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Development and Evaluation of a Cognitive Behavioural Intervention for Chronic Post-Stroke Insomnia

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Background: Cognitive behavioural therapy for insomnia (CBTI) has been successfully applied to those with chronic illness. However, despite the high prevalence of post-stroke insomnia, the applicability of CBTI for this population has not been substantially researched or routinely used in clinical practice. **Aims:** The present study developed a 'CBTI+' protocol for those with post-stroke insomnia and tested its efficacy. The protocol also incorporated additional management strategies that considered the consequences of stroke. **Method:** A single-case experimental design was used with five community-dwelling individuals with post-stroke insomnia. Daily sleep diaries were collected over 11 weeks, including a 2-week baseline, 7-week intervention and 2-week follow-up. The Insomnia Severity Index, Dysfunctional Attitudes and Beliefs About Sleep Scale, Epworth Sleepiness Scale, Fatigue Severity Scale and Stroke Impact Scale were administered pre- and post-treatment, as well as at 2-week follow-up. **Results:** At post-treatment, three participants no longer met diagnostic criteria for insomnia and all participants showed improvements on two or more sleep parameters, including sleep duration and sleep onset latency. Three participants showed a reduction in daytime sleepiness, increased quality of life and reduction in unhelpful beliefs about sleep. **Conclusions:** This study provides

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initial evidence that CBTI+ is a feasible and acceptable intervention for post-stroke insomnia. Furthermore, it indicates that sleep difficulties in community-dwelling stroke populations are at least partly maintained by unhelpful beliefs and behaviours. The development and delivery of the CBTI+ protocol has important clinical implications for managing post-stroke insomnia and highlights directions for future research.

Key words: stroke, insomnia, cognitive behavioural therapy

Introduction

Sleep disturbances are prevalent in 34–67% of people following a stroke (Campos et al., 2005; Schuiling et al., 2005; Sterr et al., 2008). Approximately 37–57% of people with stroke fulfil the criteria for insomnia (Glozier et al., 2017; Joa et al., 2017), a clinical sleep disorder defined as insufficient and/or non-restorative sleep for at least three months (American Psychiatric Association, 2013). In addition to pre-morbid sleep problems, post-stroke insomnia is attributed to disrupted neural mechanisms involved in sleep regulation and arousal (Bassetti and Valko, 2006). The physical limitations associated with stroke and fatigue may also contribute to disturbed sleep due to low levels of activity associated with the development of a weak circadian cycle, frequent napping and ultimately problematic sleep (Campos et al., 2005). While insomnia is a known contributor to poorer psychological wellbeing (Reid et al., 2006; Strine and Chapman, 2005) and increased health care costs *per se* (Wade, 2011), post-stroke sleep problems specifically impact daytime functioning including increased sleepiness, fatigue (Schepers et al., 2006) and recovery outcome (Hermann and Bassetti, 2016; Joa et al., 2017). It may also affect mobility and health care appointment attendance, as well as participation in therapies (Joa et al., 2017; Worthington and Melia, 2006). Overall, poorer quality of life after stroke is partly attributed to sleep disturbance (Schuiling et al., 2005).

Surprisinginly, addressing sleep is a poorly recognized part of post-stroke care (Castriotta and Lai, 2001; Wessendorf et al., 2000) and may only involve pharamacotherapy (Leppävuori et al., 2002; Li Pi Shan and Ashworth, 2004) which can cause sedation, tolerance, dependence and re-bound insomnia (Morin and Espie, 2003). Evidence suggests that non-pharmacological approaches are safer and more effective for long-term sleep management compared with pharmacotherapy in non-brain-injured patients (Mitchell et al., 2012; Qaseem et al., 2016); however, applications of these approaches to those with chronic physical illnesses is lacking in the literature and in the UK national health service (NHS). Sleep may be overlooked for several reasons such as lack of attention paid to sleep in medical training (Wessendorf et al., 2000), few people seeking treatment for sleep problems (Hohagen et al., 1993), low self-recognition of sleep problems (Trudel et al., 1998), and sleep being overlooked whilst coping with the other effects of stroke (Chesson et al., 2000). It has also been noted that dissemination of specific approaches to manage sleep problems is lacking amongst psychologists and therapists who have the appropriate skill set to apply non-pharmacological treatments (Troxel et al., 2012).

