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Group-based cognitive behavioral therapy program for improving poor sleep quality and quality of life in people with epilepsy: A pilot study

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ABSTRACT

Sleep difficulties are commonly reported by patients with epilepsy and can have a detrimental impact on overall quality of life. The purpose of this pilot study was to assess the efficacy of a psychotherapeutic approach, namely Cognitive Behavioral Therapy for Insomnia (CBT-I), in improving sleep quality in patients with epilepsy. Twenty outpatients with epilepsy who reported poor sleep quality were randomized to either a control or CBT-I treatment group, which involved four group-based CBT-I sessions, delivered on a weekly basis. In addition to completing a range of standardized measures related to sleep quality and quality of life, participants also monitored their sleep with a self-completed sleep diary over a two-week period, on two separate occasions. Following CBT-I treatment, no between-group difference was found on any sleep or quality of life measure. However, both the treatment and control groups improved on measures of sleep quality, quality of life, sleep hygiene behaviors, and dysfunctional beliefs about sleep. These findings suggest that sleep monitoring alone may have the potential for prompting healthy behavior change in this clinical population.

1. Introduction

Sleep difficulties are overrepresented among patients with epilepsy, compared with the general population [1–5], with insomnia and excessive daytime sleepiness being particularly common in this patient population [1,2,6–8]. These sleep difficulties can negatively impact daily functioning in patients with epilepsy because of the potential of sleep difficulties to result in daytime sleepiness and sleep deprivation, both of which can precipitate seizure activity [5,9]. Sleep difficulties are associated with lower quality of life in patients with epilepsy [1,7,8,10–13] and also lead to a worsening of seizure control and lack of seizure remission [1,7,11,13,14]. It has also been shown that seizure frequency (which is exacerbated by sleep disturbance) is associated with impaired quality of life in the population with epilepsy [12,15,16].

Despite the high prevalence and detrimental impact of sleep difficulties in patients with epilepsy, there is a paucity of research regarding psychological treatment of sleep difficulties in this population. Although pharmacotherapy has been utilized to address sleep difficulties, results have been mixed [17]. Nonpharmacological treatment for sleep disturbance in epilepsy may be preferable to avoid problematic drug interactions, and it has been argued that medication is only to be indicated when nonpharmacological therapy is unsuccessful [18].

Cognitive Behavioral Therapy for Insomnia (CBT-I) has been specifically developed for the treatment of sleep difficulties [19–24]. This treatment has been shown to be efficacious in treating insomnia in a number of different clinical populations such as cancer survivors, individuals with chronic pain, the elderly, and individuals with psychiatric disorders [25–28]. Improvements that result from CBT-I appear to be comparable with pharmacotherapy and are associated with beneficial long-term effects that extend beyond active administration of treatment [29,30]. Cognitive Behavioral Therapy for Insomnia involves sleep restriction (restricts time awake in bed to rebuild the normal homeostatic sleep drive) [31], stimulus control (to reassociate cues, such as a bedroom or bed, with normal sleep) [32], cognitions (i.e., modifying faulty beliefs about sleep) [33–35], sleep hygiene (e.g., correcting maladaptive environmental conditions incompatible with sleep) [36,37], and relaxation (reducing physiological and cognitive arousal) [38,39].

Patients with epilepsy may be ideal CBT-I candidates for a number of reasons. Those who report sleep difficulties display behaviors that are identified to perpetuate poor sleep, such as a preference for evening activity, delays in sleep timing (i.e., later than usual bedtime), potential worry about sleep [5,11,40–42], and inadequate sleep hygiene (e.g., napping and consuming caffeine in the evening) [40]. Moreover, specific CBT-I techniques could arguably be a part of standard care in patients with epilepsy [8,11,43–47]. Although CBT has been shown to

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improve quality of life in patients with epilepsy [48–54], its effect on sleep difficulties in this population remains unknown.

Given that patients with epilepsy exhibit factors that perpetuate sleep disturbance and the success of CBT-I in other populations, it seems feasible to apply CBT-I to this clinical population. The successful application of CBT-I could carry significant clinical implications as it may ultimately help to establish an evidence-based psychotherapeutic treatment for sleep disturbances in this population. This would be beneficial and important for a number of reasons such as a reduced reliance on sleep medications and overall improved quality of life.

