Title

Is digital cognitive behavioural therapy for insomnia effective in treating sub-threshold insomnia: A pilot RCT

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Abstract

Objective/Background: CBT for insomnia (CBT-I) is useful for many. It is currently unknown if those with sub-threshold insomnia also benefit. Here we assessed whether CBT-I is both feasible and acceptable in participants with sub-threshold insomnia. The primary aims were to evaluate participation rates and treatment acceptability, and to establish an effect size for symptom improvement.

Patients/Methods: A total of 199 female participants (M_{age} 20 ± 5 years) took part. Following baseline assessments, participants were randomly allocated to either a 6-week digital CBT-I intervention or a 6-week session control group receiving puzzles. Additional assessments were performed 3-weeks, 6-weeks, and 6-months later.

Results: Participation in each survey wave did not differ between the groups (*p*s > .140), though adherence to weekly tasks was lower in the CBT-I group, $p = .02$. Treatment acceptability was high (M (SD) = 33.61 (4.82), range 6 – 42). The CBT-I group showed greater improvement in insomnia symptoms at the end of the intervention compared to the control group ($p = .013$, $d = 0.42$), with significant variation in outcome (M = 4.69, SD = 5.41). Sub-threshold participants showed a similar pattern of results, whilst those meeting insomnia criteria showed a smaller between-group difference. CBT-I led to improvements in anxiety, paranoia and perceived stress between baseline and end of intervention. Changes in insomnia symptoms were mediated by cognitions about sleep and somatic pre-sleep arousal.

Conclusions: CBT-I provides a benefit even in sub-threshold insomnia. CBT-I may be useful as an early preventative intervention to tackle sleep problems before they manifest as chronic insomnia.

Keywords

cognitive behavioural therapy, insomnia, sleep complaints

1. Introduction

Insomnia occurs frequently and causes a substantial burden to society (Kessler et al., 2011). Cognitive behavioral therapy for insomnia (CBT-I) has been shown to be a highly effective in reducing insomnia symptoms (Seyffert et al., 2016; Trauer, Qian, Doyle, Rajaratnam, & Cunnington, 2015; van Straten et al., 2018). As such, CBT-I is now the first-choice treatment for insomnia (Qaseem et al., 2016; Riemann et al., 2017). As well as reducing insomnia symptoms, CBT-I can also reduce non-insomnia complaints such as anxiety and depression symptoms, levels of paranoia, and hallucinatory experience (Freeman et al., 2017; Ye et al., 2015). More generally, sleep-related quality of life and psychological well-being have been shown to be improved by CBT-I (Espie et al., 2019).

Whilst it is clear that CBT-I is beneficial to those who already have a diagnosis of insomnia, it is unknown whether those without insomnia would also see benefits from CBT-I. Two previous studies have shown that even those with sub-threshold insomnia symptoms report poorer quality of life (including mental health difficulties) than those reporting no sleep problems at all (LeBlanc et al., 2007; Léger, Scheuermaier, Philip, Paillard, & Guilleminault, 2001). Given this, it is important to investigate whether CBT-I can lead to improvements in those who report sub-threshold insomnia. Furthermore, it is possible that being able to successfully administer CBT-I when only a few symptoms are present, could act as an important preventative measure against the development of more severe insomnia disorder.

Even though approximately 70% of patients show improvements with CBT-I, not everyone responds to treatment (Ritterband et al., 2017). Understanding moderators of treatment response will aid in identifying the best therapeutic solution for a given individual. Moderators of improvement in insomnia symptoms following CBT-I are still relatively unexplored (Luik, Kyle, & Espie, 2017). Accumulating evidence of genetic predictors of CBT outcome in a range of anxiety disorders and depression have been reported (Andersson et al., 2013, 2019; Bryant et al., 2010; Hudson et al., 2013; Lonsdorf et al., 2010), and holds promise for delivering a fuller understanding of psychopathology and the ability to tailor treatments to individuals (Beevers & McGeary, 2012).

Detecting moderation, especially genetic moderators, requires a high amount of statistical power. Given recent developments of digital CBT-I platforms, administering CBT-I to large genetically sensitive cohorts, such as twin studies, is now feasible. Twin pairs can be concordant or discordant for insomnia, meaning that some participants in large cohorts may not have insomnia diagnosis. Therefore, it needs to be established whether CBT-I leads to an improvement in insomnia symptoms in a sample where some participants report sub-threshold symptoms. Furthermore, it is unknown whether such individuals would find a CBT-I intervention acceptable, and the extent to which there would be drop-out. Addressing these questions constitutes the primary aims of the present study.

The overall aim of this work was to establish the feasibility of using CBT-I in a sample not specifically selected for insomnia disorder, in order to establish the effect size of symptom change following the intervention. We were interested in further understanding the usefulness of such an intervention for those with sub-threshold insomnia, and to enable the design and implementation of a large-scale twin study of symptom change following CBT-I. The specific aims were:

1. Primary aim 1. Feasibility: Assess participant rates, adherence, and treatment acceptability throughout the study.
2. Primary aim 2: Determine the effect size for the between group (CBT-I vs control) change in insomnia symptoms from baseline to post intervention.
3. Secondary and exploratory aims: Exploratory analyses to investigate effect sizes for changes in other associated, non-insomnia, variables, and moderators and mediators of change.

2. Methods

2.1. Design

For full details of the design of this feasibility study, see the study protocol (Denis et al., 2017). The study timeline and flow of participants through the study is shown in Figure 1. Participants were recruited from three London universities. Inclusion criteria were: female, enrolled on a psychology degree at one of the study sites, and no previous experience with the intervention program. The decision to only include females was made on the basis that the recruitment pool was majority female so adding males would create heterogeneity

without sufficient power to examine possible sex differences (Denis et al., 2017). There were no other specific inclusion or exclusion criteria. Recruitment involved advertising the study at the end of student lectures, and through e-mail lists. Participants wanting to take part were given a study information document and were given the option to ask the researchers any questions. After providing informed consent, participants completed a baseline assessment.

After the collection of baseline data, participants were randomly allocated to either the intervention (6-session digital CBT-I program) or control (6 sessions of online puzzles) group. The control was chosen on the basis that it took a similar amount of time each week as the intervention, was deemed cognitively engaging/demanding, and was not expected to improve insomnia symptoms. Randomization was performed using the blockrand package for R (Snow, 2013), stratified for age, baseline insomnia symptoms, and study site. Following allocation, participants completed three further online assessments. These were carried out online at 3 weeks (mid-intervention), 6 weeks (end of intervention), and 6 months after starting the intervention (follow-up). All assessment sessions were completed using the online survey platform Qualtrics. A total of 240 participants consented to take part in the study (meeting the enrolment estimate on the trial registration), with 199 completing the baseline assessment and being allocated to a group. The study received ethical approval from the Research Ethics and Integrity subcommittee at Goldsmiths, University of London (reference number: EA 1305). The trial was registered on clinicaltrials.gov (registration number: NCT03062891).

2.2. Measures

2.2.1. Primary outcome measures

Treatment acceptability questionnaire (TAQ) (Hunsley, 1992) – A 6-item measure that assesses treatment acceptability of psychological treatments. Participants in the CBT-I group were asked the degree to which they found the treatment acceptable, ethical, effective, and about the likelihood of negative side effects on a 7-point scale. They were also asked two questions specifically about the nature of the therapist, regarding how knowledgeable and trustworthy participants judged him to be. All items were summed together to generate an overall score with a theoretical range of 6-42. A higher score equates to a more acceptable treatment.

Sleep condition indicator (SCI) (Espie et al., 2014) – In this 8-item questionnaire, participants consider a typical night in the last month and rate various aspects of their sleep. Scores can range between 0-32. Higher scores indicate fewer insomnia symptoms, and scores <= 16 indicate probable insomnia disorder. The SCI has a reliable change index (RCI) of 7, suggesting that a change of 7 or more points is a meaningful improvement (Espie et al., 2018).

