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Citation: Ellis, Jason, Cushing, Toby and Germain, Anne (2015) Treating Acute Insomnia: A Randomized Controlled Trial of a "Single-Shot" of Cognitive Behavioral Therapy for Insomnia. Sleep, 38 (06). pp. 971-978. ISSN 1550-9109

Published by: Associated Professional Sleep Societies

URL: http://dx.doi.org/10.5665/sleep.4752 <http://dx.doi.org/10.5665/sleep.4752>

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Treating Acute Insomnia: A Randomised Control Trial of a 'Single-shot' of Cognitive Behavior Therapy for Insomnia

Short Title: Treating acute insomnia using CBT-I

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Conflicts of Interest: This was not an industry-funded study. Dr Ellis has received an educational grant and speaking honoraria from UCB Pharma, and a research grant from Transport for London. He has also consulted for the British Broadcasting Corporation. None of these conflicts of interest are related to the current manuscript. Ms Cushing and Dr. Germain report no conflicts of interest.

Treating Acute Insomnia: A Randomised Control Trial of a 'Single-shot' of Cognitive Behavior Therapy for Insomnia

Key Words: Acute Insomnia, CBT-I, Brief Intervention, Short-term Insomnia, Treatment, Stepped Care

ABSTRACT

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 control trial.

Setting: Community.

Participants: 40 adults (mean age 32.9±13.72) with DSM-5 defined Insomnia Disorder, except a self-reported duration of less than 3 months (i.e. acute insomnia), who reported no previous exposure to CBT-I and were not currently taking medication for sleep.

Interventions: A single 60-70 minute 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 pre-treatment and one month 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 ($t(38)-2.24, p<.05$) at follow-up. Further, using proposed Insomnia Severity Index scores for identifying insomnia caseness (i.e. ≤10), 60% of those in the CBT-I group had remitted by one month compared to 15% of those in the control group.

Conclusions: This single-session of 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.

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 a year 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 $13.9Billion annually)[10], and largely unrelenting condition.

Standard Cognitive Behavioral Therapy for Insomnia (CBT-I), a six-eight week 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 co-morbid 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 (cCBTI)[18-21]. Despite the introduction and widespread dissemination of these innovative modalities, early indications suggest there may be a trade-off 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 x 25 minute sessions, with an accompanying pamphlet) and BBTI (a single 45-minute session with a 30 minute 'booster' session two weeks later) were sufficient to confer benefits for those with chronic insomnia[27-28]. As for the optimal dose, although Edinger and colleagues[29] found a four-session dose of CBT-I was optimal (58.3% patients demonstrated clinically-relevant improvements), they also showed that a single 45-60 minute session was superior to a two-session dose and a full eight-session dose (43.8% vs. 22.2% and 35.3% respectively). As such, it appears that a modified CBT-I / BBTI that involves contact somewhere between 45 and 75 minutes (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 (DSM-5) states, for the first time, that if full criteria for Insomnia Disorder are met, bar the minimum duration criteria of three months, 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 three days and three months, 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 to 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 two-fold 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; a) stress-reduction techniques alone would be unlikely to work with individuals with acute insomnia, and b) a cognitive – behavioral framework, such as CBT-I, is a likely treatment candidate for this group. The 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 present study was to determine the extent to which a single 60-70 minute session of CBT-I, with an accompanying self-help pamphlet, is efficacious for the treatment of acute insomnia. A single session was chosen due to its demonstrated superiority over two and eight sessions[29] and the amount of therapist contact time was broadly in line with the successful ACBT and BBTI studies with individuals with chronic isnomnia[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 Espie[41]), and the use of Imagery Distraction techniques (from Harvey & Payne[42]), 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 *Distract* – cognitive control and imagery distraction instructions). A feasibility study was undertaken with 15 undergraduate students (73.3% female, mean age 20.2 ± 1.01) 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 through 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 (Pre-pamphlet Mean 2.32 ± .64; Post-pamphlet Mean 1.38 ± .61; $t(14)=5.78$, $p<.001$) and somatic (Pre-pamphlet Mean 1.55 ± .54; Post-pamphlet Mean .96 ± .77; $t(14)=4.33$, $p<.001$) 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 PI (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: a) DSM-5-defined insomnia status, b) presence, and history, of other intrinsic sleep disorders, medical and psychiatric conditions, and c) previous history of insomnia (see Ellis and colleagues[33] 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 months) from chronic insomnia (i.e. meeting DSM-5 criteria for Insomnia Disorder, including the duration threshold of over 3 months). 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 they were thanked for their time and the interview was terminated.