This was a single-blinded, randomized controlled pilot study designed to evaluate the preliminary feasibility and efficacy of CBT-I in people with epilepsy. The primary outcome was quality of sleep as measured by a standardized sleep questionnaire. The secondary outcomes were overall patient quality of life and excessive daytime sleepiness as measured by standardized questionnaires only. It was hypothesized that CBT-I would be associated with improvements in amount of sleep, sleep quality, quality of life, and excessive daytime sleepiness compared with a control group. An additional exploratory analysis was conducted to determine which sleep parameters (sleep quality, dysfunctional beliefs about sleep, total sleep time [TST], and time spent in bed) would predict overall quality of life following CBT-I treatment.

2. Materials and methods

2.1. Participants

A total of 20 patients with epilepsy participated in the study. All participants met the following inclusion criteria: i) diagnosis of epilepsy, ii) fluency in English, iii) aged 18–65 years, and iv) poor sleep quality (evidenced by a score greater than five on the Pittsburgh Sleep Quality Index (PSQI) [55]). Treating neurologists ensured that patients met International League Against Epilepsy (ILAE) criteria for diagnosis of epilepsy [56].

Participants were recruited from a tertiary epilepsy clinic at Royal Prince Alfred Hospital (RPAH) in Camperdown, Sydney. The sleep study was advertised by their treating neurologist or neuropsychologist. Additionally, the patient database of the RPAH seizure clinic was searched for appropriate participants, and they were contacted by researchers and invited to take part in the study. This study was approved by the Ethics Review Committee (RPAH zone) of the Sydney Local Health District.

2.2. Materials

2.2.1. Sleep measures

2.2.1.1. Pittsburgh Sleep Quality Index (PSQI). The PSQI [55] is a valid and reliable 19-item self-rated questionnaire that assesses sleep quality and complaints over a one-month period. The following seven component scores are generated: subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medication, and daytime dysfunction. The sum of scores for the seven components generates one global score. Scores range from 0 to 21 with higher scores corresponding to poorer sleep quality. A global PSQI score of >5 is indicative of poor sleep quality. This score indicates that a subject is having severe difficulties in at least two areas or moderate difficulties in more than three areas.

2.2.1.2. Epworth Sleepiness Scale (ESS). The ESS [57,58] is a valid and reliable 8-item, self-administered questionnaire designed to measure the general level of daytime sleepiness in adults across a range of everyday situations. Each item is scored on a 4-point severity scale ranging from 0 to 3 (0 = would never doze, 1 = slight chance of dozing, 2 = moderate chance of dozing, and 3 = high chance of dozing). Subscores are added to provide a total score. The total score ranges from 0 to 24, with scores greater than 10 in the clinical range of severity.

2.2.1.3. Consensus Sleep Diary (CSD). The CSD [59] was used to monitor participants' sleep. Participants were required to complete the CSD daily on final awakening. This sleep diary allowed us to calculate TST, time in bed (TIB), sleep onset latency, sleep efficiency, and wake after sleep onset.

2.2.2. Quality of life and mood

2.2.2.1. Quality of Life in Epilepsy Inventory (QOLIE-31). The QOLIE-31 [60] is a valid and reliable 31-item self-report questionnaire. It contains seven multi-item scales that assess emotional well-being, social functioning, energy/fatigue, cognitive functioning, seizure worry, medication effects, and overall quality of life. The subscales are grouped into two factors, emotional/psychological effects (seizure worry, overall quality of life, emotional well-being, energy/fatigue scales) and medical/social effects (medication effects, work-driving social limits, cognitive function subscales).

2.2.2.2. Neurological Disorders Depression Inventory for Epilepsy (NDDI-E). The NDDI-E [61] is a 6-item questionnaire validated to screen for depression in people with epilepsy. Neurological Disorders Depression Inventory for Epilepsy scores above 15 are considered positive for depression.

