SCT	11.73 (1.75)	8.73 (2.71)***	1.29	6.81 (2.93)	8.39 (2.60)
ISR+SCT	11.47 (1.98)	6.37 (2.59)***	2.20	5.78 (3.54)	5.89 (3.27)
DBAS					
Control	81.56 (13.6)	77.56 (13.4)	0.32	–	–
ISR	77.44 (10.1)	67.63 (12.7)***	0.79	63.87 (9.86)	64.93 (11.8)
SCT	77.87 (13.8)	67.87 (12.3)***	0.81	64.19 (14.7)	60.15 (14.1)
ISR+SCT	84.37 (11.4)	68.84 (13.2)***	1.26	63.00 (15.4)	61.14 (11.8)
Daytime Impairment					
Control	13.61 (7.72)	12.56 (6.92)	0.16	–	–
ISR	12.94 (5.54)	9.13 (5.33)*	0.57	8.13 (4.50)	8.40 (5.84)
SCT	14.53 (5.71)	10.00 (7.75)**	0.68	9.56 (6.98)	7.00 (6.11)
ISR+SCT	15.74 (7.29)	7.79 (6.00)***	1.20	7.22 (6.38)	8.67 (6.08)
Sleep Self-Efficacy					
Control	20.56 (5.16)	21.22 (5.45)	0.15	–	–
ISR	21.06 (4.48)	26.13 (5.60)***	1.10	27.33 (5.67)	28.40 (6.01)
SCT	19.60 (3.14)	28.87 (6.16)***	1.58	30.88 (6.20)	29.69 (7.29)
ISR+SCT	20.37 (5.30)	28.59 (5.91)***	1.79	30.44 (7.06)	30.94 (8.16)

PSQI, Pittsburgh Sleep Quality Index; DBAS, Dysfunctional Beliefs and Attitudes about Sleep Scale; Daytime Impairment, DFFS, higher scores indicate greater impairment. ANOVA (P) statistics for Pre-Treatment to Post-Treatment change, *** ≤ 0.001, ** < 0.01, * < 0.05.

group effect ($F_{3,63}$ = 7.38, P < 0.001), with all treatment groups being significantly different from control group self-efficacy values (P < 0.01) There were no differences between active treatment groups at this time point.

Linear mixed model analyses compared active treatment groups on SES scores across time, following treatment. Results indicated that there were no significant main effects for treatment group ($F_{2,47.8}$ = 1.62, P = 0.21), and no interaction effects ($F_{4,44.4}$ = 1.50, P = 0.23) when controlling for baseline values. There was, however, a significant main effect for time ($F_{2,44.4}$ = 3.23, P < 0.05), with continued improvements on this variable from Post-treatment to Follow-up 2 (P < 0.05). These data suggest a comparable treatment response across active interventions.

Dysfunctional Beliefs and Attitudes about Sleep Scale

ANOVA analyses indicated a significant decrease on DBAS scores from Pre-treatment to Post-treatment (P < 0.001), with no significant change in the control group. Using linear mixed model analyses at Post-treatment, comparisons across all treatment groups resulted in a main effect for treatment group ($F_{3,63}$ = 3.53, P < 0.05), with ISR (P < 0.05), SCT (P < 0.05) and ISR+SCT (P < 0.01) all resulting in mean DBAS scores that were significantly lower than the control group. There were no other differences between the three active treatment groups at Post-treatment.

After treatment, linear mixed model analyses compared treatment groups across time. Analyses indicated that there were no significant main effects for treatment group ($F_{2,46.4}$ = 1.39, P = 0.26), showing a similar DBAS change in response to the different interventions. There was a significant main effect for time ($F_{2,43.5}$ = 7.28, P < 0.005), with continued reductions across treatment groups in DBAS scores from Post-treatment to Follow-up 1 (P < 0.01) and to Follow-up 2 (P < 0.001). The interaction effect was non-significant ($F_{4,43.6}$ = 1.13, P = 0.36).