If the subject met full study criteria (i.e. they met DSM-5 criteria for Insomnia Disorder but reported a duration of less than three months for the current episode, and had no previous exposure to CBT-I or were taking a sleep medication) they were asked if they would like to take part in a non-pharmacological treatment study. If subjects agreed they were posted the ISI and a sleep diary the following day (pre-

treatment measures). Randomization was by achieved by picking, from an envelope, a piece of paper labeled as either - 1 = CBT-I or 2 = wait list control on a quota-sampling basis by gender. In other words, once the quota of available spaces for females or males in a group was met, the next subject of that gender 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 weeks' time (i.e. from the date they received the initial questionnaires) to complete (wait-list 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 five years' experience delivering CBT-I). The session structure began with sleep education (e.g. the two process model of sleep[43] and Spielman's 3P model[44]) 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 impact on the sleep homeostat and the circadian rhythm. Following, the principles of sleep restriction (SR) were introduced and together, using the sleep diaries, each individual's current sleep efficiency (SE), prescribed time-to-bed, and prescribed time-out-of-bed, was derived. The session then focused on sleep restriction titration (i.e. 15 minute reduction in TIB based upon <85% SE, no change in TIB between 85-90% SE, and 15 minutes increase in TIB based on >90% SE). Subjects were told never to go below 4.5hours TIB and not to start titration until one week 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 (post-treatment assessment). Four weeks following the treatment session, the intervention group was sent the ISI to complete (follow up assessment) and were asked to send it back a week 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 as they had planned to undergo a course of cCBTI during the study period. 40 subjects completed the present study (18 males and 22 females: Mean age 32.9 $\pm$ 13.72).

Insert Figure 1 Here

Measures

Insomnia Severity Index. The ISI[40] is a seven-item measure of the nature, severity and impact of insomnia. Based on a five point Likert-type scale (0 = no problem – 4 = a severe problem), scores range from 0 – 28.

Although the traditional cut-off score for sub-clinical insomnia is ≥8, recently, Morin and colleagues[45] provided an optimal cut-off score (≥10) for identifying 'caseness' for insomnia in community samples. Remission rates were defined as those subjects who reported scores lower than the sub-clinical insomnia and optimal cut-off 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 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 diary[46] was used to derive core measures of subjective sleep continuity (Time in Bed [TIB], Sleep Latency [SL], Wake After Sleep Onset [WASO], Number of Awakenings [NWAK], Total Sleep Time [TST], and to calculate Sleep Efficiency [SE]) over a period of seven continuous days at pre-treatment and at follow-up. The diary data was considered a secondary measure in the present 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 upon the findings from Edinger and colleagues[29] 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 .05, the minimum required number in each group was 17. Baseline group differences were examined using independent t-tests. Percentage and change scores are reported for clinical caseness and chi-square analysis for group differences based upon clinical caseness. A multivariate ANOVA was used to examine

between group differences on pre-post changes in sleep continuity. For all analyses a significance level of $p<.05$ was chosen. Missing data was 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 9 males and 11 females in each group. There were no significant between-group differences in age (Mean age CBT-I = 32.9 $\pm$ 14.02, Mean age controls = 31.8 $\pm$ 11.38, $t(38)=.27$, $p=.79$). 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 non-medical/psychiatric stressor (e.g. family problems, relationship problems, financial issues, change in occupational circumstances). Of the remaining 9, 1 was associated with a diagnosis of cancer, 2 with chronic fatigue syndrome, 2 with hypertension, 2 with depression, 1 with a diagnosis of sleep apnoea, and 1 with chronic pain. In all co-morbid cases the subject's condition was being managed (e.g. Continuous Positive Airway Pressure for sleep apnoea – 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 co-morbidity ($p>.05$). The overall pre-treatment score on the ISI was 14.63 $\pm$ 3.43.

Insert Table 1 Here

Pre-treatment Comparisons

T-tests were conducted to determine between-group parity on the sleep variables. There were no differences on ISI scores (t(38)=.78, p=.44) or any sleep continuity variables: SL (t(38)=.57, p=.57), NWAK (t(38)=.9, p=.46), WASO (t(38)=.64, p=.53), TST (t(38)=.48, p=.63), or SE (t(38)=.53, p=.6) (see Table 2 for descriptive statistics).