Unhelpful behaviours and cognitions about sleep are important maintenance factors in the development and maintenance of insomnia (Edinger and Means, 2005; Harvey, 2002). Cognitive behavioural therapy for insomnia (CBTI) aims to address unhelpful thought patterns and behaviour in poor sleep (Edinger and Means, 2005; Morin and Espie, 2003) and has been extensively used as a non-pharmacological alternative to treat insomnia. CBTI has shown clinically sustained improvements for sleep (Morin et al., 2009; Trauer et al., 2015) as well as quality of life (Morin et al., 2006) in otherwise healthy participants and in those with

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long-term medical conditions (Smith et al., 2005) including cancer (Johnson et al., 2016), diabetes (Perfect and Elkins, 2010), cardiovascular disease (Conley and Redeker, 2015) and chronic pain (Tang et al., 2015).

Few studies have examined the efficacy of CBT for sleep problems in people with brain injury. The work of Ouellet and Morin (2004, 2007) applied CBTI to patients with traumatic brain injury (TBI). They showed that eight out of 11 participants no longer fulfilled the criteria for insomnia post-treatment (Ouellet and Morin, 2007). The protocol for the study consisted of the main components of CBTI (Morin and Espie, 2003) with the addition of fatigue management. More recent studies have attempted to replicate these findings in TBI populations, revealing that CBTI has benefits for sleep, fatigue and mood (De La Rue-Evans et al., 2013; Nguyen et al., 2017a; Ouellet and Morin, 2015) and some yielding less significant effects (Lu et al., 2016).

The initial evidence suggests that CBTI is a feasible and effective approach for treating sleep problems in TBI. Although both stroke and TBI are brain injuries, TBI is qualitatively different from stroke (Nair et al., 1993) because it is caused by an external force, whereas a stroke occurs within the brain. Stroke patients tend be older, manifest a focal neurological damage and report higher rates of depression (Nair et al., 1993). TBI is also associated with overlapping post-traumatic stress disorder (PTSD) and sleep disturbance (Kaplan et al., 2010). It is also known that different types of brain injuries are associated with particular sleep disorders, including sleep apnoea (Ouellet and Morin, 2015). It is therefore important to examine specific neurological populations to further understand how CBTI can be utilized to support recovery where sleep management is equally as important as daytime rehabilitation (Duss et al., 2017). There is only one other known study that has examined the benefits of CBT for sleep disturbance within a stroke population (Nguyen et al., 2017b).

Nguyen et al. (2017b) applied an adapted CBT protocol drawn using previous fatigue and insomnia treatment manuals to a sample of people post-stroke with reported fatigue and poor sleep quality. The authors did not use insomnia criteria for inclusion. They showed that adapted CBT supported the improvement of fatigue in 5/8 participants and sleep quality in 4/8, as determined by validated questionnaires. Insomnia symptoms reported on the questionnaire improved following treatment, but this was not sustained at follow-up. This paper shows initial evidence for CBT usefulness in a stroke population and suggests that a CBTI protocol with a greater emphasis on insomnia symptoms could be of benefit for post-stroke insomnia *per se*. The additional emphasis on fatigue shows how modifying CBTI could further increase the relevance for those coping with the consequences of stroke.

With the current policies that people with long-term health problems in England will be treated by Improving Access to Psychological Therapies (IAPT), it is crucial to consider the effectiveness of current IAPT interventions for this patient group (Mental Health Task Force, 2016). CBTI is increasingly being offered in IAPT services given the evidence base and clinical demand (Espie, 2009; Manber et al., 2012; Perlis and Smith, 2008), this could therefore be an effective intervention that can be offered to people with insomnia following stroke. However, other IAPT interventions have required adaptations for them to be effective for people with long-term medical conditions (e.g. Wroe et al., 2015) and it is therefore necessary to research and adapt CBTI protocols to expand its use in other populations and service contexts (Vitiello et al., 2013) including those coping with the challenges of a stroke. Non-pharmacological approaches for stroke such as CBT foster self-management skills that are highly beneficial for improving recovery outcomes (Jones, 2006).