2.2.2. Secondary and exploratory measures

The following measures were included as associated outcome measures, and potential moderators and mediators. A full description can be found in the study protocol (Denis et al., 2017): Anxiety symptoms (state trait anxiety index; Spielberger, Gorsuch, Lushene, Vagg, & Jacobs, 1983); depression symptoms (mood and feelings questionnaire; Angold et al., 1995); ADHD symptoms (ADHD symptoms questionnaire; Gregory, Agnew-Blais, Matthews, Moffitt, & Arseneault, 2017); psychotic experiences - paranoia, hallucinations, and cognitive disorganization (specific psychotic experiences questionnaire; Ronald et al., 2014); positive mental health (positive mental health scale; Lukat, Margraf, Lutz, van der Veld, & Becker, 2016); perceived life stress (perceived stress scale; Cohen, Kamarck, & Mermelstein, 1983); threatening life events (list of threatening experiences; Brugha, Bebbington, Tennant, & Hurry, 1985; Coddington, 1983); general sleep quality (Pittsburgh sleep quality index; Buysse, Reynolds, Monk, Berman, & Kupfer, 1989); dysfunctional beliefs about sleep (Dysfunctional beliefs and attitudes about sleep questionnaire; Espie, Inglis, Harvey, & Tessier, 2000); somatic and cognitive pre-sleep arousal (Pre-sleep arousal scale; Nicassio, Mendlowitz, Fussell, & Petras, 1985); sleep disturbances typically related to trauma (PSQI addendum; Germain, Hall, Krakow, Katherine Shear, & Buysse, 2005); and chronotype (Munich chronotype questionnaire; Roenneberg, Wirz-Justice, & Merrow, 2003).

Scale reliabilities are displayed in **Supplementary Table 1**. Full information about the measures administered at each wave can be found in the published study protocol (Denis et al., 2017).

Groups

2.3. Intervention: Digital CBT-I

Participants received 6 weekly CBT-I sessions delivered by an animated 'virtual therapist' via the online platform 'Sleepio'. The program comprised a fully automated media-rich web application. It is driven dynamically by baseline, adherence, performance and progress data, and provides additional access to elements such as an online library with background information, a community of fellow users and support, prompts and reminders sent by email. The Sleepio program covers behavioral (e.g., sleep restriction, stimulus control) and cognitive (e.g., putting the day to rest, thought restructuring, imagery, articulatory suppression, paradoxical intention, mindfulness) strategies, as well as additional relaxation strategies (progressive muscle relaxation and autogenic training) and advice on lifestyle and bedroom factors (sleep hygiene). As part of the intervention, participants filled in a daily sleep diary. For more details see elsewhere (Espie et al., 2012). The intervention was based on a previously validated manual (Espie et al., 2007, 2008; Espie, Inglis, & Harvey, 2001). Sleepio has been shown to improve sleep and associated daytime functioning in adults with insomnia complaints (Bostock, Luik, & Espie, 2016; Cheng et al., 2018; Espie et al., 2012, 2019; Freeman et al., 2017).

2.4. Control: Puzzles

Participants in the control group were sent weekly puzzles to complete using Qualtrics. Each puzzle was designed to be cognitively engaging, and time taken to complete a puzzle was matched as closely as possible to the time taken to complete one session of digital CBT-I. Puzzles were sent directly to participants via automated distribution emails sent at 7-day intervals. In order to track whether participants were completing the puzzles, they were required to enter their participant ID number at the start of each puzzle. The types of puzzles administered to participants included word searches, crosswords and lateral thinking problems.

2.5. Statistical analysis

The primary aim of the study was to estimate feasibility parameters, estimate the variability in outcome between treatment groups and effect sizes from the group differences in mean outcome. Statistical analysis was based on the intention to treat principle with participants

analysed according to the treatment group to which they were assigned. Treatment differences were calculated at both end of intervention and follow-up, with end-of-intervention being the main timepoint of interest.

2.5.1. Primary outcomes

Participation rate was assessed as the proportion of participants who completed each survey. Adherence was assessed as the proportion of participants who completed each weekly task. Whilst the focus was on participation in the intervention group, comparisons to the control group were made using chi-square. Ninety-five percent confidence intervals around the proportions were obtained using the proportion command in STATA, and reflects with 95% confidence where the population proportion lies (StataCorp, 2013). Mean treatment acceptability score, measured during the end of intervention survey, was used to measure treatment acceptability.

The Sleep Condition Indicator was a primary outcome of interest. Variability of the treatment difference was calculated via the pooled standard deviation across group for change in score from baseline to end of treatment. This was then used to estimate effect sizes (Cohen's d, plus its 95% confidence interval) of the between group difference in insomnia symptoms change (from baseline to end of intervention) (the primary outcome). An independent samples t-test was used to test statistical significance. Cohen's U_3, percentage overlap of change scores between the two groups, and the probability of superiority were calculated to facilitate interpretation of the effect size (see study protocol for more information, Denis et al., 2017).

To investigate whether these changes were driven mainly by participants with the poorest baseline sleep problems, we also looked at effect size separately for those whose baseline insomnia symptoms were either above or below the insomnia threshold. Additionally, we examined the change in percentage of participants who went from meeting threshold criteria for insomnia to not. The percentage of participants whose change scores were equal or higher than the sleep condition indicator's reliably change index were also calculated. Statistical significance was assessed using chi-square.

Finally, the relative effects of group, time, and the group * time interaction were assessed using a generalized estimating equation. Insomnia symptoms at mid- and end of intervention were entered as dependent variables. Group (control group set as reference), time (mid intervention set as reference), and the group * time interaction were entered as predictors. Baseline insomnia symptoms and age were entered as co-variates.

2.5.2. Secondary and exploratory outcomes

Changes in associated variables were assessed using generalized estimating equation (GEE) models. For each variable, a GEE model was fitted using the associated variable score assessed at mid- and end-of-intervention as dependent variables. Predictors were group (control group set as reference), time (mid-intervention set as reference), and the group * time interaction. Baseline score on the associated variable, baseline insomnia symptoms, and age were entered as co-variates. A total of 8 GEE models were run, using the following dependent variables: anxiety symptoms, depression symptoms, ADHD symptoms, experiences of paranoia, experiences of hallucinations, cognitive disorganization, positive mental health, and life stress. ADHD and measures of paranoia, hallucinations, and cognitive disorganization were not assessed mid-assessment. As such, for these measures, no main effect of time or group * time interaction could be calculated. Co-efficient size and 95% CI were taken as the measure of effect size.

Moderators of treatment outcome were assessed using multiple regression models. For each potential moderator, a regression model with insomnia symptoms at the end of assessment was included as the dependent variable, and group, moderator, and group * moderation interaction as predictors. Age and baseline insomnia symptoms were controlled in each model. The standardized co-efficient and 95% CI for each group * moderator interaction was taken as an estimate of the moderator's effect size. A total of 10 regressions were performed, for each of the following potential baseline moderators (insomnia symptoms, anxiety symptoms, depression symptoms, ADHD symptoms, experiences of paranoia, experiences of hallucinations, cognitive disorganization, positive mental health, life stress, and exposure to potential threatening life events).

Given that individual moderators often show a small effect size, we also calculated a combined moderator term using the method described by Kraemer (Kraemer, 2013; Wallace,

Frank, & Kraemer, 2013). A regression of end of assessment insomnia symptoms on group, the combined moderator, and the group * combined moderator interaction was run. This analysis was done after the individual moderator analyses. Bootstrapped mediated regression models (5000 repetitions) were used to test for mediators of treatment outcome. For each potential mediator, insomnia symptoms at the end of treatment was used as the dependent variable. The predictor variable was group, with age, baseline mediator score, and baseline insomnia symptoms as co-variates. Six models were run, with the following variables (all measured at the end-of-intervention assessment) as mediators: general sleep quality, cognitions about sleep, somatic pre-sleep arousal, cognitive pre-sleep arousal, trauma-related sleep disturbances, and chronotype.

All secondary and exploratory analyses were performed using the full sample. As we were primarily interested in effect size, reported *p* values are uncorrected for multiple comparisons.

2.5.3. Missing data

Given there was participant drop-out through the trial we used a 2-step process which assumed data were missing at random. A binary variable was created to indicate whether data were missing or not. Predictors of missing data at the end of intervention assessment were then examined using logistic regression. All baseline measures and treatment acceptability at mid-intervention were investigated as potential predictors in the same regression model. In the second step, multiple imputation was used to estimate missing data, carried out in STATA using an imputation-chained-equations algorithm. Significant predictors of missingness were included in the imputation model. A total of 25 imputed datasets were created. All variables that had missing data of <30% and were deemed missing completely at random or missing at random were entered into the multiple imputation algorithm.