Insert Table 2 Here

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 (post-treatment values) in the intervention group. The results suggest 12/20 (60%) were adherent to within 15 minutes of their prescribed TIB and 13/20 (65%) were adherent to within 30 minutes of their prescribed TIB.

Assessment of Insomnia Status at Follow Up

At follow up there was a significant difference between those in the CBT-I group and those in the control group on ISI scores (t(38)=2.24, p<.05). Using the criteria of an ISI score $\leq$10 being indicative of insomnia remission, at follow up 12 out of 20 (60%) participants in the treatment group had remitted compared to 3 out of 20 (15%) in the control group. This difference in caseness at follow-up was significant (χ^2=8.64, df = 1, p<.003) with the CBT-I group reporting significantly higher levels of remission compared to controls. Using the more traditional criteria of $\leq$8 on the ISI, 10 out of 20 (50%) participants in the treatment group had remitted at follow up compared to 2 out of 20 (10%) in the control group. Again, this difference was significant (χ^2=7.62, df = 1, p<.01) with the treatment group demonstrating better outcomes than the

control group. The next analysis examined whether the severity of insomnia, as indexed by pre-treatment 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=.02, n = 20, p=.93).

Changes in Sleep Profiles

A one-way between-groups MANOVA, was used to determine group differences (CBT-I vs controls) in change scores (pre-treatment vs 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=.01, Wilkes' Lambda = .66; partial eta squared = .34. When each individual sleep continuity variable was examined only SL (F(1,38)=4.75, partial eta squared = .11), WASO (F(1,38)=5.59, partial eta squared = .13), and SE (F(1,38)=4.47, partial eta squared = .11) were significant (all at p<.05). In each case those in the CBT-I group reported better sleep outcomes than those in the control group. Cohen's d's were 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. 1 session of 60-70 minutes). 14 (70%) subjects in the control group asked for treatment compared to 1 (5%) subject in the intervention group. Interestingly, although not wanting an additional full-course of CBT-I, 8 (40%) subjects 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 the majority of 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 to 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 sleep latency, wake after sleep onset, and sleep efficiency, compared to 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 present 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 envisaged that patients should be allocated to a level of intensity of CBT-I commensurate with their needs. Where the model, in its present 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 pre-sleep 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 pre-sleep 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; a) an argument for the inclusion of a cognitive

component in brief therapies for acute insomnia, and b) 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 scale (DBAS)[40] 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 present study (*d*=.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 population[33], 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 PSQI[28]and ISQ[29] compared to the ISI in the present study). Irrespective, 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 1 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 present 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 present single session, with accompanying pamphlet, against a two-session dose to see whether it improves outcomes, or as in the case of Edinger and colleagues[29] relates to poorer treatment outcomes in this population.

Limitations

There are limitations to the present 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 present sample broadly matches what has previously been reported in the only epidemiological study on acute insomnia in terms of age, gender, 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 present 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 minimal 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 month post treatment) could be considered too short a time period to determine whether the changes observed in the treatment group were sustainable. Whilst

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 ever study to attempt to treat insomnia during the acute phase, a full course of CBT-I was offered to all subjects as soon after the follow-up period as possible. This was to ensure that those who had, by then, transited from acute insomnia to chronic insomnia were treated using the best evidence-based intervention. Of note, follow-up data at three months with 19 of the 20 individuals who were in the initial treatment group (one subject was excluded as they were given a full course of CBT-I after the one-month follow up) using the <10 criteria on the ISI showed that all 12 who had remitted by the one-month follow up were still in remission at three months. Furthermore, two additional participants who had previously not remitted by the one-month follow-up had remitted by three months (i.e. a total three-month remission rate of 14/19 = 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 present 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. As there is no consensus on the measurement of adherence for CBT-I[52] the rationale for using this method was two-fold; 1) the majority of 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 present 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 as this is the first CBT-I study to attempt to treat acute insomnia. However, these preliminary results are promising. Alongside 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.

Acknowledgements:

We would like to thank Kate Fennell for her support in collecting and collating the data. The PI (JGE) had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Trial Registration:

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

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