The present study aimed to use the traditional CBTI protocol for chronic post-stroke insomnia and incorporate additional approaches relevant for those coping with the consequences of stroke. This included ensuring that the psychoeducation components of the treatment covered the effects of stroke on sleep mechanisms and neuroplasticity. Psychoeducation is a necessary part of CBTI for understanding the treatment approach (Carney and Edinger, 2010) and it also highly relevant in brain injury (Ostwald et al., 2014). It was also ensured that stroke-related problems were part of the shared CBT formulation of sleep difficulties to support particpants' understanding of maintenance factors and potentially find modifiable areas for treatment.

Traditional CBTI incorporates strategies that aim to break the association between being awake in bed, where sleeplessness has become 'conditioned' (Perlis et al., 1997). In order to break this association, 'sleep restriction' and the '15 minute rule' are applied to prevent time spent awake in bed. Restricting sleep through postponing bedtime and anchoring earlier wake time, in addition to getting up after 15 minutes if sleep has not been achieved, can result in a temporary increase in tiredness and for some, initial distress (Morin and Espie, 2003). These strategeies may be additionally challenging for those with long-term conditions and could not only pose a safety risk but also affect engagement in the treatment. Seeking alternatives to these particular strategies could increase feasibility for this population. Therefore, replacing sleep restriction with 'sleep compression' (Morin et al., 1999) as a more gradual approach to reducing time spent awake in bed was employed in this study.

Although CBTI advises avoiding napping (Stepanski and Wyatt, 2003), it is often used by individuals to alleviate daytime sleepiness and fatigue after stroke (Campos et al., 2005). Given that sleepiness and fatigue affect daytime activities, mood and ability to function safely, 'healthy napping' was incorporated into the treatment protocol.

Overall, the above considerations factored into the traditional CBTI approach in this study is referred to as 'CBTI+'. This study aimed to explore the efficacy of CBTI+ using a single-case experimental design (SCED), an appropriate method for assessing acceptability and feasibility of newly developed interventions (Rassafiani and Sahaf, 2010) using insomnia symptoms as the primary outcome.

Method

Design

The present study used a case series methodology with an A-B-A design. This entailed a 2-week baseline (A) followed by 7 weeks of treatment (B) with a follow-up 2 weeks after the end of treatment (A).

Participants

Community-dwelling individuals post-stroke for at least 12 months, therefore in the chronic phase of recovery, were approached via several local links, including stroke groups and charities, using flyers and posters. Potential participants who responded to study adverts were contacted for a telescreening appointment lasting approximately 20 minutes to check eligibility for the study and provide further information. Eleven individuals were telescreened and all met

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Table 1. Participant demographics and scores on questionnaires used for inclusion criteria

Demographic	P1	P2	P3	P4	P5
Age (years)	53	68	73	58	47
Gender	Female	Male	Female	Female	Male
Time since injury (years:months)	21:2	7:11	4:5	3:3	6:11
Affected hemisphere	Right	Left	Right	Left	Left
MMSE	30/30	29/30	30/30	28/30	29/30
ADL (SIS)	82.50	70.00	55.00	62.50	60.71
Sleep duration	3–4 h	4–7 h	3 h	6 h	4 h
SOL	2–3 h	30+ min	1+ h	30+ min	0–2 h
SDQ: total score for items 2, 4, 6, 10, 12	10/20	12/20	9/20	6/20	10/20
SDQ: total	7/48	21/48	20/48	20/48	17/48
SHPS: sleep hygiene behaviours	Exercise before bed; worry about inability to sleep; napping; cigarettes	Napping; using sleep medication; varied sleep lengths	Napping; varied sleep lengths	Napping; caffeine	Napping; exercise before bed

Sleep duration and sleep onset latency (SOL) were as reported on average per night. SDQ includes number of items rated 'often'. SHPS includes the reported unhelpful sleep behaviours.

the preliminary criteria based on the DSM-IV[1] criteria for insomnia: (1) sleep onset is more than 30 minutes, (2) sleep duration is less than 6.5 hours, (3) three or more awakenings symptoms, (4) taking longer than 30 minutes to get back to sleep, (5) awakening 30 minutes before desired time, and (6) symptoms are associated with distress or impairment. These symptoms are based on self-reports and should occur three or more times a week.