Daytime feelings and functioning scale

Statistically significant improvements in various aspects of daytime feeling and functioning explored in the DFFS were observed in all treatment groups from Pre-treatment to Post-treatment (P < 0.05), but not in the control group. Comparisons between treatment groups at Post-treatment using linear mixed model analyses, controlling for baseline values, indicated a significant main effect for group ($F_{3,63}$ = 4.02, P < 0.05). The combined ISR+SCT treatment group was the only active treatment group to significantly differ from control (P < 0.001), with no other differences between groups at this time.

Linear mixed model analyses between active treatment groups following treatment demonstrated no significant main effects for treatment group ($F_{2,44.2}$ = 1.46, P = 0.24), time ($F_{2,42.9}$ = 1.56, P = 0.86), or interaction effects ($F_{4,42.9}$ = 0.48, P = 0.84). Results indicate a trend for similar response to treatment on aspects of daytime functioning for active treatment groups.

Responder Analysis

In addition to demonstrating statistically significant changes, and reporting change effect sizes, the clinical significance of treatment-related change is important in assessing treatment response. Although there are no currently agreed upon criteria for assessing clinically significant change, many studies use a percentage change in SOL, or characterize treatment responders as reaching an outcome within a normative range for sleep vari-

Table 4—Within treatment group N and % of sleep diary "treatment responders" at Follow-up 1

	Responder Criteria 1		Responder Criterion 2	
	N	%	N	%
ISR	7	46.7%	7	36.8%
SCT	6	37.5%	6	30.0%
ISR+SCT	11	61.1%	13	65.0%

Criteria 1 = (a) reduction of SOL from Pre-treatment to Follow-up 1 to an average of < 30 min, or a reduction of SOL by ≥ 50%, and (b) sleep efficiency increase to ≥ 85%. Criterion 2 = a reduction in SOL by 33% to < 35 minutes.

ables such as SOL and SE. For instance, Morin and colleagues[31] outline various indicators of clinical significance, including (a) the proportion of clients who experience a reduction of 50% on sleep variables of SOL or WASO, and reach an absolute value of near or below the 30-min criterion used to define insomnia, (b) the proportion of clients whose sleep efficiency moves into a normative range (i.e., > 80% to 85%), or (c) a reduction in hypnotic drug use.

The current study utilized a dual sleep diary criterion for the assessment clinical significance. Classification of response required (a) reduction of SOL to an average < 30 min, or a reduction of SOL by ≥ 50%, and (b) SE increase to ≥ 85%. Clinically significant treatment response was assessed at Follow-up 1 (beginning 6 weeks following treatment). Table 4 reports the proportion of clients who met these criteria for a "responder," in addition to those meeting criteria utilized in meta-analyses (reduction in SOL by 33% to < 35 min as the criterion for clinically meaningful change).[7] Given that data were not available at Follow-up 1 for the control group, these analyses pertain to active treatment groups only. Overall, this classification of response resulted in 40% to 45% of participants showing a clinically significant response. These data indicate the greatest number and percentage of "responders" in the combination ISR+SCT treatment group, with over 60% achieving a clinically significant change.

DISCUSSION

The aim of this study was to further explore the efficacy of intensive sleep retraining (ISR) treatment for chronic insomnia. This treatment approach is novel in attempting to treat insomnia in an intensive, single treatment period. ISR was compared to, and combined with, stimulus control therapy, and compared with a "sleep hygiene only" control group, with participants randomly allocated to treatment group. Outcome variables included the primary outcome of subjective sleep parameters, in addition to objective sleep variables and psychological and daytime functioning measures.