2.6. Deviations from protocol

In our original protocol, we had planned to perform GEE models to assess predictors of treatment outcome (Denis et al., 2017). However, on the advice of statisticians involved in the analyses, we changed these analyses to multiple regressions with SCI scores at a single timepoint (end of intervention). This change was made to simplify the interpretations of

results and to increase statistical power to address our questions. Additionally, we originally planned to analyze data from the 6-month follow-up assessment. However, due to a high drop-out at that assessment, we focused our analyses on the end-of-intervention assessment instead. For transparency, analyses focusing on the 6-month follow-up are presented as **Supplementary results**. Finally, we conducted post-hoc sub-group analyses on our primary outcomes separately for participants who were either above or below the SCI threshold for insomnia at baseline. These analyses were not initially planned but were added to allow us to check whether any changes identified might be driven by those with insomnia reporting a reduction in symptoms over time.

3. Results

Participant characteristics are shown in **Table 1.** Descriptive statistics for variables at each wave are shown in **Table 2.** At baseline insomnia symptom scores ranged from 3-31, meaning that all participants endorsed some sleep complaints. Lower positive mental health and higher perceived stress at baseline were found to significantly predict missing data at the end of the intervention ($p < .05$), and were included in the imputation model.

3.1. Primary outcomes

Participation and adherence rates at each wave are shown in **Table 3**. For participation rates, there were no significant differences between groups at any of the waves (all χ^2 (1) > 1.85, p > .140). With regards to adherence, significantly more participants in the control group completed all six weekly sessions (puzzles) than in the intervention group (CBT-I; χ^2 (1) > 4.82, p = .028). For both participation and adherence, there were no differences between those above the threshold for insomnia, and those below it (all *ps* > .342). Mean treatment acceptability score for the CBT-I program was 33.61 (SD = 4.82, min = 22, max = 42). Responses to each item are shown in **Figure 2**. There were no differences in treatment acceptability between those who fell above or below the threshold for insomnia at baseline (M_{diff} = 0.32, SD = 5.30, p = .829, d = 0.06).

Insomnia symptoms scores at each assessment are shown in **Figure 3**, and a detailed summary of the effect of group on insomnia symptoms are displayed in **Table 4.** At the end of the intervention, the CBT-I group showed a larger improvement in insomnia symptoms

than the control group; t (140) = 2.51, p = .013, d = 0.42). Histograms showing variation in SCI change scores for the two groups are shown in **Supplementary Figure S1**). Additional effect size measures (Cohen's U_3, percent overlap, and probability of superiority) are shown in **Table 4**. We then looked at the effect of group separately for participants that either did not (n = 97) or did (n = 45) meet the threshold requirement for insomnia at baseline. For those that did *not* meet the criteria for insomnia, a similar group effect was found to when looking at the whole sample (t (95) = 2.49, p = .015, d = 0.51). The effect size was smaller for participants who did meet the criteria for insomnia at baseline (t (43) = 0.68, p = .497, d = 0.21).

We then looked within each group (CBT-I or control) and compared change scores in those who did and did not meet insomnia criteria at baseline. Within the CBT-I group, there was a non-significant higher change in insomnia symptoms for those that did meet insomnia criteria at baseline (t (66) = 1.73, p = .087, d = 0.44). Within the control group, those who met insomnia criteria at baseline showed a significantly higher change in insomnia symptoms than those that did not meet criteria (t (72) = 2.26, p = .027, d = 0.59).

Additionally, within the CBT-I group, there was a significant reduction in the percentage of participants meeting the criteria for insomnia at the end of the intervention (36%), compared to baseline (17%); χ^2 (1) = 6.00, p = .013. This change within the control group was not significant (end of intervention = 18%, baseline = 28%,; χ^2 (1) = 2.69, p = .101). The between-group change was not significant – although there was a trend (χ^2 (1) = 3.77, p = .052). A significantly higher percentage of participants in the CBT-I group had change scores that met or exceeded the sleep conditional indicator's reliable change index (35%) compared to the control group (17%); χ^2 (1) = 6.23, p = .013.

A GEE model predicting change in SCI score from group, time, and group * time interaction showed a significant main effect of group, with a larger change in the intervention group compared to the control group (β = 1.94, 95% CI = 0.42 – 3.47, p = .013). Full model results are shown in **Supplementary Table 2.**

Analyses were repeated for the 6-month follow-up assessment (see **Supplementary results**), with a similar pattern of findings being obtained. The between-group effect size at

6-month follow up was d = 0.44, with again the effect being larger in those who did not meet insomnia criteria at baseline (d = 0.55) compared to those who did (d = 0.11).

3.2. Secondary and exploratory outcomes

A set of GEE models were performed to examine whether group allocation predicted change in associated variables. Coefficients and 95% CI for the effect of group on the change in outcome variable are shown in **Figure 4A,** and full statistical information is shown in Supplementary Table 3. Anxiety symptoms (β = -2.57, 95 % CI = -4.97 – -0.25, p = .030), experiences of paranoia (β = -1.68, 95% CI = -3.31 – -0.07, p = .041), and perceived life stress (β = -2.07, 95% CI = -3.88 – -0.26, p = .025) all showed greater reductions in the intervention group compared to the control. When the 6-month follow up time point was also included, results were similar, but experiences of hallucinations showed a reduction in the intervention group, as opposed to paranoia (**Supplementary Figure S2A**).

Regression coefficients for each group * moderator interaction are shown in **Figure 4B**, with full statistical information in **Supplementary Table 4**. None of the baseline variables were moderators of treatment outcome. At the 6-month follow-up, results were largely unchanged, however the combined moderator had 95% confidence intervals that did not cross zero (**Supplementary Figure S2B**).

To examine whether the effect of CBT-I on insomnia symptoms at the end of intervention was mediated by other sleep-related variables, a set of mediated regression models were run. Coefficients and 95% CIs for the indirect effect of each potential mediator on insomnia symptoms at the end of intervention are shown in **Figure 4C**. **Supplementary Table 5** provides the full statistical information. Dysfunctional beliefs about sleep (β = 1.41, 95% CI = 0.68– 2.39), and somatic pre-sleep arousal (β = 1.20, 95% CI = 0.47 – 2.31) showed indirect effects. At the six-month follow-up, only dysfunctional beliefs about sleep appeared to mediate the relationship (**Supplementary Figure S2C**).

4. Discussion

This study aimed to assess the feasibility of administering CBT-I in a sample not selected for insomnia disorder, and establish effect sizes for changes in insomnia symptoms following a digital CBT-I intervention.

We found no significant differences in terms of participation rates between the two groups, although adherence to the tasks was significantly higher in the control group. In addition, there were no differences in terms of participation rate for those who met criteria for insomnia at baseline compared to those who did not. These results suggest that individuals who only endorse mild sleep complaints are willing to take part in a 6-week CBT-I program, and are no more likely to drop out than those with more severe complaints. They did however show lower adherence to the weekly tasks than the control group. Relatedly, participants rated CBT-I as highly acceptable. Again, no differences were found between those who either met or did not meet criteria for insomnia at baseline. Perhaps most interestingly, a large majority rated the treatment as being effective or highly effective. This is important because it suggests that in our sample of participants (including those with mild sleep complaints), they still believed that CBT-I was effective in improving their sleep.

The effect size for the between-group change in insomnia symptoms at the end of the intervention was smaller than that found in studies using insomnia patients (Espie et al., 2012). Additional metrics, such as Cohen's U_3 and the probability of superiority further illustrated the relatively small effect size obtained. Despite this, changes were still significantly larger in the CBT-I group than the control group, suggesting that CBT-I is effective at improving insomnia symptoms in a sample not selected for insomnia disorder. Additionally, for those that did not meet the threshold for insomnia disorder, we found a clear benefit of CBT-I on insomnia symptoms, meaning that improvement was not driven solely by those with the worst insomnia symptoms at baseline. An important implication of this finding is that even for those who only endorse sub-threshold symptoms of insomnia, CBT-I can still lead to improvements.