Five participants who met the initial criteria agreed to come for the full assessment, which lasted approximately 90 minutes. Following assessment, all five participants met the full study inclusion/exclusion criteria. In addition to the DSM-IV criteria for insomnia, this included rating at least one response as 'often true' (given a score of 3/4) for the following five unhelpful cognitions listed on the Sleep Disturbance Questionnaire (SDQ; Espie, Brooks and Lindsay, 1989): 'My mind keeps turning things over'; 'I get too "worked up" at not being able to sleep'; 'My mind takes a long time to unwind'; 'I am unable to empty my mind'; and 'I worry that I won't cope tomorrow if I don't sleep'. The Sleep Hygiene Awareness and Practice Scale (SHAPS; Lacks, 1987) was used to indicate presence of unhelpful behaviours that maintain poor sleep. Presence of at least two unhelpful behaviours for sleep were used for inclusion criteria (e.g. napping, varied sleep lengths or caffeine use). Other inclusion criteria included GP approval and being at least one year post-stroke. Exclusion criteria were sleep disorders other than insomnia, medication that adversely impacts sleep, other serious co-morbid conditions, untreated psychological difficulties, significant cognitive difficulties (<25 on the Mini-Mental State Exam; Folstein and Folstein, 1975), uncorrected visual impairment and aphasia. All participant details, referred to as P1 to P5, are presented in Table 1. P2 used temzepam

[1] DSM-V criteria were not published at the time of recruitment for this study.

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(10–20 mg) once to twice a week if he could not get to sleep, but the frequency could increase if sleep was particularly poor.

Ethics

Ethical approval was gained from the relevant university ethics committees and all participants gave their informed consent prior to participation.

Measures

Self-report daily sleep diaries were provided to all participants in paper form and were completed over 11 weeks, which included the 2-week baseline, 7-week intervention and 2-week follow-up. The sleep diaries collected information on bed time, wake time, get up time, sleep duration (SD; min) and sleep onset latency (SOL; min). Sleep efficiency (SE; %) was obtained by calculating the percentage time spent sleeping whilst in bed. Averages of sleep diary parameters were calculated per week.

A battery of questionnaires was administered at 2 weeks prior to treatment and at follow-up. Scores on the Insomnia Severity Index (ISI; Bastien et al., 2001) were used to measure insomnia severity where $\geq$7 suggests not clinically significant, 8 to 14 suggests sub-threshold for insomnia, 15 to 21 indicates moderate insomnia, and $\geq$22 is severe. To provide additional therapeutic information about the patient's experience of having a sleep problem, the Dysfunctional Beliefs and Attitudes About Sleep Scale (DBAS-16; Morin et al., 2007) was administered. It was also used to detect any change in attitude towards sleep post-treatment. A higher score on the DBAS-16 suggests that a person has a greater presence of unhelpful beliefs about sleep.

Daytime functioning measures were the Epworth Sleepiness Scale (ESS; Johns, 1991) and the Fatigue Severity Scale (FSS; Krupp et al., 1989). The ESS records sleep propensity through eight hypothetical situations from which participants were asked to rate how likely they would fall asleep using a 4-point Likert scale ranging from 0 (no chance of dozing) to 3 (high chance of dozing). Items are summed to give an overall score. Scores between 0 and 6 indicate low sleepiness, 7 to 9 is average, and 10 to 24 indicates severe sleepiness. The FSS was used to measure fatigue, defined as physical or mental weariness as a result of exertion (Hossain et al., 2005). It consists of nine statements relating to the impact of fatigue from which participants rate on an 8-point Likert scale how much they agree or disagree from 0 to 7. The mean score $>$4 suggests a moderate to high fatigue (Mathiowetz et al., 2001).