2.2.3. Cognitive Behavioral Therapy for Insomnia specific measures

2.2.3.1. Dysfunctional Beliefs and Attitudes about Sleep Scale-16 (DBAS-16). The DBAS-16 [62] is an abbreviated version of the original DBAS [33,63]. The DBAS-16 is a valid and reliable 16-item self-reported questionnaire used to measure an individual's a) perceived consequences of insomnia, b) worry and helplessness about insomnia, c) sleep expectations, and d) beliefs about medications. A higher score reflects greater dysfunctional beliefs about sleep.

2.2.3.2. Sleep Hygiene Index (SHI). The SHI [64,65] is a valid and reliable 13-item scale assessing the practice of sleep hygiene behaviors with an inverse 5-point Likert scale (0 = never, 4 = always). Total scores range from 0 to 52, with a higher score representing poorer sleep hygiene.

2.2.4. Intervention

Because of time restrictions, the study implemented a condensed version of the CBT-I treatment detailed in the manual by Morin and Espie [34]. The treatment consisted of four, 2-h sessions delivered weekly in a group format. The first session delivered psychoeducation on sleep difficulties (e.g., information on circadian rhythms, insomnia, and sleep difficulties in epilepsy) and introduced sleep scheduling, which included stimulus control therapy and sleep restriction. Sleep restriction involves limiting time spent in bed to a specific period that should be used for sleep only, with the aim of preventing patients remaining awake while in bed. Because of the detrimental effects of sleep deprivation in patients with epilepsy, sleep restriction was set at a minimum of 6 h.

The second session involved a revision of sleep scheduling, which was also repeated in the third and fourth sessions. Sleep hygiene, relaxation, and the cognitive–behavioral model of insomnia were introduced.

The third session included psychoeducation about sleep and additional relaxation exercises to further assist participants in employing relaxation at bedtime. Additionally, participants were taught how to identify and challenge dysfunctional cognitions about sleep.

In the final session, all treatment components were revised, and individualized relapse prevention plans were created for each participant.

Finally, resources and contacts for further sleep treatment were provided to participants.

2.3. Procedure

The study protocol was as follows: Participants were administered the aforementioned sleep measures. Participants who met inclusion criteria and provided consent for the study were randomly allocated to either a control or treatment group. Participants allocated to the control condition were not provided with treatment but were provided with the CBT-I handouts and program information once data had been collected from all participants. All participants were blind to the condition to which they were allocated (i.e., they were not aware whether they had been placed in a control or treatment group). Investigators were not blind to the conditions that participants had been randomized to. Those allocated to the treatment group completed the sleep diary for two weeks both prior and following the four-week intervention. The sleep measures were also readministered to all participants two weeks after the final CBT-I session. Control participants completed the sleep diary in a similar fashion to those in the treatment group, that is, they completed the sleep diary for two weeks and (following a four-week interval) they then recompleted the diary for an additional two-week period.

2.4. Statistical analyses

Kolmogorov–Smirnov tests were used to determine the suitability of variables for parametric analyses. *t*-Tests were used to assess between-group differences for continuous background and clinical variables, while chi-square tests were used to examine between-group differences on categorical variables. Data from the sleep diary and all standardized measures were assessed with a two-way (Group × Time) repeated measures analysis of variance (ANOVA). In order to determine whether the experimental CBT-I treatment should be assessed in subsequent studies with larger sample sizes (GO/NO-GO decision), the following criteria needed to be met: i) significance: high confidence that any improvements in amount and quality of sleep were due to the CBT-I intervention (i.e., an interaction effect which showed a relative improvement in amount and quality of sleep over the course of the CBT-I treatment, relative to the control group) and ii) relevance: moderate confidence that CBT-I has a relevant clinical effect (i.e., an observed improvement of at least 10% in amount and quality of sleep, relative to the control group).

