

TREATING ACUTE INSOMNIA WITH A SINGLE SESSION OF CBT-I

Treating Acute Insomnia: A Randomized Controlled Trial of a “Single-Shot” of Cognitive Behavioral Therapy for Insomnia

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Study Objectives: Despite considerable evidence supporting cognitive behavioral therapy for insomnia (CBT-I) for chronic insomnia, it remains untested within the context of acute insomnia. This study examined the efficacy of a single session of CBT-I, with an accompanying self-help pamphlet, for individuals with acute insomnia.

Design: A pragmatic parallel group randomized controlled trial.

Setting: Community.

Participants: Forty adults (mean age 32.9 ± 13.72 y) with Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) defined insomnia disorder, except a self-reported duration of less than 3 mo (i.e., acute insomnia), who reported no previous exposure to CBT-I and were not currently taking medication for sleep.

Interventions: A single 60- to 70-min session of CBT-I (n = 20), with an accompanying self-help pamphlet, or wait list control group (n = 20). All subjects were offered a full individual course of CBT-I on completion of the study, regardless of group allocation.

Measurements and Results: Subjects completed sleep diaries and the Insomnia Severity Index (ISI) pretreatment and 1 mo following treatment. There were no between-group differences on baseline ISI scores or subjective sleep continuity. The intervention group reported significantly lower ISI scores than controls (t(38) 2.24, P < 0.05) at follow-up. Further, using proposed ISI scores for identifying insomnia caseness (i.e., ≥ 10), 60% of those in the CBT-I group had remitted by 1 mo compared to 15% of those in the control group.

Conclusions: This single session of cognitive behavioral therapy for insomnia (CBT-I) is sufficiently efficacious for a significant proportion of those with acute insomnia. The results are discussed in terms of integrating this brief form of CBT-I into the “stepped care” model of insomnia.

Trial Registration: Testing the efficacy of an early intervention for acute insomnia (SRCTN05891695) <http://www.controlled-trials.com/ISRCTN05891695>.

Keywords: acute insomnia, brief intervention, CBT-I, short-term insomnia, stepped care, treatment

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INTRODUCTION

Chronic insomnia is a significant public health concern and has been linked to the development and/or worsening of a number of physical and psychiatric conditions.^{1–6} At symptom level, between 30–48% of the population report having chronic insomnia and at syndrome level the prevalence, albeit significantly lower at between 6–15%, still represents a major challenge at both individual and societal levels.^{7,8} Importantly, chronic insomnia is also a persistent disorder with one study demonstrating the majority (74%) of individuals reporting insomnia at baseline still report having insomnia 1 y later and almost half (46%) report having insomnia at three consecutive annual assessment points.⁹ In essence, chronic insomnia is a highly prevalent, costly (the direct costs of chronic insomnia alone are estimated to be in the region of \$13.9 billion annually),¹⁰ and largely unrelenting condition.

Standard Cognitive Behavioral Therapy for Insomnia (CBT-I), a 6- to 8-w intervention targeting sleep-incompatible thoughts, beliefs, and behaviors, has been consistently

demonstrated as efficacious, effective, and durable.^{11,12} In fact CBT-I is considered the first-line treatment for both “pure” chronic insomnia and chronic insomnia comorbid with other diseases, disorders, or disabilities.^{13–15} Despite the overwhelming evidence for CBT-I, two main issues hamper its widespread uptake—a lack of qualified providers and high levels of non-adherence and attrition.^{16,17} To improve access and treatment completion, there has been a recent upsurge in studies exploring alternative treatment delivery modalities such as group therapy, telehealth, and computerized CBT-I (cCBT-I).^{18–21} Despite the introduction and widespread dissemination of these innovative modalities, early indications suggest there may be a tradeoff in terms of reduced efficacy compared to individual face-to-face contact.^{22,23} Furthermore, where attempts have been made to identify specific issues that can affect tolerability and ultimately early termination of CBT-I, such as patient expectations, patient depression levels, levels of therapeutic alliance, and partner involvement,^{24–26} these findings have yet to be integrated into standard care.