The Stroke Impact Scale (SIS v.3; Duncan et al., 2003) is a multidimensional measure of the impact of stroke on quality of life and was used to examine changes following improved sleep. The SIS contains 59 items corresponding to eight subscales including: strength (S), hand function (HF), activities of daily living (ADL), mobility (M), communication (C), emotion (E), memory and thinking (MT), and social participation (SP). Response choices for each question are weighted on a 5-point Likert scale, ranging from 1 to 5. Scores for each item were transformed using the following algorithm: [(actual raw score – lowest possible raw score)/possible raw score]$\times$100. Higher scores signify better quality of life. Due to the large amount of data provided by the SIS, all outcomes are presented in the Supplementary Material. To enable the reader to gain an understanding of level of disability following the stroke for each participant, ADL scores at pre-treatment are provided in Table 1.

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All questionnaires have been shown to be valid and reliable measures when used with brain-injured populations, except the DBAS and ISI. However, these measures have been routinely used to investigate CBTI outcomes (Bastien et al., 2001; Carney and Edinger, 2006; Morin et al., 2007).

CBTI+

A seven-session treatment manual was drafted by the authors drawing on standard CBTI techniques (Morin and Espie, 2003; Perlis et al., 2005) with the addition of professional expertise in stroke rehabilitation and feedback from service users. The resultant manual covered: (1) psychoeducation, (2) identifying maintenance factors of poor sleep through developing a formulation, (3) sleep hygiene, stimulus control and relaxation, (4) sleep scheduling, (5) managing emotions, (6) noticing and managing styles of thinking and (7) relapse prevention.

The 'CBTI+' protocol was delivered in the context of stroke, which ensured that other health factors were recognized and considered during treatment. This included providing information about how sleep processes are affected post-stroke, considerations around medication, neuroplasticity mechanisms and how the physical limitations associated with stroke can affect sleep routines and activity as well as information about insomnia within the psychoeducation component. When advising on reducing sedentary lifestyle as part of sleep hygiene, consideration was given to physical limitations commonly observed post-stroke. Participants were encouraged to increase activity where manageable and to use 'pacing' techniques as advised in both fatigue and chronic pain literature (Neilson et al., 2013).

The '15 minute rule' aspect of the stimulus control component requires a person to leave the bedroom when failing to fall asleep within 15 minutes. Due to potential difficulties with implementing this, an alternative, such as a relaxation audio, was advised.

The traditionally applied sleep restriction approach aims to curtail time spent in bed to average sleep length, e.g. five hours, and gradually increasing time in bed by 15 minutes when sleep efficiency (% time spent asleep whilst in bed) reaches 90%. We exchanged this strategy for sleep compression as a gentler approach to managing learned sleeplessness that was considered more approapriate for those with long-term conditions. Sleep was compressed by reducing time in bed by dividing the difference between sleep time and time in bed by five. This time is then added to bed time so the person will go to bed later for one week. Should their sleep efficiency not reach 90%, sleep is further compressed by this amount. Should participants require to nap as part of managing their day, we advised that naps should occur only in bed and for no longer than 30 minutes, as well as before 4 p.m.

Furthermore, some of the cognitive work on thought management involved discussing the impact of having a stroke, as well as sleep problems. This also relied on the therapist to ensure that the work was orientated towards managing sleep in the context of stroke rather than CBT for coping with the consequences of stroke.

Procedure

Sessions were administered on an individual basis over 7 to 12 weeks and lasted between 60 and 90 minutes. All sessions were administered by a trainee clinical psychologist (K.H.) under supervision. K.H. was in the third year of a clinical psychology doctoral programme at Royal

Holloway, University of London, having previously completed a PhD in sleep-related research and received training in CBTI by Professor Colin Espie.

Analysis

Statistically significant changes in the questionnaire measures were identified using the 'reliable change index' (RCI; Jacobson and Truax, 1991). The RCI uses published reliability and population norms of the measure in question to determine whether any changes in scores are greater than that expected through measurement error (O'Neill, 2010). The RCI determines a minimum change score required for statistical significance. The RCI was calculated using online software (http://www.psyctc.org/stats/rcsc1.htm) that provides a modified calculation based on the original formula (*SD* = standard deviation, *r* = published reliability of the measure): $RCI = SD \times \sqrt{(2)} \times \sqrt{(1-r)}$ (Evans, 1998). The minimum scores for RCI calculations for each questionnaire are described in the Results section. Norms are provided for insomnia cohorts.