Finally, linear regressions were undertaken to identify predictor variables of QOLIE-31 scores. Only variables which correlated with QOLIE-31 scores were included in the regression analyses. As this was an exploratory step, one-tailed *p*-values were used to select potential predictors. Variables were included in a hierarchical multiple regression analysis to assess their impact in predicting QOLIE-31 scores. Their order of entry into the regression analysis was determined by their strength of association with the outcome variable, with strongest predictor variables entered first and weakest associations entered last. Sleep quality (as measured by the PSQI) and beliefs about sleep were entered first because of their strong correlation with posttreatment QOLIE-31 scores. Additionally, prior aforementioned research [1,7,8,10,12,13] has already established the role of sleep quality and disturbance in determining quality of life. Total sleep time and TIB were also entered into the regression model to determine the extent to which they predicted patient quality of life. All assumptions regarding linearity, normality, equality of variance, and independence were met prior to running regression analyses. One case was removed after being identified as an outlier because their QOLIE-31 score was more than three standard deviations above the group mean. At the given recruitment of 20 patients, we calculated that the power to distinguish a five-point improvement in the PSQI between treated and control subjects was 78% assuming an alpha level of 0.05. Given that a minimum PSQI score of five is used to classify poor sleep quality, this pilot study aimed to determine whether CBT-I could result in a five-point difference in PSQI scores between the control and treatment groups.

3. Results

3.1. Background and clinical characteristics

Background and clinical characteristics for each group are summarized in Table 1. A total of 20 participants completed the study, with 11 participants in the active treatment group and nine in the control group. No between-group differences in age, gender distribution, or education level were found. Similarly, the groups did not differ with respect to age at epilepsy diagnosis, number of prescribed antiepileptic drugs (AEDs), or seizure frequency. All patients in the study were prescribed AEDs (6 on monotherapy, 14 on polytherapy), with lamotrigine (n = 9) and levetiracetam (n = 8) being the most commonly prescribed drugs. Both groups approached the cutoff score for depressive symptomatology on the NDDI-E; however, there were no differences between the groups.

Exclusion criteria included i) another neurological disorder or ii) a major sensory deficit. No participants were excluded from the study based on these criteria. Two participants were retroactively excluded from the study following the first group session as one of these participants revealed that they had completed a similar program previously (and felt they would not benefit) and another was unable to commit to further group sessions. As these participants were not included in an intent to treat design, their baseline data were deleted, and they were excluded from further consideration.

3.2. Sleep outcome data

Data related to a range of sleep measures for both groups are presented in Table 2. No differences by group were apparent in any outcome measures. Outcome change scores from baseline improved significantly across most standardized measures regardless of group. Two-way repeated measures ANOVAs were conducted to assess between-group differences for all sleep diary and standardized measures from pre- to post-CBT-I. With respect to the standardized measures, main effects of time, but not group, were found for QOLIE-31 ($F_{1, 18} = 5.10$, $p = 0.037$, $F_{1, 18} = 2.10$, $p = 0.165$), PSQI ($F_{1, 18} = 13.73$, $p = 0.002$, $F_{1, 18} = 0.56$, $p = 0.464$), SHI ($F_{1, 18} = 5.13$, $p = 0.036$, $F_{1, 18} = 1.00$, $p = 0.330$), and DBAS ($F_{1, 17} = 13.47$, $p = 0.002$, $F_{1, 17} = 1.34$, $p = 0.264$), where both the treatment and control groups exhibited improvements for each measure following the CBT-I intervention. No main effects of time or group were found for ESS scores ($F_{1, 18} = 2.40$, $p = 0.139$, $F_{1, 18} = 0.02$, $p = 0.878$). Similarly, no interaction between group and time was found for QOLIE-31 ($F_{1, 18} = 0.88$, $p = 0.361$), PSQI ($F_{1, 18} = 0.72$, $p = 0.407$), SHI ($F_{1, 18} = 0.07$, $p = 0.789$), DBAS ($F_{1, 17} = 3.70$, $p = 0.071$), or ESS ($F_{1, 18} = 0.21$, $p = 0.654$).