One alternative perspective, which has the potential to address issues of access and tolerability, is to look more closely at the intervention itself. With that in mind there have been several trials of hybrid versions of CBT-I, such as Abbreviated Cognitive Behavioral Insomnia Therapy (ACBT),²⁷ Brief Behavioral Treatment for Insomnia (BBTI),²⁸ and one study that aimed to identify the optimal dose of CBT-I.²⁹ In terms of outcome, both ACBT (two 25-min sessions, with an accompanying pamphlet) and BBTI (a single 45-min session with a

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30-min “booster” session 2 w later) were sufficient to confer benefits for those with chronic insomnia.^{27,28} As for the optimal dose, although Edinger and colleagues²⁹ found a four-session dose of CBT-I was optimal (58.3% patients demonstrated clinically relevant improvements), they also showed that a single 45- to 60-min session was superior to a two-session dose and a full eight-session dose (43.8% versus 22.2% and 35.3%, respectively). As such, it appears that a modified CBT-I/BBTI that involves contact somewhere between 45 and 75 min (in one or four sessions) has the potential to affect change in people with chronic insomnia.

When examining the literature, one further consideration, which may afford a new and novel approach to treatment, should be borne in mind—all the aforementioned studies have conceptualized insomnia, albeit based on differing nosologies, as a chronic condition. This is timely considering the latest edition of the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5) states, for the first time, that if full criteria for insomnia disorder are met, bar the minimum duration criteria of 3 mo, the condition (i.e., acute insomnia) may still warrant attention.³⁰ There are numerous advantages to treating insomnia during an acute phase. If successful, there is potential for significant savings in terms of long-term health care utilization, lost productivity, and accidents. This becomes more pertinent when the costs associated with other illnesses, for which insomnia is known to be a risk factor (e.g., depression), are also taken into account. More importantly, acute insomnia is likely to be easier to treat and as such less burdensome for both therapist and patient compared to chronic insomnia. The rationale for this is twofold. Not only should conditioned arousal (to the bedroom and bedroom routine), a significant feature of chronic insomnia,^{31,32} be in its infancy and thus easier to correct but the self-schemata as “an insomniac,” presumably based on repeated patterns of sleep related dysfunctional thinking (e.g., prolonged worry, rumination, catastrophizing), should either not be in evidence or fully realized at this stage of the disorder. Support for this last statement comes, albeit anecdotally, from earlier recruitment attempts, across several studies^{33–35} whereby individuals will self-identify as having “a sleep problem” during the first few weeks/months of insomnia but not as having “acute insomnia.”

What Would an Intervention for Acute Insomnia Look Like?

Surprisingly we know little about insomnia during its acute phase.^{36,37} The only epidemiological study specifically focusing on acute insomnia, conceptualized as DSM-5 defined insomnia disorder, lasting between 3 days and 3 mo, found a prevalence rate of 7.9% and an annual incidence rate of 36.6%.³³ In terms of the sleep of individuals with acute insomnia, this group self-reports and objectively demonstrates poorer sleep compared with normal sleepers.^{34,35} Interestingly, a recent, albeit preliminary study demonstrated that individuals with acute insomnia and chronic insomnia do not differ with respect to objective sleep parameters (actigraphy), levels of worry, or levels of sleep preoccupation. Those with chronic insomnia, however, reported higher symptom severity compared to those with acute insomnia.³⁸ Additionally, where accumulated evidence has shown that having chronic insomnia confers a twofold increased risk for the development of major

depression,⁶ a recent study from our group demonstrates that the transition from acute to chronic insomnia represents a “critical vulnerability period” for the onset of a first-ever episode of depression, independent of the precipitant that caused the initial insomnia (i.e., number of life events experienced and levels of perceived stress).³⁴ Finally, in one study, the main predictors of the transition to chronic insomnia, as opposed to natural remission, were largely cognitive in nature (i.e., higher levels of sleep preoccupation, more sleep related dysfunctional attitudes and beliefs, higher self-reported fatigue) although behavioral elements were also evident (i.e., reported use of substances as a coping mechanism).³⁹ As such, it can be concluded that (1) stress-reduction techniques alone would be unlikely to work with individuals with acute insomnia, and (2) a cognitive behavioral framework, such as CBT-I, is a likely treatment candidate for this group. To further clarify, a treatment was required that would address the cognitive—sleep related dysfunctional attitudes and beliefs, sleep preoccupation, and worry (i.e., using sleep education, imagery distraction techniques, and cognitive control), symptomatic—objective and subjective sleep disturbances (i.e., using sleep restriction and stimulus control), and behavioral—the use of substances (i.e., using sleep education), elements associated with acute insomnia.