We were surprised to discover that those who met threshold criteria for insomnia did not show greater improvements after CBT-I compared to the control task as the effectiveness of CBT-I for those with insomnia has been previously established (Seyffert et al., 2016). The reason for this finding is unclear. Whilst these participants did show a large improvement within the CBT-I group, they also showed a large improvement in the control group. This led

to a between-group effect size being approximately half that of those who did not meet the threshold criteria for insomnia at baseline. This also contradicts previous work finding lower insomnia severity to be associated with less successful treatment outcome (Yeung, Chung, Ho, & Ho, 2015). One possibility is that the insomnia symptoms reported at the beginning of the study did not necessarily reflect long-term issues (the SCI asks for symptoms over the past month), and hence there could have been a regression towards the mean over time in both the CBT-I and puzzles group. It is also noteworthy that the subgroup analyses were not pre-planned, and power was low when looking specifically at those with insomnia. This meant that statistical power may not have been adequate to detect the small effects found here.

Even if CBT-I can improve insomnia symptoms in individuals whose symptoms are subthreshold, there is the practical question of why this is useful. If an individual thinks they suffer from sub-threshold insomnia, why should they receive CBT-I? Previous studies have shown that sub-threshold symptoms can have a negative effect on quality of life. As such, efforts to alleviate even minor complaints might result in a number of important benefits (Léger et al., 2001). Other studies of CBT-I have shown that it can bring numerous additional benefits, such as reduced anxiety, paranoia, and overall improvements in sleep-related quality of life and psychological well-being (Espie et al., 2019; Freeman et al., 2017; Ye et al., 2015). Exploratory analyses in this study suggest that these additional benefits also extend to our sub-threshold insomnia symptom sample. We found greater reductions in anxiety symptoms, experiences of paranoia, and perceived life stress after CBT-I compared to the control group. These effects were obtained after controlling for baseline insomnia symptoms. This suggests that additional, non-insomnia, benefits of CBT-I can be gained even when insomnia symptoms themselves are sub-threshold. Finally, the CBT-I program may offer novel advice about how to improve insomnia symptoms in those whose symptoms are low. The tips offered in the CBT-I programme may be beneficial to these individuals. For example, we found that dysfunctional beliefs about sleep mediated the change in insomnia symptoms. This suggests that participants might have learned some important information about sleep that helped lead to overall improvements. Our finding that CBT-I is effective in reducing sub-threshold symptoms suggests that a brief intervention could act to prevent full insomnia disorder, by targeting symptoms early whilst they are still mild. Whilst our results speak to the idea that CBT-I is effective in treating symptoms when the severity is low, we did not examine the potential effects of symptom chronicity. Future research should

investigate whether CBT-I can improve sub-threshold symptoms when symptoms have been present for a long period of time, or if it is most effective when administered soon after symptom onset.

A number of limitations should be considered. Our participants were female students of psychology, and it is conceivable that these participants may react differently to a psychological therapy than those from other groups and may rate the effectiveness of the therapy differently. The study only included females, so possible sex differences between females and males could not be investigated. Additionally, the control task may not have been ideally balanced to match the CBT-I. For instance, CBT-I required a daily sleep diary to be completed and the weekly sessions required participants to engage with the program. For the online puzzles, although they were matched for length to the weekly CBT-I sessions, there was not a daily task and it may have been easier for participants to complete them without as much engagement and attention. This would mean that those in the puzzles group had a lower participant burden as compared to those in the CBT-I group. This difference may potentially account for the lower adherence found in the CBT-I group. It is worth pointing out however that adherence in the present study was similar to other studies using the same CBT-I program (Freeman et al, 2019; Christensen et al, 2016). Finally, we did not collect any objective measurements of sleep (e.g. actigraphy or polysomnography). Whether changes in objective sleep parameters (e.g. sleep onset latency, total sleep time, sleep efficiency) can be seen following CBT-I for sub-threshold insomnia is an exciting area for future research.

A major reason for performing this feasibility study was to provide preliminary data that would be useful in the design of a large-scale study examining genetic and non-genetic predictors of response to CBT-I. To this end, the current study has given the following information about a sample not specifically selected for insomnia: 1 – CBT-I leads to greater improvements in insomnia symptoms compared to a control group, with a small-to-medium effect size; 2 – Additional, non-insomnia related improvements also occur, especially with regards to anxiety symptoms, experiences of paranoia, and perceived life stress; 3 – There was no clear evidence of specific moderators, though at 6-month follow-up the combined moderator 95% confidence intervals showed a non-zero crossing. This suggests a combined moderator may be important to assess in future work to establish whether this is a robust

finding, and; 4 – Preliminary evidence for mediation was found with regards to dysfunctional beliefs about sleep, and somatic pre-sleep arousal.

In conclusion, this study has shown that participants who do not meet criteria for insomnia disorder still show a significant benefit of CBT-I, both in terms of insomnia symptoms themselves, and non-insomnia complaints including anxiety, paranoia, and perceived stress. Future work can utilize the results of this study to design a large-scale twin study of predicators of treatment outcome for CBT-I, looking at both genetic and non-genetic predictors.

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Declaration of interest

CAE is the cofounder and CMO of Big Health Ltd, the company behind the digital CBT-I programme evaluated in this study. CAE also holds shares in Big Health Ltd. AMG has provided guidance and educational content for a freely available educational website focused on infant sleep. This website is partially supported by Johnson and Johnson, but they do not have any influence over content and do not advertise on it. AMG also contributes to BBC Focus Magazine and regularly contributes to media reports on sleep. She is author of popular science books Nodding Off (Bloomsbury, 2018) and The Sleepy Pebble and Other Stories (Flying Eye Books, 2019). She has delivered a talk about sleep for business.

References

Andersson, E., Crowley, J. J., Lindefors, N., Ljótsson, B., Hedman-Lagerlöf, E., Boberg, J., … Rück, C. (2019). Genetics of response to cognitive behavior therapy in adults with major depression: a preliminary report. *Molecular Psychiatry*, *24*(4), 484–490. doi:10.1038/s41380-018-0289-9

Andersson, E., Rück, C., Lavebratt, C., Hedman, E., Schalling, M., Lindefors, N., … Furmark, T. (2013). Genetic polymorphisms in monoamine systems and outcome of cognitive behavior therapy for social anxiety disorder. *Plos One*, *8*(11), e79015. doi:10.1371/journal.pone.0079015

Angold, A., Costello, E. J., Messer, S. C., Pickles, A., Winder, F., & Silver, D. (1995). Development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *International Journal of Methods in Psychiatric Research*, *5*(4), 237–249.

Beevers, C. G., & McGeary, J. E. (2012). Therapygenetics: moving towards personalized psychotherapy treatment. *Trends in Cognitive Sciences*, *16*(1), 11–12. doi:10.1016/j.tics.2011.11.004

Bostock, S., Luik, A. I., & Espie, C. A. (2016). Sleep and productivity benefits of digital cognitive behavioral therapy for insomnia: A randomized controlled trial conducted in the workplace environment. *Journal of Occupational and Environmental Medicine*, *58*(7), 683–689. doi:10.1097/JOM.0000000000000778

Brugha, T., Bebbington, P., Tennant, C., & Hurry, J. (1985). The List of Threatening Experiences: a subset of 12 life event categories with considerable long-term contextual threat. *Psychological Medicine*, *15*(1), 189–194. doi:10.1017/S003329170002105X

Bryant, R. A., Felmingham, K. L., Falconer, E. M., Pe Benito, L., Dobson-Stone, C., Pierce, K. D., & Schofield, P. R. (2010). Preliminary evidence of the short allele of the serotonin transporter gene predicting poor response to cognitive behavior therapy in posttraumatic stress disorder. *Biological Psychiatry*, *67*(12), 1217–1219. doi:10.1016/j.biopsych.2010.03.016

Buysse, D. J., Reynolds, C. F., Monk, T. H., Berman, S. R., & Kupfer, D. J. (1989). The Pittsburgh Sleep Quality Index (PSQI): A new instrument for psychiatric practice and research. *Psychiatry Research*, *28*(2), 193–213.

Cheng, P., Luik, A. I., Fellman-Couture, C., Peterson, E., Joseph, C. L. M., Tallent, G., … Drake, C. L. (2018). Efficacy of digital CBT for insomnia to reduce depression across demographic groups: a randomized trial. *Psychological Medicine*, 1–10. doi:10.1017/S0033291718001113

Coddington, R. D. (1983). Life events scale for children and adolescents: Measuring the stressfulness of a child's environment. In *Stress in Childhood*. New York: AMS Press.

Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior*, *24*(4), 385–396. doi:10.2307/2136404

Denis, D., Eley, T. C., Rijsdijk, F., Zavos, H. M. S., Keers, R., Espie, C. A., … Gregory, A. M. (2017). Sleep Treatment Outcome Predictors (STOP) Pilot Study: a protocol for a randomised controlled trial examining predictors of change of insomnia symptoms and associated traits following cognitive-behavioural therapy for insomnia in an unselected sample. *BMJ Open*, *7*(11), e017177. doi:10.1136/bmjopen-2017-017177

Espie, C.A, Inglis, S. J., Harvey, L., & Tessier, S. (2000). Insomniacs' attributions. psychometric properties of the Dysfunctional Beliefs and Attitudes about Sleep Scale and the Sleep Disturbance Questionnaire. *Journal of Psychosomatic Research*, *48*(2), 141–148.

Espie, C.A, Emsley, R., Kyle, S. D., Gordon, C., Drake, C. L., Siriwardena, A. N., … Luik, A. I. (2019). Effect of Digital Cognitive Behavioral Therapy for Insomnia on Health, Psychological Well-being, and Sleep-Related Quality of Life: A Randomized Clinical Trial. *JAMA Psychiatry*, *76*(1), 21–30. doi:10.1001/jamapsychiatry.2018.2745

Espie, C.A, Farias Machado, P., Carl, J. R., Kyle, S. D., Cape, J., Siriwardena, A. N., & Luik, A. I. (2018). The Sleep Condition Indicator: reference values derived from a sample of 200 000 adults. *Journal of Sleep Research*, *27*(3), e12643. doi:10.1111/jsr.12643

Espie, C.A, Kyle, S. D., Hames, P., Gardani, M., Fleming, L., & Cape, J. (2014). The Sleep Condition Indicator: a clinical screening tool to evaluate insomnia disorder. *BMJ Open*, *4*(3), e004183. doi:10.1136/bmjopen-2013-004183

Espie, C.A, Kyle, S. D., Williams, C., Ong, J. C., Douglas, N. J., Hames, P., & Brown, J. S. L. (2012). A randomized, placebo-controlled trial of online cognitive behavioral therapy for chronic insomnia disorder delivered via an automated media-rich web application. *Sleep*, *35*(6), 769–781. doi:10.5665/sleep.1872

Freeman, D., Sheaves, B., Goodwin, G. M., Yu, L.-M., Nickless, A., Harrison, P. J., … Espie, C. A. (2017). The effects of improving sleep on mental health (OASIS): a randomised controlled trial with mediation analysis. *The Lancet. Psychiatry*, *4*(10), 749–758. doi:10.1016/S2215-0366(17)30328-0

Germain, A., Hall, M., Krakow, B., Katherine Shear, M., & Buysse, D. J. (2005). A brief sleep scale for Posttraumatic Stress Disorder: Pittsburgh Sleep Quality Index Addendum for PTSD. *Journal of Anxiety Disorders*, *19*(2), 233–244. doi:10.1016/j.janxdis.2004.02.001

Gregory, A. M., Agnew-Blais, J. C., Matthews, T., Moffitt, T. E., & Arseneault, L. (2017). ADHD and sleep quality: longitudinal analyses from childhood to early adulthood in a twin cohort. *Journal of Clinical Child and Adolescent Psychology*, *46*(2), 284–294. doi:10.1080/15374416.2016.1183499

Hudson, J. L., Lester, K. J., Lewis, C. M., Tropeano, M., Creswell, C., Collier, D. A., … Eley, T. C. (2013). Predicting outcomes following cognitive behaviour therapy in child anxiety disorders: the influence of genetic, demographic and clinical information. *Journal of Child Psychology and Psychiatry, and Allied Disciplines*, *54*(10), 1086–1094. doi:10.1111/jcpp.12092

Hunsley, J. (1992). Development of the treatment acceptability questionnaire. *Journal of Psychopathology and Behavioral Assessment*, *14*(1), 55–64. doi:10.1007/BF00960091

Kessler, R. C., Berglund, P. A., Coulouvrat, C., Hajak, G., Roth, T., Shahly, V., … Walsh, J. K. (2011). Insomnia and the performance of US workers: results from the America insomnia survey. *Sleep*, *34*(9), 1161–1171. doi:10.5665/SLEEP.1230

LeBlanc, M., Beaulieu-Bonneau, S., Mérette, C., Savard, J., Ivers, H., & Morin, C. M. (2007). Psychological and health-related quality of life factors associated with insomnia in a population-based sample. *Journal of Psychosomatic Research*, *63*(2), 157–166. doi:10.1016/j.jpsychores.2007.03.004

Léger, D., Scheuermaier, K., Philip, P., Paillard, M., & Guilleminault, C. (2001). SF-36: evaluation of quality of life in severe and mild insomniacs compared with good sleepers. *Psychosomatic Medicine*, *63*(1), 49–55.

Lonsdorf, T. B., Rück, C., Bergström, J., Andersson, G., Ohman, A., Lindefors, N., & Schalling, M. (2010). The COMTval158met polymorphism is associated with symptom relief during exposure-based cognitive-behavioral treatment in panic disorder. *BMC Psychiatry*, *10*, 99. doi:10.1186/1471-244X-10-99

Luik, A. I., Kyle, S. D., & Espie, C. A. (2017). Digital Cognitive Behavioral Therapy (dCBT) for Insomnia: a State-of-the-Science Review. *Current Sleep Medicine Reports*, *3*(2),

48–56. doi:10.1007/s40675-017-0065-4
Lukat, J., Margraf, J., Lutz, R., van der Veld, W. M., & Becker, E. S. (2016). Psychometric properties of the Positive Mental Health Scale (PMH-scale). *BMC Psychology*, *4*, 8. doi:10.1186/s40359-016-0111-x
Nicassio, P. M., Mendlowitz, D. R., Fussell, J. J., & Petras, L. (1985). The phenomenology of the pre-sleep state: the development of the pre-sleep arousal scale. *Behaviour Research and Therapy*, *23*(3), 263–271. doi:10.1016/0005-7967(85)90004-X
Qaseem, A., Kansagara, D., Forciea, M. A., Cooke, M., Denberg, T. D., & Clinical Guidelines Committee of the American College of Physicians. (2016). Management of chronic insomnia disorder in adults: A clinical practice guideline from the american college of physicians. *Annals of Internal Medicine*, *165*(2), 125–133. doi:10.7326/M15-2175
Riemann, D., Baglioni, C., Bassetti, C., Bjorvatn, B., Dolenc Groselj, L., Ellis, J. G., … Spiegelhalder, K. (2017). European guideline for the diagnosis and treatment of insomnia. *Journal of Sleep Research*, *26*(6), 675–700. doi:10.1111/jsr.12594
Ritterband, L. M., Thorndike, F. P., Ingersoll, K. S., Lord, H. R., Gonder-Frederick, L., Frederick, C., … Morin, C. M. (2017). Effect of a Web-Based Cognitive Behavior Therapy for Insomnia Intervention With 1-Year Follow-up: A Randomized Clinical Trial. *JAMA Psychiatry*, *74*(1), 68–75. doi:10.1001/jamapsychiatry.2016.3249
Roenneberg, T., Wirz-Justice, A., & Merrow, M. (2003). Life between clocks: daily temporal patterns of human chronotypes. *Journal of Biological Rhythms*, *18*(1), 80–90. doi:10.1177/0748730402239679
Ronald, A., Sieradzka, D., Cardno, A. G., Haworth, C. M. A., McGuire, P., & Freeman, D. (2014). Characterization of psychotic experiences in adolescence using the specific psychotic experiences questionnaire: findings from a study of 5000 16-year-old twins. *Schizophrenia Bulletin*, *40*(4), 868–877. doi:10.1093/schbul/sbt106
Seyffert, M., Lagisetty, P., Landgraf, J., Chopra, V., Pfeiffer, P. N., Conte, M. L., & Rogers, M. A. M. (2016). Internet-Delivered Cognitive Behavioral Therapy to Treat Insomnia: A Systematic Review and Meta-Analysis. *Plos One*, *11*(2), e0149139. doi:10.1371/journal.pone.0149139
Snow, G. (2013). blockrand: Randomization for block random clinical trials (1.3). Computer software, R.
Spielberger, C. D., Gorsuch, R. L., Lushene, R., Vagg, P. R., & Jacobs, G. A. (1983). *Manual for the State-Trait Anxiety Inventory*. Palo Alto, CA: Consulting Psychologists Press.
StataCorp. (2013). *Stata 13 Base Reference Manual*. College Station, TX: StataCorp LP.
Trauer, J. M., Qian, M. Y., Doyle, J. S., Rajaratnam, S. M. W., & Cunnington, D. (2015). Cognitive Behavioral Therapy for Chronic Insomnia: A Systematic Review and Meta-analysis. *Annals of Internal Medicine*, *163*(3), 191–204. doi:10.7326/M14-2841
van Straten, A., van der Zweerde, T., Kleiboer, A., Cuijpers, P., Morin, C. M., & Lancee, J. (2018). Cognitive and behavioral therapies in the treatment of insomnia: A meta-analysis. *Sleep Medicine Reviews*, *38*, 3–16. doi:10.1016/j.smrv.2017.02.001
Ye, Y.-Y., Zhang, Y.-F., Chen, J., Liu, J., Li, X.-J., Liu, Y.-Z., … Jiang, X.-J. (2015). Internet-Based Cognitive Behavioral Therapy for Insomnia (ICBT-i) Improves Comorbid Anxiety and Depression-A Meta-Analysis of Randomized Controlled Trials. *Plos One*, *10*(11), e0142258. doi:10.1371/journal.pone.0142258
Yeung, W.-F., Chung, K.-F., Ho, F. Y.-Y., & Ho, L.-M. (2015). Predictors of dropout from internet-based self-help cognitive behavioral therapy for insomnia. *Behaviour Research and Therapy*, *73*, 19–24. doi:10.1016/j.brat.2015.07.008