No main effects of time or group were found for any sleep diary variable including sleep efficiency ($F_{1, 15} = 0.27$, $p = 0.610$, $F_{1, 15} = 0.07$, $p = 0.794$, respectively), TST ($F_{1, 15} = 0.01$, $p = 0.934$, $F_{1, 15} = 0.26$, $p = 0.618$), TIB ($F_{1, 15} = 0.36$, $p = 0.559$, $F_{1, 15} = 0.55$, $p = 0.472$), sleep onset latency ($F_{1, 15} = 1.87$, $p = 0.192$, $F_{1, 15} = 0.44$, $p = 0.519$), or wake after sleep onset ($F_{1, 14} = 0.41$, $p = 0.531$, $F_{1, 14} = 0.03$, $p = 0.861$). Similarly, no Group × Time interaction effects were found for sleep efficiency ($F_{1, 15} = 0.01$, $p = 0.926$), TST ($F_{1, 15} = 1.14$, $p = 0.303$), TIB ($F_{1, 15} = 2.07$, $p = 0.171$), sleep onset latency ($F_{1, 15} = 0.28$, $p = 0.605$), or wake after sleep onset ($F_{1, 14} = 1.10$, $p = 0.312$).

Table 1
Background and clinical data by group.

	Treatment (n = 11) Mean (SD)	Control (n = 9) Mean (SD)	Test statistic	*p*
Age	39.18 (10.87)	47.44 (12.19)	$t = -1.60$	0.127
Sex (F/M)	9/2	7/2	$X^2 = 0.05$	0.822
Education (years)	13.22 (3.11)	12.00 (2.96)	$t = 0.85$	0.406
NDDI-E	14.5 (0.9)	14.6 (0.8)	$t = 0.78$	0.510
Age at diagnosis (years)	18.13 (10.91)	23.00 (18.24)	$t = 0.63$	0.543
Seizure frequency (no. per month)	5.06 (8.64)	2.19 (3.57)	$t = -0.63$	0.545
Number of AEDs	1.91 (0.54)	2.00 (1.12)	$t = -0.24$	0.814

AED: antiepileptic drug; NDDI-E: Neurological Disorders Depression Inventory for Epilepsy.

3.3. Regression analysis

No differences in background or clinical characteristics were found between the control and treatment groups, so they were merged in order to increase statistical power for the following hierarchical regression analysis. This analysis (Table 3) indicated that 86% of the variance in overall QOLIE-31 scores was accounted for by PSQI, DBAS, TST, and TIB ($F_{4, 12} = 18.26$, $p < 0.001$). This indicated that worse sleep quality, more dysfunctional beliefs about sleep, greater TIB, and greater TST predicted worse quality of life. Additionally, the analyses suggest that PSQI ($p < 0.001$) and TIB ($p = 0.043$) both made significant independent contributions to quality of life.

4. Discussion

This pilot study aimed to assess the efficacy of a group-based CBT-I program for sleep difficulties in patients with epilepsy in order to inform whether a larger-scale study would be justified. It was hypothesized that the program would improve sleep quality and excessive daytime sleepiness and that improvements in sleep would be associated with improved quality of life. Contrary to our hypothesis, the current results indicate that when compared with a sleep monitoring control group, the four-session CBT-I program did not affect participants' sleep according to data captured by a self-completed sleep diary. In contrast, both the control and CBT-I groups exhibited improvements captured by standardized measures including sleep quality, overall quality of life, sleep hygiene, and dysfunctional beliefs about sleep. When the control and CBT-I groups were considered as a whole, it was found that overall quality of life was largely predicted by select sleep parameters including sleep quality, TIB, TST, and dysfunctional beliefs about sleep.

The absence of improvement in any sleep diary parameter seems to be inconsistent with previous studies that have employed CBT-I [23,29] and may be explained by a number of reasons. Firstly, the apparent failure of CBT-I to be associated with a clear increase in TST has been previously documented in the literature [23,66] partly because of the sleep restriction requirements of this treatment. However, studies utilizing longer follow-up periods [22,23,66] have also shown that CBT-I may result in a very slight increase (or even a reduction) of TST during the initial phase of the treatment, with increases in TST only becoming apparent over the longer term, as individuals seem to practice their skills acquired during the treatment. It has also been suggested that flow-on benefits for other sleep parameters, such as sleep efficiency and daytime sleepiness, may occur after TST increases. Accordingly, one reason why sleep benefits were not observed in our study may be due to the relatively short follow-up period used (due to time restrictions), which meant that participants may not have had ample opportunity to demonstrate the full benefit gained from CBT-I.