Study Aims

The aim of the current study was to determine the extent to which a single 60- to 70-min session of CBT-I, with an accompanying self-help pamphlet, is efficacious for the treatment of acute insomnia. A single session was chosen because of its demonstrated superiority over two and eight sessions²⁹ and the amount of therapist contact time was broadly in line with the successful ACBT and BBTI studies with individuals with chronic insomnia.^{27,28} It was hypothesized that this variant of CBT-I would result in significantly higher levels of remission, as defined by caseness scores on the Insomnia Severity Index (ISI)⁴⁰ at follow-up, compared to controls. The secondary hypothesis was that there would be significant reductions in insomnia symptoms, as measured through sleep diaries and pre-post change scores on the ISI, for those in the treatment group compared to controls.

METHODS

Development and Feasibility of the Self-Help Pamphlet

A self-help pamphlet, outlining the principles of Stimulus Control, Cognitive Control (from Espie⁴¹), and the use of Imagery Distraction techniques (from Harvey and Payne⁴²), was developed to augment the single session of CBT-I. This was framed in a simple message of the 3D's (*Detect* – how to record a sleep diary; *Detach* – stimulus control instructions; and *Disrupt* – cognitive control and imagery distraction instructions). A feasibility study was undertaken with 15 undergraduate students (73.3% female, mean age 20.2 ± 1.01 y) with self-reported acute insomnia, recruited through advertisements on campus, to examine the capacity of the information pamphlet to reduce both cognitive and somatic arousal. Subjects were given a week-long sleep diary, including an additional section which asked the subject to record the response to two questions

“How mentally alert were you in bed last night?” and “How physically tense were you in bed last night?” Both questions were scored on a Likert-type scale, from 0 = “not at all” to 4 = “very.” Scores were averaged across the number of nights completed (range 0–4: Mean completion at each assessment 7 ± 0 days). These questions provided indices of cognitive and somatic arousal, with higher scores indicating higher levels of arousal. A week following the baseline sleep diaries, subjects were met and given the self-help pamphlet, told to read it thoroughly, and given the opportunity to ask questions regarding its content. Subjects were then asked to implement the strategies outlined in the pamphlet and record their sleep using another week-long sleep diary (with the additional questions). Repeated *t* tests showed that subjects reported significantly less cognitive (prepamphlet mean 2.32 ± 0.64 ; postpamphlet mean 1.38 ± 0.61 ; $t(14) = 5.78$, $P < 0.001$) and somatic (prepamphlet mean 1.55 ± 0.54 ; postpamphlet mean 0.96 ± 0.77 ; $t(14) = 4.33$, $P < 0.001$) arousal at follow-up.

Recruitment and Procedure

Prospective subjects were recruited from a regional media campaign in the North East of the UK, which included a public engagement event hosted at the Centre for Life in Newcastle by the study's principal investigator (JGE). At each event contact details were provided. Potential subjects were contacted for an initial clinical interview by telephone to determine eligibility. The clinical interview began with a brief description of the study. Interested individuals provided informed consent and began the interview to establish (1) DSM-5-defined insomnia status; (2) presence and history of other intrinsic sleep disorders, or medical and psychiatric conditions; and (3) previous history of insomnia (see the study by Ellis and colleagues³³ for detailed description of the interview).

If subjects met DSM-5 criteria for insomnia, three additional questions were asked. The first, “...for how long has this [the sleep problem] been going on?” was to differentiate acute insomnia (i.e., meeting DSM-5 criteria for insomnia disorder but for a duration of less than 3 mo) from chronic insomnia (i.e., meeting DSM-5 criteria for insomnia disorder, including the duration threshold of over 3 mo). The second question, “...can you recall an event or circumstance which may have triggered this episode of poor sleep?” was used to provide details on precipitating factors, including illnesses, that may influence treatment outcome. The final question, “...are you currently, or have you in the past, received any treatment for your sleep problem?” was to exclude subjects on the basis of having previous exposure to CBT-I or current use of sleep medication. If a subject did not meet criteria for acute insomnia at this point, he or she was thanked for his or her time and the interview was terminated.