Table 1 – Sample characteristics

	Intervention			Control		
	%	M	SD	%	M	SD
Age		19.73	2.94		20.22	5.69
Ethnicity						
Arab	5.41			1.23		
Asian or Asian British	13.52			24.69		
Black or Black British	8.11			6.17		
Mixed ethnicity	8.10			9.87		
Other	6.76			6.17		
White	58.11			50.63		
Education						
At least GCSE (or equivalent)	98.65			100.00		
At least A-level (or equivalent)	98.31			89.09		
Employment						
Student	98.65			98.78		
Working full-time	1.35			0.00		
Working part-time	24.32			21.95		
Parent full-time	2.70			2.44		
On government benefits	1.22			1.35		
Marital status						
Legally separated/divorced	0.00			0.00		
Living with partner	2.44			5.41		
Married	0.00			1.22		
Single	90.24			82.43		
Other	6.10			12.16		
General health						
Excellent	16.05			13.70		
Very good	39.51			30.14		
Good	30.86			38.36		
Fair	12.35			15.07		
Poor	1.23			2.74		
Medication						
Prescription last 6 months (yes)	53.66			59.46		
Over-the-counter last 6 months (yes)	46.34			64.86		
Psychiatric diagnosis						
Lifetime diagnosis mood disorder	20.73			25.68		
Lifetime diagnosis schizophrenia	0			0		

Note. M = Mean, SD = Standard deviation. Numbers for each construct may not sum to 100 due to missing data and possibility of selecting multiple options per construct.

Table 2 – Descriptive statistics for study variables at each wave

Variable	Intervention				Control			
	Baseline M (SD)	Mid-intervention M (SD)	End of intervention M (SD)	Follow-up M (SD)	Baseline M (SD)	Mid-intervention M (SD)	End of intervention M (SD)	Follow-up M (SD)
Insomnia symptoms	19.70 (6.56)	22.50 (7.56)	24.52 (6.84)	24.27 (6.98)	20.22 (6.53)	21.04 (6.79)	22.78 (6.84)	22.02 (6.74)
Anxiety symptoms	49.93 (10.64)	46.68 (11.86)	44.57 (11.20)	45.02 (10.94)	47.53 (9.92)	46.58 (10.26)	45.32 (10.07)	45.92 (11.47)
Depression symptoms	10.09 (6.71)	8.70 (7.20)	6.91 (5.73)	7.09 (6.23)	8.65 (6.27)	8.00 (7.01)	6.51 (5.87)	8.27 (7.33)
ADHD symptoms	21.47 (11.69)	-	16.51 (10.53)	16.91 (11.24)	22.09 (11.62)	-	18.53 (13.33)	17.97 (12.83)
Experiences of paranoia	6.66 (5.76)	-	4.59 (5.77)	4.69 (5.53)	6.43 (5.20)	-	6.07 (5.92)	6.25 (5.66)
Experiences of hallucinations	3.03 (3.71)	-	1.59 (3.24)	1.84 (3.64)	2.02 (2.55)	-	1.31 (2.42)	2.25 (3.28)
Cognitive disorganization	3.19 (1.55)	-	2.65 (1.89)	2.55 (2.03)	3.33 (1.55)	-	3.07 (1.52)	2.43 (1.84)
Positive mental health	26.00 (6.44)	25.39 (7.01)	26.42 (6.18)	26.22 (6.86)	26.64 (5.76)	26.46 (5.69)	27.09 (6.09)	26.58 (6.63)
Perceived life stress	20.14 (6.82)	17.92 (8.24)	16.96 (6.57)	16.60 (7.87)	20.05 (6.12	19.14 (7.56)	17.71 (8.06)	17.95 (8.68)
Threatening events	2.57 (2.31)	0.80 (1.36)	0.63 (1.16)	0.73 (1.27)	2.72 (2.69)	1.08 (1.51)	0.74 (1.37)	1.13 (1.56)
General sleep quality	7.75 (3.39)	7.51 (3.53)	6.12 (3.06)	7.44 (3.78)	8.13 (3.52)	7.51 (3.30)	6.67 (3.44)	7.10 (3.66)
Specific sleep disturbances	3.66 (2.95)	-	2.49 (3.17)	3.27 (3.77)	3.24 (3.17)	-	2.32 (2.61)	3.10 (3.33)
Somatic pre-sleep arousal	13.92 (5.46)	13.53 (5.98)	12.14 (4.15)	13.38 (5.67)	14.02 (5.57)	15.31 (6.29)	13.82 (5.70)	14.05 (6.58)

Cognitive pre-sleep arousal	21.81 (7.40)	19.23 (8.37)	17.59 (7.59)	18.22 (7.45)	21.39 (7.34)	19.63 (8.17)	18.27 (7.54)	20.18 (8.58)
Dysfunctional beliefs about sleep	55.95 (15.83)	22.09 (11.62)	22.09 (11.62)	22.09 (11.62)	52.30 (17.55)	50.39 (17.01)	48.65 (19.95)	22.09 (11.62)
Chronotype	07:44 (01:40)	-	07:47 (01:30)	06:56 (01:43)	07:10 (01:58)	-	07:46 (01:42)	07:50 (01:42)
Treatment acceptability	-	34.12 (4.15)	33.24 (5.56)	-	-	-	-	-

Note. M = Mean, SD = Standard deviation. For all measures, a higher score indicates more symptoms, with the exception of insomnia symptoms, where a higher score indicates lower symptoms.. A higher positive mental health score indicates better positive mental health. Chronotype is estimated based on the midpoint between sleep onset and sleep offset on work free days, corrected for sleep debt accumulated over the work week. A higher treatment acceptability score indicates higher treatment acceptability.

Table 3 – Participations rates and adherence

	Intervention		Control		
	%	95% CI	%	95% CI	sig
Participation					
Mid-intervention assessment	69	59; 77	74	65; 82	.676
End-of-intervention assessment	68	58; 76	78	69; 85	.408
Six-month follow-up	47	36; 55	62	52; 71	.150
Adherence					
Completed all 6 weekly tasks	57	44; 69	83	73; 91	.028

Note. CI = Confidence interval, sig = chi-square statistical significance.