As predicted, participants in all three active treatment groups experienced significant improvements in subjective sleep variables following treatment. This was in comparison to the minimal changes observed in the sleep hygiene treatment control group. ISR seemed to produce immediate improvements in sleep, as demonstrated by decreased SOL and increased TST in the first week following the treatment weekend. Indeed, the main advantage of ISR treatment appears to be the immediacy of treatment response. Under the assumption that the ISR technique acts via a conditioning effect, the treatment response to the ISR procedure provides evidence for the explanatory value of the conditioning etiologic model, and supports the use of conditioning treatments in insomnia. In addition, it must be noted that the sleep/wake state feedback during the ISR treatment may constitute sleep perception or sleep/wake discrimination training, which has been found to improve sleep in individuals suffering from insomnia.[32,33] More systematic exploration of sleep perception improvements with ISR treatment may assist in clarifying treatment mechanisms in future studies. Furthermore, this treatment also provides exposure to the single element that is most feared by individuals with insomnia, the experience of sleep deprivation. Indeed, the sleep deprivation alone, with an acutely increased homeostatic drive, may be responsible for the generation of some improvements in sleep. Further research will be needed to explore the effective components of this ISR treatment.

Participants undergoing stimulus control therapy (SCT) also experienced significantly improved sleep from Pre- to Post-treatment. However, treatment response indicated by SOL and TST were slower to eventuate over the treatment weeks in comparison to those groups receiving ISR. It must be noted that an early response to SCT has been indicated in some research, with significant effects emerging in some sleep variables after only one week of treatment in some patients.[18,34] Although the current study did not investigate individual response, a rapid mean response was not apparent across all sleep variables following SCT. However, consistent with this previous research, the WASO and SE variables did demonstrate immediate improvements in the SCT group.

The combination ISR+SCT treatment group showed trends for outperforming single treatments, producing immediate treatment gains that were impressively maintained. The combination ISR+SCT group was associated with the strongest treatment response, resulting in SOLs close to the 30-minute cutoff criterion for "good sleeper" status, larger effect sizes, and a somewhat greater proportion of treatment responders. Indeed, 61% of participants in the ISR+SCT treatment group achieved a clinically significant treatment response according to the sleep diary criterion, achieving "good sleeper" status, in comparison to 47% of ISR only recipients and 38% of SCT recipients. In addition, this combination group produced fewer treatment dropouts than other treatment conditions. The changes up to Post-treatment in subjective sleep and daytime functioning variables for the combination ISR+SCT treatment group, as indicated by effect sizes, were consistently greater than in the other treatment groups. The results obtained in this group were consistent, and even superior in some cases, to those reported in meta-analytic studies of insomnia treatments.[6,7] For instance, the ISR+SCT treatment group showed increases in TST that were close to one hour. Reductions in mean WASO in the ISR+SCT group were comparable to the improvements obtained with SCT, reduced to around 40 minutes.

Nevertheless, there were relatively few statistically significant differences between active treatment groups, suggesting that these groups often improved to a similar extent, and at a similar rate, over time. Alternatively, it must be recognized that

significant differences between active interventions are difficult to obtain with the typical small absolute differences in response. It may be that a much larger sample size would have been required in order to establish any consistent differences in outcome between treatments. Nevertheless, the sample size is comparable to that in other treatment comparison research,[31] and the clinical significance of such small between-group differences may not be particularly meaningful. With limited power and a truncated participation of the control group (to Post-treatment) due to ethical and dropout considerations, longer-term control comparisons were unable to be made in this study.

Actigraphy and questionnaire measures also provided interesting information about treatment-related change. Questionnaire measures supported significant changes in daytime functioning, beliefs about sleep and confidence in sleep. Whether these cognitive changes occur as a function of successful behavioral intervention, or are key processes in treatment outcome may be addressed by further research in this area. The actigraphy data failed to support significant changes in sleep, despite using an adjusted manual scoring method and a sensitivity setting in the scoring algorithm that calibrated actigraphy TST to PSG TST. Actigraphy has similarly failed to mirror subjective sleep changes in other treatment studies in insomnia,[35] and objective measures (i.e., EEG) fail to replicate the extent of subjective sleep changes in clinical insomnia treatment studies.[36] The current findings are consistent with conclusions regarding the limited usefulness of actigraphy in assessing sleep in insomnia.[37] In addition, although the data were not presented, the actigraphic data derived from the automatic (with sensitivity setting) scoring method similarly failed to indicate a treatment response over time. Unfortunately, no follow-up PSG comparisons were made in this sample. This may have provided a validation of, and a corresponding preference for, either method of actigraphy scoring. Similarly, this kind of follow-up PSG assessment may have provided information regarding whether objective changes in sleep were apparent following treatment. It remains possible that the lack of response to treatment indicated by actigraphy validly indicates a lack of objective change in sleep. This would suggest that the changes in sleep diary data reflect the subjective nature of both the complaint and change in sleep variables.