If the subject met full study criteria (i.e., met DSM-5 criteria for insomnia disorder but reported a duration of less than 3 mo for the current episode, and had no previous exposure to CBT-I or were taking a sleep medication), he or she was asked if to take part in a nonpharmacological treatment study. If subjects agreed, a copy of the ISI and a sleep diary were sent to them the following day (pretreatment measures). Randomization was by achieved by selecting from an envelope a piece of paper labeled as either 1 = CBT-I or 2 = wait list control on a

quota-sampling basis by sex. In other words, after the quota of available spaces for females or males in a group was met, the next subject of that sex was automatically assigned to the other condition. This was to ensure equal numbers of males and females between each group. Randomization occurred when the recruitment of all subjects was complete and the individual who randomized was blind to group allocation. All subjects were contacted by email or telephone after randomization, and either appointments made to attend the treatment session at the Northumbria Centre for Sleep Research approximately 8 days following the posting of the ISI and sleep diary (CBT-I condition), or informed to expect another copy of the ISI and sleep diary in approximately 5 w (i.e., from the date they received the initial questionnaires) to complete (waitlist control group). This was to coincide with the follow-up sleep diary and ISI in the treatment group.

Single Session CBT-I

The single CBT-I session was delivered on a one-to-one basis by the same therapist (JGE, a practicing health psychologist and somnologist with 5 years' experience delivering CBT-I). The session structure began with sleep education (e.g., the two-process model of sleep⁴³ and Spielman et al.'s 3P model⁴⁴) and a discussion on individual differences in sleep need across the lifespan. This was done to identify and address sleep related dysfunctional thinking and provide a platform from which sleep hygiene could be discussed in relation to its effect on the sleep homeostat and the circadian rhythm. Afterward, the principles of sleep restriction (SR) were introduced and together, using the sleep diaries, each individual's current sleep efficiency (SE), prescribed time in bed (PTIB), and prescribed time out of bed (PTOB), was derived. The session then focused on sleep restriction titration (i.e., 15-min reduction in TIB based upon $< 85\%$ SE, no change in TIB between $85\text{--}90\%$ SE, and 15 min increase in TIB based on $> 90\%$ SE). Subjects were told never to go less than 4.5 h TIB and not to start titration until 1 w of sleep restriction had taken place. Finally, the pamphlet was introduced and the principles of stimulus control, cognitive control, and the use of imagery distraction techniques outlined and discussed (a copy of the pamphlet is available from the corresponding author).

One week following treatment the intervention group was asked to send their sleep diary to the research team (posttreatment assessment). Four weeks after the treatment session, the intervention group was sent the ISI to complete (follow-up assessment) and were asked to send it back 1 w later along with their last sleep diary. At the end of the study all subjects were offered a full course of CBT-I. The protocol for the study received ethical approval from Northumbria University Faculty of Health and Life Science Ethics Committee, and conformed to the Declaration of Helsinki's ethical principles.

As can be seen in Figure 1, 88 telephone-screening interviews were conducted; 43 individuals were excluded on the basis of not meeting criteria for acute insomnia. Of the remaining 45 respondents, four refused to take part and one was deemed ineligible because he or she had planned to undergo a course of cCBTI during the study period. Forty subjects completed the current study (18 males and 22 females: mean age 32.9 ± 13.72 y).