Table 4. Effects of CBT-I on insomnia symptoms

	Intervention			Control			Between group sig		
	M	95% CI		M	95% CI		*p*	*d*	95% CI
Change in insomnia symptoms (full sample)	4.69	3.82; 5.99		2.34	1.00; 3.67		.013	0.42	0.08; 0.75
Change in insomnia symptoms (did not meet insomnia threshold at baseline)	3.84	2.51; 5.16		1.44	0.08; 2.80		.015	0.51	0.10; 0.91
Change in insomnia symptoms (did meet insomnia threshold baseline)	6.16	3.37; 8.94		4.75	1.46; 8.03		.497	0.21	-0.79; 0.38
			Within group sig			Within group sig			
	%	95% CI	*p*	%	95% CI	*p*	*p*		
Change in % meeting insomnia threshold	52	40; 63	.013	34	53; 84	.101	.052		
% of change scores meeting exceeding RCI	35	23; 46	<.001	17	8; 25	.015	.013		
<u>Additional effect size quantifications</u>									
	%								
Cohen's U_3	66								
% overlap of change scores between groups	83								
Probability of superiority	62								

Note. M = Mean, CI = Confidence interval, RCI = Reliable change index. Note that positive change values indicate an improvement (i.e. fewer) in insomnia symptoms.

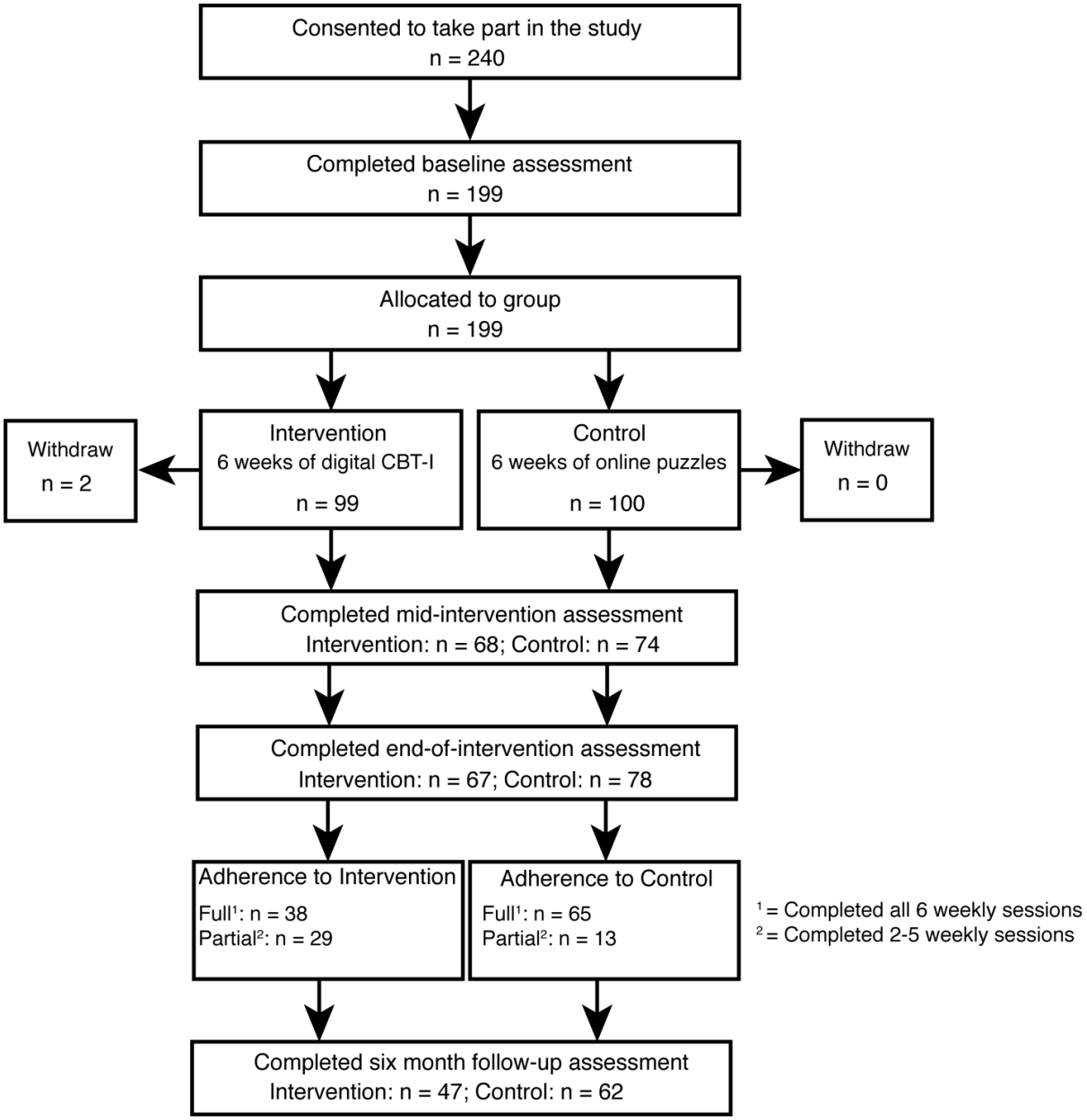

Figure 1. Flow of participants through the study

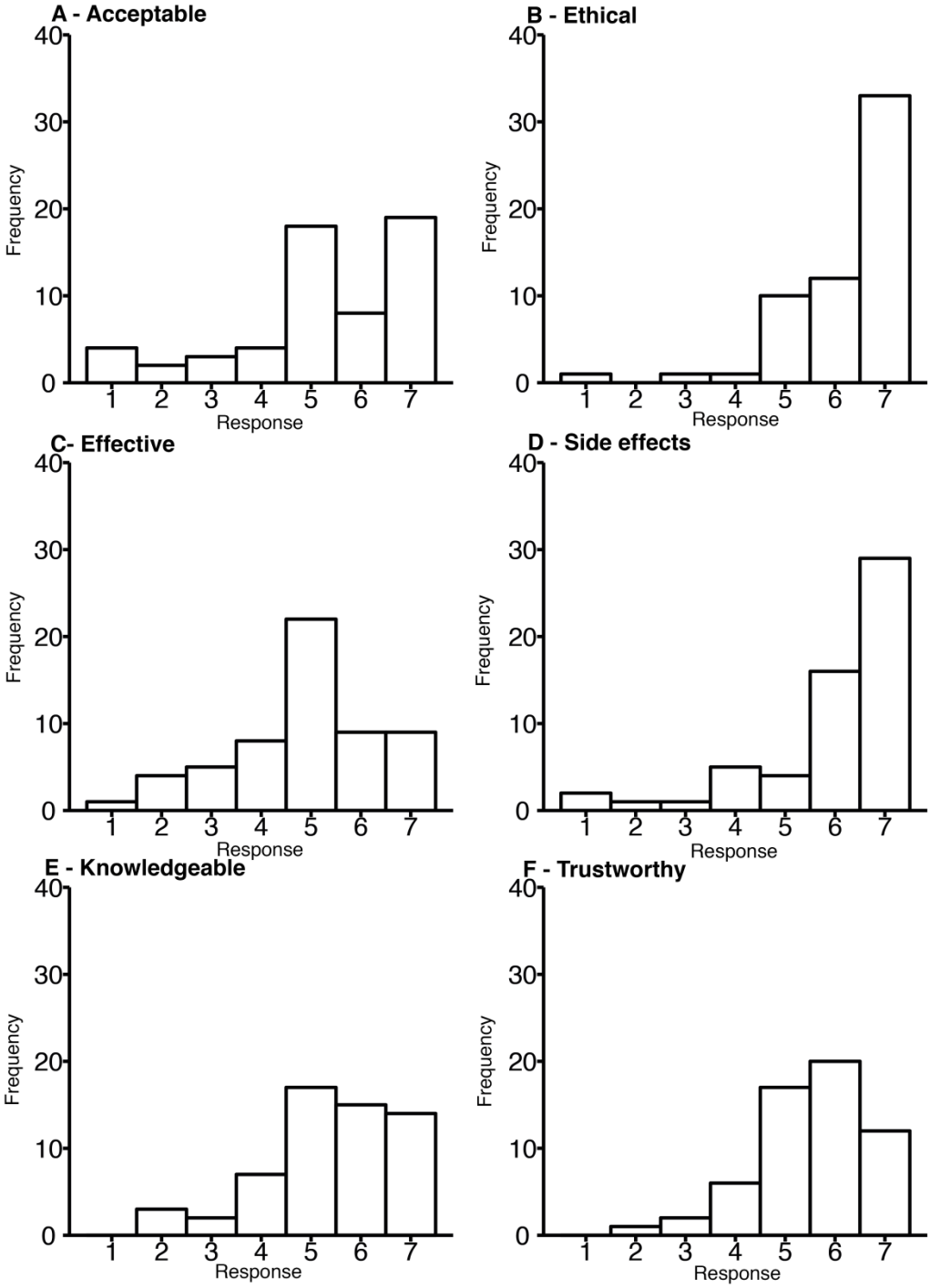

Figure 2. Treatment acceptability. **A** – Acceptable, responses range from 1 (Very unacceptable) to 7 (Very acceptable). **B** – Ethical, responses range from 1 (Unethical) to 7 (Fully ethical). **C** – Effective, responses range from 1 (Very ineffective) to 7 (Very effective). **D** – Side effects, responses range from 1 (Very likely) to 7 (Very unlikely). **E** – Knowledgeable, responses range from 1 (Not knowledgeable) to 7 (Very knowledgeable). **F** – Trustworthy, responses range from 1 (Not trustworthy) to 7 (Very trustworthy).