For sleep diaries, improvements in the direction of healthy sleep at post-treatment and follow-up were examined using population norms. 'Clinically meaningful change' (CMC) allows the researcher to consider the extent to which the data changes in the target outcome measure, towards that expected in the normal population. Jacobson et al. (1984) outlined a method to examine the data to determine clinical change. This can be achieved in two ways: (a) when a person has moved more than 2*SD* from the clinical group (e.g. insomnia normative data); and (b) when a person moves within 2*SD* of the normal population. For the purposes of this study, both (a) and (b) will be examined as there is overlap between sleep behaviour in the general population and in those with clinically significant insomnia. This may occur because normative data, particularly in self-reported sleep, may contain data from less healthy sleepers that do not fulfil the criteria for insomnia. Both normative and insomnia population data, for comparison with the data in the current study, were extracted from large studies as shown in Table 3a.

In order to allow the data to be compared with larger data sets including randomized controlled trials, effect sizes (ES) of the aggregated data for all questionnaires except the SIS and sleep diary measures were calculated. The following formula was used: ES (*d*) = [(pre-CBTI+ mean) – (post-CBTI+ mean)]/pre-CBTI+SD.

Results

All participants, referred to as P1 to P5, completed the intervention and provided follow-up data.

Insomnia symptoms

Three participants fell within the moderately severe clinical range on the ISI at pre-treatment and the remaining two participants were subclinical (Table 2). According to the RCI calculation for the ISI (Beaulieu-Bonneau et al., 2007), change scores ≥4.9 and ≥5.9 are significant based on normal and insomnia population norms, respectively. At post-treatment, all participants reported a reduction in the severity of their insomnia symptoms, with three meeting the criteria for reliable improvement and maintaining the gains at follow-up. These three participants

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Table 2. ISI, DBAS, ESS and FSS outcomes per participant at pre-treatment, post- and follow-up

Cases	Measure	Pre-treatment	Post-treatment	Follow-up	Difference pre/post
P1	ISI	16	11	19	–5†
	DBAS	6.1	5.3	–	−0.8
	ESS	1	5	3	+4
	FSS	5.56	6.56	6.0	+1
P2	ISI	11	7	8	−4
	DBAS	3.1	3.2	–	+0.1
	ESS	5	6	5	+1
	FSS	3.44	3.67	4.00	–0.23
P3	ISI	14	8	6	–6†‡
	DBAS	5.8	3.1	–	–2.7†‡
	ESS	11	7	9	–4*
	FSS	1.8	2.6	2.6	+0.78
P4	ISI	17	5	1	–12†‡
	DBAS	5.1	3.5	–	–1.6†‡
	ESS	15	4	3	–11*†‡
	FSS	6.67	5.89	6.00	–0.78†
P5	ISI	18	8	5	–10*†‡
	DBAS	6.1	3.8	–	–2.3†‡
	ESS	16	13	12	–4*
	FSS	6.22	6.67	6.89	0.45

Symbol key indicates significance when RCI is based on normal sleeper†, insomnia‡ and general* population norms.

were in the 'not clinically significant insomnia' range by the time of the follow-up (see Table 2). Despite the improvement observed for P1 at post-treatment from moderate insomnia to subclinical, her symptoms worsened by follow-up into the clinical range. P2 did, however, move into the subclinical range from the moderately severe range which remained at follow-up. Two of these participants remained out of clinical range at follow-up. Although P5 was within the subclinical range at post-treatment, this came out of clinical range at follow-up. ES (*d*) was large at 2.67 for ISI.

Impact on sleep cognitions and daytime functioning

The significant decreases found in DBAS scores (Table 2), using an RCI ≥1.6 (Carney and Edinger, 2006), suggests that three participants (P3, P4 and P5) developed helpful belief changes about sleep post-treatment (Table 2) with a group ES (*d*) of 1.15. Prior to treatment, three participants were above the clinical cut-off for sleepiness and moderate fatigue according to the ESS and FSS (Table 3). ESS RCI could be provided for general (≥4.8), normal (≥4.7) and insomnia populations (≥5.3) (Anderson and Horne, 2008; Sanford et al., 2006). Three participants reported significant improvements in sleepiness with one participant out of clinical range. Fatigue scores revealed no significant change at post-treatment and follow-up when using FSS RCI for normal and insomnia norms of ≥0.81 and ≥0.37, respectively (Lichstein et al., 1997; Valko, et al. 2008). ES (*d*) were lower for these measures at 0.40 and –0.17 for ESS and FSS, respectively.