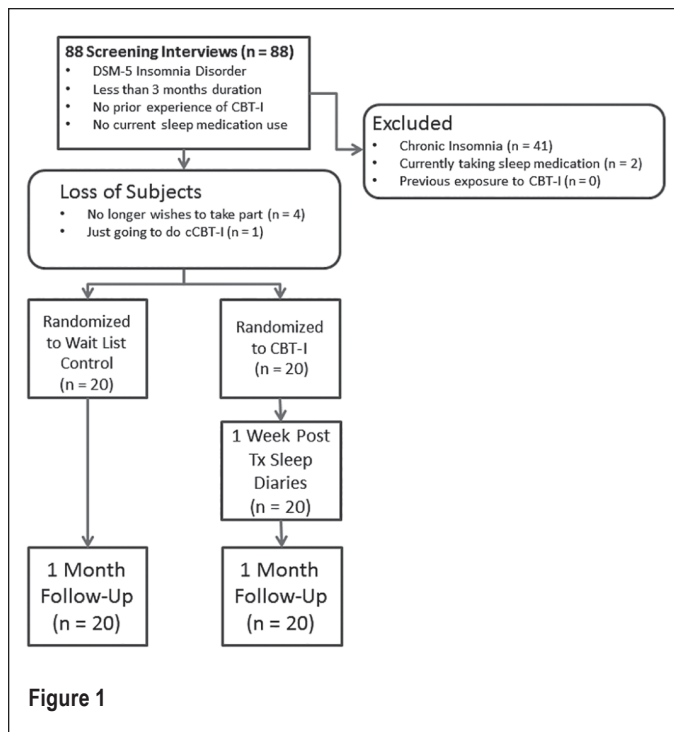


Figure 1

Measures

Insomnia Severity Index

The ISI⁴⁰ is a seven-item measure of the nature, severity, and effect of insomnia. Based on a five point Likert-type scale (0 = no problem; 4 = a severe problem), scores range from 0 to 28. Although the traditional cutoff score for subclinical insomnia is ≥ 8 , recently, Morin and colleagues⁴⁵ provided an optimal cutoff score (≥ 10) for identifying “caseness” for insomnia in community samples. Remission rates were defined as reported scores lower than the subclinical insomnia and optimal cutoff thresholds on the ISI at follow-up (i.e., those who scored < 8 or < 10) and were the primary outcome measure with pre-post change ISI scores serving as a secondary outcome measure. Although the usual duration criteria of the ISI covers the previous month, subjects in this study were asked to complete the ISI on the basis of the previous week to coincide with the sleep diaries.

Subjective Sleep

A consensus sleep diary⁴⁶ was used to derive core measures of subjective sleep continuity (TIB, sleep latency [SL], wake after sleep onset [WASO], number of awakenings [NWAK], total sleep time [TST], and to calculate SE) over a period of 7 continuous days at pretreatment and at follow-up. The diary data were considered a secondary measure in the current study. Subjects were instructed to complete the diary each morning upon waking. Mean values were derived for each variable based upon the number of nights completed (mean continuous completion 7 days ± 0 at baseline and 7 days ± 0 at follow-up).

Power Calculation and Data Analysis

Based on the findings from Edinger and colleagues²⁹ we calculated their effect size as the difference from baseline to the first follow-up assessment point on the Insomnia Symptoms

Questionnaire (ISQ) for the group that received one session of CBT-I. With an effect size of 1.02, power set at 80% and an alpha of 0.05, the minimum required number in each group was 17. Baseline group differences were examined using independent *t* tests. Percentage and pre-post change scores are reported for clinical caseness and chi-square analysis for group differences based upon clinical caseness. A multivariate analysis of variance was used to examine between group differences on pre-post changes in sleep continuity. For all analyses a significance level of $P < 0.05$ was chosen. Missing data were treated by mean substitution when less than 5% of that scale or measure was missing. Above 5% of data missing from a measure resulted in casewise deletion. On this basis no subjects were excluded for missing data.

RESULTS

The final sample consisted of nine males and 11 females in each group. There were no significant between-group differences in age (mean age CBT-I = 32.9 ± 14.02 y, mean age controls = 31.8 ± 11.38 y, $t(38) = 0.27$, $P = 0.79$). Of the sample, 13 (32.5%) reported it as a first episode and 27 (67.5%) described it as a recurrent episode. In terms of precipitants of the current episode of insomnia, 31 reported a nonmedical/psychiatric stressor (e.g., family problems, relationship problems, financial issues, change in occupational circumstances). Of the remaining nine subjects in the sample, one reported having a diagnosis of cancer, two with chronic fatigue syndrome, two with hypertension, two with depression, one with a diagnosis of sleep apnea, and one with chronic pain. In all comorbid cases the subject’s condition was being managed (e.g., continuous positive airway pressure for sleep apnea; see Table 1) by a primary care physician or other healthcare provider. No subjects were undergoing any other psychological intervention at the time of the trial. There were no between-group differences on either the episodic nature of the insomnia or having comorbidity ($P > 0.05$). The overall pretreatment score on the ISI was 14.63 ± 3.43 .

Pretreatment Comparisons

T tests were conducted to determine between-group parity on the sleep variables. There were no differences on ISI scores ($t(38) = 0.78$, $P = 0.44$) or any sleep continuity variables: SL ($t(38) = 0.57$, $P = 0.57$), NWAK ($t(38) = 0.9$, $P = 0.46$), WASO ($t(38) = 0.64$, $P = 0.53$), TST ($t(38) = 0.48$, $P = 0.63$), or SE ($t(38) = 0.53$, $P = 0.6$) (see Table 2 for descriptive statistics).