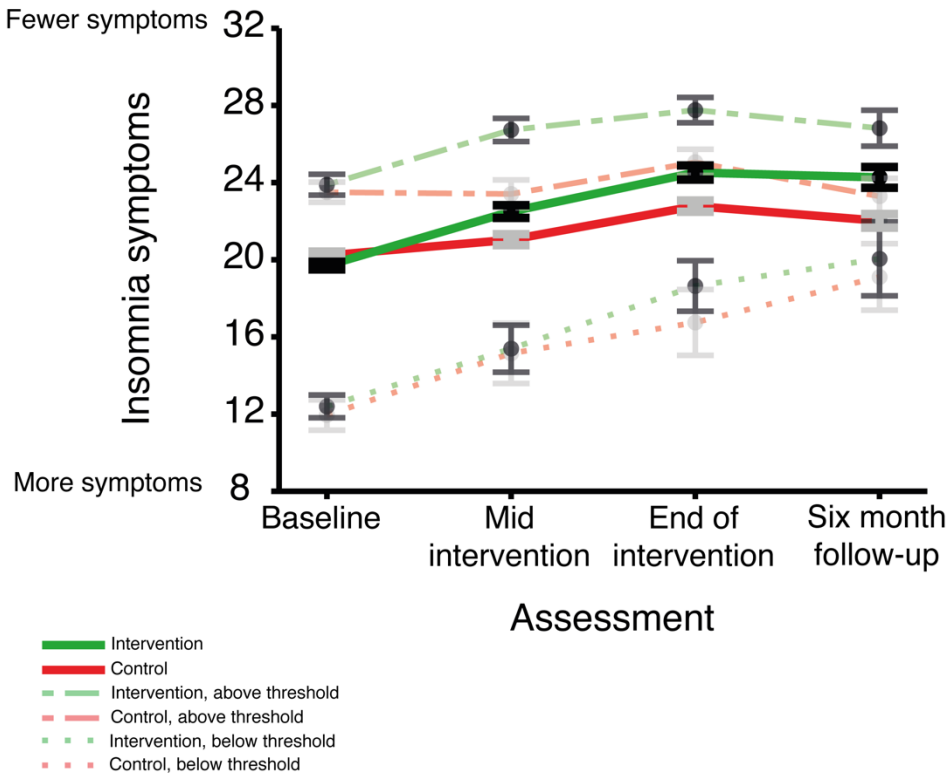

Figure 3. SCI scores at each assessment. Solid lines represent scores across the full sample. Dashed and dotted lines show scores for participants above and below the threshold for insomnia respectively. Error bars indicate the standard error.

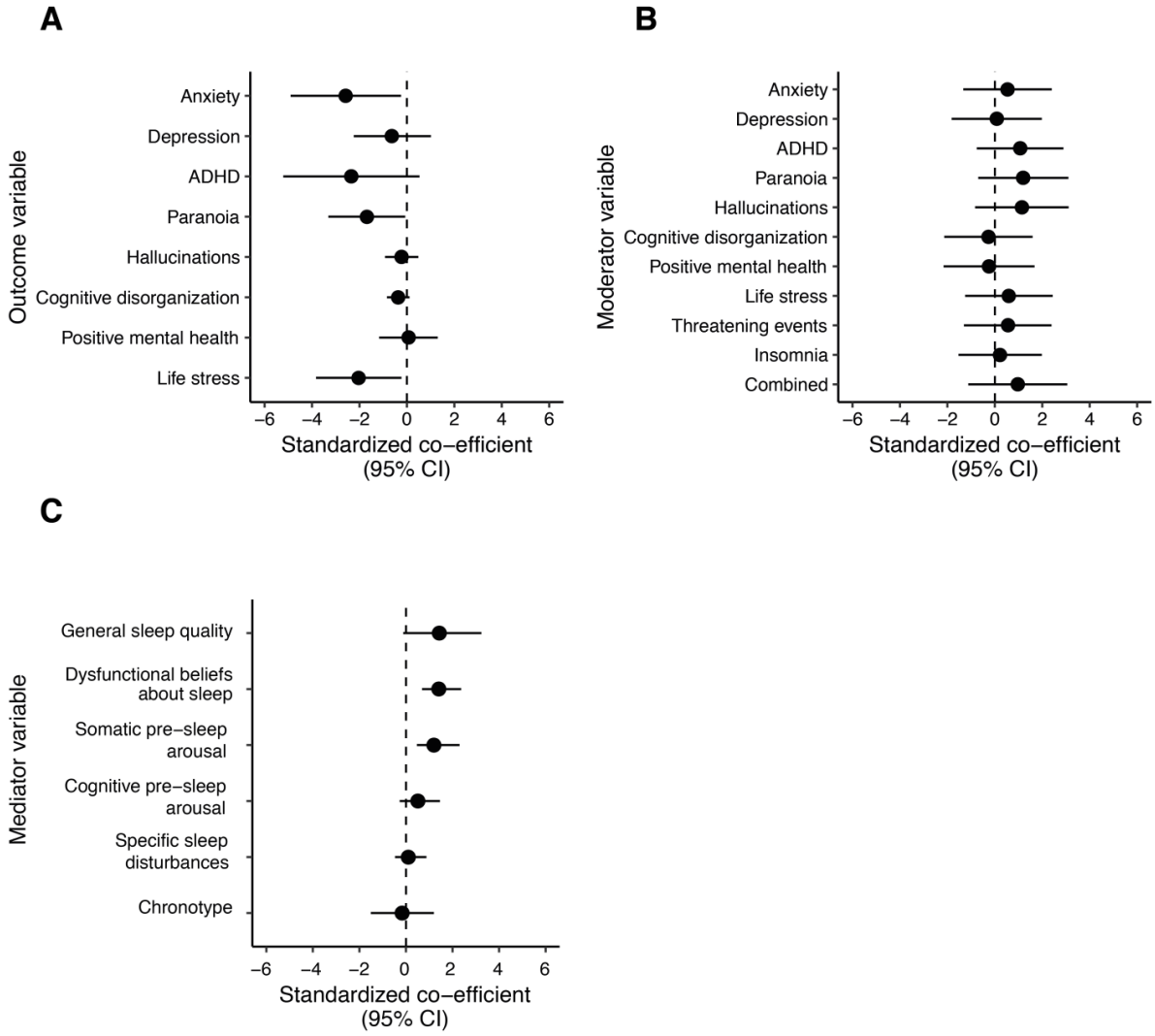

Figure 4. Changes in associated variables, moderation, and mediation results. Control group is always used as the reference. **A** – Changes in associated variables: Standardized coefficients and 95% confidence intervals for the effect of group for each associated variable on the change in insomnia symptoms from baseline to end-of-intervention. **B** – Moderation: Standardized coefficients and 95% confidence intervals for each of the individual group * baseline predictor interactions and the group * combined moderator interaction for each potential moderator of insomnia symptoms at end-of-intervention. **C** – Mediation: Standardized coefficients and bias-corrected 95% confidence intervals for each potential mediator of insomnia symptoms at end of intervention.