This RCT represents the first comprehensive study into the efficacy and comparative effectiveness of intensive sleep retraining and, as such, represents a novel contribution to the insomnia treatment literature. Although the creation and assessment of novel treatment ideas are important for the advancement of the field, ISR fails to fulfill all requirements of an ideal intervention. Despite significant treatment benefits, ISR alone did not normalize sleep parameters, on average. With a more rapid and larger magnitude of response when combined with SCT, this combined treatment approach may provide two advantages, a rapid treatment response (i.e., comparative to the rapidity of drug therapy) and the maintenance and enhancement of treatment gains. Indeed, a synergistic effect may be possible, with an initial rapid response to ISR improving consequent compliance to and therefore response to SCT. Further studies may systematically measure compliance to treatment to explore this possibility. However, if ISR is to provide an appealing supplemental treatment or treatment alternative, it will need to prove to be both clinically effective and cost-effective. The advantage, over and above traditional therapies, must be demonstrated in order to justify its use.

These advantages may include the rapid treatment response, treatment effectiveness in particular populations, or in addressing particular components of the sleep disorder, factors open to further investigation. The superior treatment response for sleep onset problems (with less impact on WASO) may indicate suitable ISR treatment recipients. With the assumption of conditioning-based treatment mechanisms, responses to ISR may indicate that there is some difference between the conditioned insomnia episodes that occur prior to sleep onset from those occurring during the night. Further research may further determine the sleep profile of those individuals with insomnia that best respond to this procedure.

Given that ISR is an intensive, monitored, in-laboratory procedure lasting 25 hours, the administration of this treatment is very costly. In order for this to become a treatment that is able to be easily disseminated to the sleep disordered community, alternative administration of the treatment routine may need to be devised. One such possibility is a self-administered version of ISR at home. Home administration of ISR treatment then need not require a transfer of the conditioning training to the home environment, perhaps resulting in a superior treatment response. Therefore, an ISR treatment version administered in the home environment of individuals suffering from insomnia is an important avenue for future research.

The findings of the current study suggest that a brief, intensive, conditioning treatment procedure may be a useful treatment possibility for individuals suffering from primary insomnia, particularly when combined with traditional behavioral treatments. ISR rivals SCT, presently the most supported single behavioral intervention. Further investigations into determining appropriate treatment recipients, and alternative forms of implementation of such a brief conditioning treatment are warranted.

ABBREVIATIONS

ISR, intensive sleep retraining
SCT, stimulus control therapy
OSA, obstructive sleep apnea
DSPD, delayed sleep phase disorder
PLMS, periodic limb movements of sleep
BDI-II, Beck Depression Inventory-II
SOL, sleep onset latency
BMI, body mass index
RDI, respiratory disturbance index
WASO, wake time after sleep onset
SE, sleep efficiency
TST, total sleep time
PSQI, Pittsburgh Sleep Quality Index
SES, Sleep Self Efficacy Scale
DBAS, Dysfunctional Beliefs and Attitudes about Sleep
DFFS, Daytime Feeling and Functioning Scale
PSG, polysomnography
EEG, electroencephalography
EOG, electrooculography
EMG, electromyography
LOT, lights out time
SSS, Stanford Sleepiness Scale

SH, sleep hygiene
ANOVA, analysis of variance
SD, standard deviation
RCT, randomized controlled trial

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