Adherence to Behavioral Strategies

Adherence was operationalized by comparing each individual’s TIB prescribed at the CBT-I session against their reported TIB over the following week (posttreatment values) in the intervention group. The results suggest 12 of 20 (60%) were adherent to within 15 min of their prescribed TIB and 13 of 20 (65%) were adherent to within 30 min of their prescribed TIB.

Assessment of Insomnia Status at Follow-up

At follow-up there was a significant difference in ISI scores between those in the CBT-I group and those in the control group ($t(38) = 2.24$, $P < 0.05$). Using the criteria of an ISI score < 10 being indicative of insomnia remission, at follow-up

Table 1—Condition and management of condition by group.

Group Allocation	Condition	Management/Treatment
CBT-I	Chronic pain	Co-codamol
CBT-I	Hypertension	Calcium channel blocker
CBT-I	Sleep apnea	Continuous positive airway pressure
CBT-I	Chronic fatigue syndrome	Low dose amitriptyline
CBT-I	Chronic fatigue syndrome	Nothing
CBT-I	Depression	Awaiting talking therapy appointment
Wait list control	Cancer	Hormone therapy
Wait list control	Depression	SSRI
Wait list control	Hypertension	ACE inhibitor

In each case the intervention had been prescribed for at least 4 weeks. ACE, angiotensin-converting enzyme; CBT-I, cognitive behavioral therapy for insomnia; SSRI, selective serotonin reuptake inhibitor.

Table 2—Sleep variables by treatment group.

	CBT-I Group (n = 20)						Control Group (n = 20)				Between-Group Differences on Change Scores Cohen d ^a
	Pretreatment		Posttreatment		Follow-up		Pretreatment		Follow-up		
	M	SD	M	SD	M	SD	M	SD	M	SD	
Insomnia Severity Index (scores)	14.2	3.92	n/a	n/a	9.6	4.99	15.05	2.89	12.65	3.51	0.64
Sleep latency (min)	41.79	21.59	30.02	15.12	21.43	10.48	37.64	24.6	34.6	18.06	0.71
Number of awakenings	1.97	0.92	1.76	0.57	1.48	0.63	1.74	1.03	1.6	1.16	0.27
Wake after sleep onset (min)	57.46	27.27	40.96	20.73	31.55	19.19	63.25	29.89	54.82	23.28	0.77
Total sleep time (min)	344.75	45.88	349.33	38.71	349.03	43.23	352.6	56.6	344.12	60.4	0.28
Sleep efficiency (percentage)	78.38	12.28	73.49	14.13	88.92	14.69	76.3	12.55	75.02	16.19	0.69

^aCohen d was calculated between groups using the mean change scores (pretreatment to follow-up) on each variable. CBT-I, cognitive behavioral therapy for insomnia; M, mean; SD, standard deviation.

12 of 20 participants (60%) in the treatment group had remitted compared to three of 20 (15%) in the control group. This difference in caseness at follow-up was significant ($\chi^2 = 8.64$, $df = 1$, $P < 0.003$) with the CBT-I group reporting significantly higher levels of remission compared to controls. Using the more traditional criteria of ≥ 8 on the ISI, 10 of 20 participants (50%) in the treatment group had remitted at follow-up compared with two of 20 (10%) in the control group. Again, this difference was significant ($\chi^2 = 7.62$, $df = 1$, $P < 0.01$) with the treatment group demonstrating better outcomes than the control group. The next analysis examined whether the severity of insomnia, as indexed by pretreatment scores on the ISI, was related to treatment outcome (change on ISI scores) in the treatment group. The results suggest that initial insomnia severity had no impact on treatment outcome ($r = 0.02$, $n = 20$, $P = 0.93$).

Changes in Sleep Profiles

A one-way between-groups multivariate analysis of variance was used to determine group differences (CBT-I versus controls) in change scores (pretreatment versus follow-up) on the sleep continuity variables from the sleep diaries (SL, NWAK, WASO, TST, and SE). The combined model was significant, $F(5,34) = 3.57$, $P = 0.01$, Wilkes lambda = 0.66; partial eta squared = 0.34. When each individual sleep continuity variable was examined only SL ($F(1,38) = 4.75$, partial eta squared = 0.11), WASO ($F(1,38) = 5.59$, partial eta squared = 0.13), and SE

($F(1,38) = 4.47$, partial eta squared = 0.11) were significant (all at $P < 0.05$). In each case those in the CBT-I group reported better sleep outcomes than those in the control group. Cohen *d* was then calculated to provide between-group effect sizes on change scores for each sleep variable (Table 1).