The overall brevity of our CBT-I program could be another potential explanation for our failure to observe improvements between groups in excessive daytime sleepiness or any sleep diary measure. Because of the time restrictions of the current study, the CBT-I program was only delivered over four weekly 2-h therapy sessions. The CBT-I program is recommended to consist of approximately six 50-min individual weekly therapy sessions or fewer 90-min group sessions [34]. Our program duration and structure was in line with these guidelines and was sufficient for all content and techniques to be taught and implemented; however, this may have not been long enough for participants to consolidate, retain, and practice the material sufficiently to have an impact on certain sleep outcomes.

The results of our regression analysis indicated that when taken together, overall sleep quality, TIB, TST, and dysfunctional beliefs about sleep reported prior to the intervention predicted overall quality of life in patients with epilepsy following the intervention period. In particular, greater dysfunctional beliefs about sleep and worse sleep were strongly predictive of poorer patient quality of life. The overall finding of sleeping patterns predicting patient quality of life is consistent with previous studies which have found sleep difficulties including poor sleep quality and insomnia to be predictive of lower quality of life [1,7,8,12,13]. Neurologists or health professionals involved in the management and care of patients with epilepsy may accordingly ascribe

Table 2
Sleep data (from Consensus Sleep Diary and standardized measures) by group, pre- and post-CBT-I intervention.

	Treatment (n = 11)		Control (n = 9)	
	Pre-CBT-I M (SD)	Post-CBT-I M (SD)	Pre-CBT-I M (SD)	Post-CBT-I M (SD)
TST (minutes)	438.80 (92.15)	423.50 (130.95)	459.25 (77.57)	475.00 (66.53)
TIB (minutes)	559.60 (75.62)	522.60 (85.32)	575.75 (120.27)	587.75 (85.53)
SE (%)	76.70 (12.88)	80.00 (19.20)	80.75 (13.80)	82.00 (12.80)
SOL (minutes)	35.62 (19.71)	27.53 (23.59)	27.56 (32.77)	18.11 (18.88)
WASO (minutes)	43.91 (40.74)	27.39 (24.76)	33.16 (47.53)	34.59 (53.35)
PSQI global score	11.82 (3.79)	9.73 (5.14)	11.11 (3.92)	7.78 (3.99)
SHI	19.36 (6.89)	17.18 (8.02)	17.00 (4.80)	14.22 (4.63)
Overall QOLIE-31	43.18 (13.41)	49.64 (17.16)	54.67 (15.24)	57.33 (15.57)
DBAS	5.45 (2.11)	3.60 (1.80)	5.94 (2.12)	5.25 (1.97)
ESS	7.45 (5.72)	6.18 (5.78)	8.33 (5.17)	6.00 (5.68)

DBAS: Dysfunctional Beliefs about Sleep; ESS: Epworth Sleepiness Scale; PSQI: Pittsburgh Sleep Quality Index; QOLIE-31: Quality of Life in Epilepsy Inventory; SE: sleep efficiency; SHI: Sleep Hygiene Index; SOL: sleep onset latency; TIB: time in bed; TST: total sleep time; WASO: wake after sleep onset.

Table 3
Hierarchical regression analyses of diary and standardized sleep measures for prediction of QOLIE-31 overall score.

	B	SE *B*	β	R^2 change	*F* for change in R^2
PSQI	−2.92	0.51	−0.70	0.52	16.06**
DBAS	−1.58	1.01	−0.22	0.21	10.70**
TST	−0.03	0.03	−0.18	0.07	4.67*
TIB	−0.05	0.02	−0.32	0.06	5.12*

DBAS: Dysfunctional Beliefs about Sleep; PSQI: Pittsburgh Sleep Quality Index; QOLIE-31: Quality of Life in Epilepsy Inventory; TIB: time in bed; TST: total sleep time.
* $p < 0.05$.
** $p < 0.01$.

more weight to these variables when reported by patients because of their apparent predictive power on quality of life. These aspects should be assessed as they can act as prompts for treating clinicians to explore any potential sleep difficulties.