Follow-up Treatment

Although not a central hypothesis, the number of requests for a full course of CBT-I on completion of the study was used as a gross indicator of treatment acceptability (i.e., one session of 60–70 min). Fourteen subjects (70%) in the control group asked for treatment compared with one subject (5%) in the intervention group. Interestingly, although not wanting an additional full-course of CBT-I, eight subjects (40%) in the intervention group asked for a single “booster” session instead. The primary reason given for this was to discuss relapse prevention.

DISCUSSION

The primary aim of this study was to determine whether a “single-shot” of CBT-I, with an accompanying information pamphlet, was efficacious in the treatment of acute insomnia; additionally, whether this variant of CBT-I would result in improved sleep. The results suggest that this variant of CBT-I is suitable for most individuals with acute insomnia. The intervention was significantly more efficacious compared to the

levels of natural remission seen in the control group (50–60% compared with 10–15%). Furthermore, there were significant differences in sleep continuity between the groups at follow-up with those in the treatment group reporting better outcomes, specifically in terms of SL, WASO, and SE, compared with controls. Together these findings suggest that this variant of CBT-I is sufficient and appropriate to elicit change within this population. Not only does this have economic and social implications, at least in terms of the direct costs associated with insomnia,¹⁰ but considering that chronic insomnia is a risk factor for the development of mood and anxiety disorders, chronic physical conditions, and substance use disorders,^{1–6} the findings have, potentially, much wider ramifications.

The current findings also hold relevance for the stepped-care model for the treatment of insomnia, first proposed by Espie in 2009.⁴⁷ Within that framework it is envisioned that patients should be allocated to a level of intensity of CBT-I commensurate with their needs. Where the model, in its current form, does not differentiate acute from chronic insomnia, there is an implied, and logical, assumption contained within suggesting individuals would most likely need a less intensive form of CBT-I during the early phase of insomnia. This is presumably due to a lack, or at least lower levels, of conditioned presleep arousal and “insomnia” self-schemata (i.e., less influence from perpetuating factors). Where no studies to date have compared individuals with acute insomnia and chronic insomnia on levels of presleep arousal, or indeed some of the cognitive variables that make up the self-defining characteristics of having “insomnia,” the findings from both the feasibility and main study provide (1) an argument for the inclusion of a cognitive component in brief therapies for acute insomnia, and (2) a compelling case for this variant of CBT-I being sufficient for this population. It remains to be seen whether the reason for the success of this intervention, in this population, is due to fewer ingrained perpetuating factors to address, a lower severity of symptoms at initial presentation, or perhaps a combination thereof. Future intervention research in this population may wish to consider including measures such as the Glasgow Sleep Effort Scale (GSES),⁴⁸ Pre-Sleep Arousal Scale (PSAS),⁴⁹ Sleep Preoccupation Scale (SPS),⁵⁰ and/or Dysfunctional Beliefs and Attitudes to Sleep (DBAS) scale⁴⁰ to assess the contribution of these constructs on treatment outcome.

The main effect size for the outcome measure (between groups difference on ISI change scores) observed in the current study ($d = 0.64$), albeit large, was not as robust as those reported or derived from the previous studies using hybrid variants of CBT-I.^{27,28} Where it could be suggested that this intervention may be less than optimal for this group, there may be several alternative explanations for this. Primarily, the high levels of natural remission, normally seen in this population³³ and observed here, would have had the potential to dilute the observed between-group differences. As this is less of an issue in chronic insomnia, due to low levels of natural remission,⁹ it is reasonable to assume that this may explain the differences in effect sizes. Alternatively, these differences may simply reflect the different outcome measures used in previous studies (i.e. the PSQI²⁸ and ISQ²⁹ compared with the ISI in the current study). However, the significant main effect and changes in the sleep variables demonstrate the superiority of the CBT-I

intervention above that of natural remission. Moreover, the effect sizes for SL, WASO, and SE appear to be broadly similar to what has been observed previously. Future studies might wish to triangulate outcome measures to determine which gives the best representation of actual outcomes following CBT-I in this population.

Interestingly, only one individual in the treatment group asked for a full course of CBT-I on completion of the study compared to 70% in the control group. This finding could be interpreted in two ways; either the current delivery format was acceptable for the population on the whole, with only one person wanting to “step-up” to the next level of treatment intensity on offer, or that the next “step-up” was perceived as too intense. In the only study that has examined the stepped care model in practice, 15.79% of their sample of individuals with chronic insomnia went from a single-session dose to a full course of CBT-I, delivered individually.⁵¹ As such, the current finding of only 5% wanting to “step-up” is encouraging. That said, 40% of those who received the intervention requested an additional booster session to address relapse prevention. Based on these findings it would certainly be interesting to conduct a “head-to-head” trial of the current single session, with accompanying pamphlet, against a two-session dose to see whether it improves outcomes, or as in the case of Edinger and colleagues²⁹ relates to poorer treatment outcomes in this population.

Limitations

There are limitations to the current study that suggest a degree of caution when interpreting the results. This was a self-selecting sample and as such the findings may not be representative of all individuals with acute insomnia. That said, the composition of the current sample broadly matches what has previously been reported in the only epidemiological study on acute insomnia in terms of age, sex, frequency of occurrence, and natural remission rates.³³ Further, as the use of sleep medication was an exclusion criterion, it is unknown how effective the current intervention would be for a population of individuals with acute insomnia taking sleep medications. However, of those individuals recruited with acute insomnia only two were excluded on the basis of currently taking sleep medication, suggesting this may be a minor issue for this population. Irrespective, a replication study with a more representative group of individuals with acute insomnia, including those on sleep medication, would be a next logical step to shed light on these issues. Additionally, the follow-up period (1 mo post-treatment) could be considered too short a time period to determine whether the changes observed in the treatment group were sustainable. Although this is true and as such no claims can be made regarding the long-term efficacy of the intervention, the decision to select this time period was made largely on ethical grounds. Considering this was the first study to attempt to treat insomnia during the acute phase, a full course of CBT-I was offered to all subjects as soon as possible after the follow-up period. This was to ensure that those who had, by then, transitioned from acute insomnia to chronic insomnia were treated using the best evidence-based intervention. Of note, follow-up data at 3 mo with 19 of the 20 individuals who were in the initial treatment group (one subject was excluded

because he or she was given a full course of CBT-I after the 1-mo follow-up) using the ≤ 10 criteria on the ISI showed that all 12 who had remitted by the 1-mo follow-up were still in remission at 3 mo. Furthermore, two additional participants who had previously not remitted by the 1-mo follow-up had remitted by 3 mo (i.e., a total 3-mo remission rate of 14 of 19 = 73.68%). However, it should be noted that these are observational outcomes as opposed to experimental results and should be interpreted as such. Certainly, now the acceptability and initial efficacy of the intervention have been demonstrated, longer-term follow-ups would be a most welcome and logical next step. The other main limitation was the measure of adherence used in the current study. Subjects were not directly assessed as to whether they adhered to all components in the intervention; only adherence to “prescribed” sleep restriction over the first week of the intervention was used as an indicator. Because there is no consensus on the measurement of adherence for CBT-I,⁵² the rationale for using this method was twofold: (1) most studies previously examining adherence to CBT-I used sleep diaries as their main or only outcome measure,⁵² and (2) the behavioral components of CBT-I are particularly vulnerable to non-adherence and as such are likely to act as either a good, or even conservative, indicator of overall adherence.^{54,55} Again, future studies should be mindful of how they measure adherence, possibly triangulating between TIB measures, self-reports, manipulation checks, and actigraphy.

CONCLUSION

The aim of the current study was to determine whether a “single-shot” of CBT-I, with an accompanying self-help pamphlet, was sufficient to treat acute insomnia. The findings suggest that it is, in approximately 50–60% of cases, but an additional “booster” session may be a helpful adjunct to address patients concerns about relapse. Certainly there is a need for replication because this is the first CBT-I study to attempt to treat acute insomnia. However, these preliminary results are promising. Along with replication we should also be mindful about how this treatment and this population fits not only into the stepped care model of insomnia but how the stepped care model, with these findings embedded, should be integrated into primary care.

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