Despite the absence of a between-group difference in self-reported sleep activity over the course of the intervention, both groups nonetheless exhibited improvements in overall sleep quality, quality of life, sleep hygiene behaviors, and dysfunctional sleep-related beliefs. The fact that this improvement occurred within the control group suggests that sleep monitoring alone may somehow account for these improvements. It is also acknowledged that we utilized an 'active' control group as participants completed sleep diary monitoring and were contacted weekly to ensure that their sleep diaries were being completed accurately. Our findings suggest that self-monitoring of sleep behavior, in addition to attention from facilitators, is useful on its own for stimulating changes and improvements in these areas. This is not a novel finding as other CBT-I/sleep studies have also observed improvements in sleep parameters and behaviors, often equivalent to active treatment groups in monitoring-only control groups [67–69].

The results of this study carry some notable clinical implications such as the potential of self-monitoring to improve sleep in patients with epilepsy. This intervention has been successfully used to change a variety of health-related behaviors such as weight management and physical activity [70–73]. Our findings therefore lead to consideration of self-monitoring as a possible mediator of therapeutic change and suggest that it may play a role in influencing sleep behavior change. According to control theory [74–79], it is possible that monitoring sleep behavior created an accurate awareness of poor sleep which is discrepant from a participant's desired state and may have acted as a cue to engage in more productive sleep behavior (e.g., implementing better sleep hygiene skills). The use of self-monitoring for sleep improvement in patients with epilepsy would be useful given its convenience as a patient-administered intervention which requires minimal health professional contact.

Our study was not without some limitations. Firstly, our ability to assess the efficacy of our CBT-I intervention was somewhat limited (due to 78% power) to detect a clinically meaningful difference between treatment groups. Given that the power for the current study was approaching a minimum level of acceptability, future studies may benefit from recruiting a larger sample size. As highlighted above, the follow-up period could be extended in future research to assess for potential long-term benefits of the intervention that extend well beyond the treatment period. Future studies could also consider how medication and other epilepsy-related variables (such as location of seizure onset or lifetime duration of epilepsy) may impact the response to CBT-I and whether the presence of other sleep disorders (such as sleep apnea) may potentially interfere with the effectiveness of CBT-I. A further limitation of this study was the absence of an inclusion criterion for pretreatment maximum sleep time, which places a ceiling effect on TST outcomes and should be addressed in future studies. Additionally, we acknowledge that a formal diagnosis of insomnia did not feature in our inclusion criteria. Since poor sleep quality was used as a surrogate marker for insomnia in the present study, future studies may benefit a formal insomnia diagnosis as an inclusion criterion. A further consideration is that there was no follow-up data gathered on possible depressive symptomatology. This is a limitation as both sleep and quality of life may be influenced by depressive symptoms. Furthermore, no researchers on the investigation team were blinded, and this could be rectified in future studies in order to adhere to best practice. We also acknowledge that participant observation may partly explain the changes observed in this study. Finally, this study relied on subjective sleep evaluation. Research indicates that participants with sleep difficulties often underestimate their TST [80,81], and the lack of objective sleep evaluation (e.g., polysomnography or other objective physiological measure) is a limitation of the study, which could be addressed with the utilization of objective measures in future research studies.

In conclusion, the results of the present research demonstrate that CBT-I did not improve sleep or related quality of life outcomes significantly more than a control group. However, the significant improvement of both groups on overall sleep quality, sleep hygiene behaviors, beliefs about sleep, and quality of life outcomes indicates that the role of self-monitoring (which was common to both groups) may be a potential mediator of therapeutic change. This study is preliminary, and it is anticipated that it will initiate research into psychological treatment of sleep difficulties in patients with epilepsy. If future research identifies self-monitoring alone to be useful in improving sleep and quality of life outcomes in patients with epilepsy, this would further support the use of this time-efficient and low-cost intervention in improving sleep and overall patient well-being.

Declaration of competing interest

The authors declare no conflicts of interest in the submission of this manuscript.

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