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Table 3a. Mean self-reported sleep parameters for clinically meaningful change analyses drawn from Lichstein et al. (2004, 2006) matched for age and gender for the normal sample and for insomnia norms

Sleep diary parameter	Females 50–59 years	Female 70–79 years	Males 40–49 years	Males 60–69 years	Insomnia norms
SOL (min)	24.3 (19.9)	28 (19.4)	22.0 (18.9)	16.8 (12.1)	42.3 (24.8)
SD (min)	407.7 (62.7)	413.0 (70.0)	401.4 (57.8)	430 (65.7)	384 (72.7)
SE (%)	86.0 (9.0)	82.3 (10.5)	86.3 (10.6)	88.5 (6.5)	75.5 (9.5)

Values given are means (*SD*).

RCI for SIS indicates that any changes ≥9.4 indicate reliable change as drawn from Duncan et al. (2003). All participants reported improved quality of life in one or more domains according to the SIS at post-treatment (see Supplementary Material for all SIS results). P1 reported a significant improvement within the emotional functioning domain ($E_{pre} = 83.33$; $E_{post} = 97.22$), which did not extend to follow-up. P1 showed significant improvement in social participation ($SP_{pre} = 60.71$; $SP_{post} = 75.00$) that did not remain at follow-up. She also reported worse memory and thinking ($MT_{pre} = 92.86$; $MT_{post} = 67.86$) at post-treatment, likely to reflect her relapse in poorer sleep. P3 described increased social participation ($SP_{pre} = 28.13$; $SP_{post} = 50.00$), and better memory and thinking ($MT_{pre} = 85.71$; $MT_{post} = 96.43$), which remained at follow-up. Emotional functioning ($EF_{pre} = 69.44$; $EF_{post} = 94.44$), social participation ($SP_{pre} = 43.43$; $SP_{post} = 82.14$) and communication ($C_{pre} = 57.14$; SIS: $C_{post} = 71.43$) improved for P4, which extended to follow-up. P5 reported improved activities of daily living ($ADL_{pre} = 85.71$; $ADL_{post} = 96.43$), which remained at follow-up.

Sleep diary

According to large insomnia population data (Table 3a), the self-reported sleep duration (SD) for all participants were within 1*SD* of insomnia norms, in line with clinically poor sleep (Table 3b). All patients reported at least 16 minutes average increase in sleep duration. Three participants reported achieving at least 30 minutes more sleep on average at post-treatment (Table 3b). P5's sleep duration was within both the lower limit of the clinical range and upper limit for the normal range at pre-treatment; sleep duration then remained within the normal range only at post-treatment and follow up. ES (*d*) for the group was 0.26.

Although four out of five participants reported taking over 30 minutes to fall asleep at post-treatment, a clinical indicator of insomnia (American Psychiatric Association, 2013), all reported reductions in subjective SOL, which remained at follow-up in three participants. P2 was the only participant to move out of clinical range post-treatment and at follow-up for SOL. The SOL group ES (*d*) was 0.56.

SE was below the healthy level of 85% in four participants at pre-treatment. In three participants, SE improved towards the healthy level of 85% at post-treatment, which was maintained at follow-up. P3 moved into 1*SD* of normal population for 17% improvement in sleep efficiency, which was not maintained at follow-up although still showed a 13% gain. As a group, the ES (*d*) was 0.55.

Despite P5 being reported at assessment to fit inclusion criteria for the study, SOL and SE were out of clinical range for insomnia according to sleep diaries at pre-treatment.

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Table 3b. Clinically meaningful change is indicated when a participant report falls within at least 1*SD* of normal* and insomnia† populations

Case	Sleep diary parameter	Pre-treatment	Post-treatment	Post minus pre	Follow-up
P1	SOL (min)	300†	75†	–225	210†
	SD (min)	180†	210†	+30	200†
	SE (%)	37.5†	55.7†	+17.2	43.9†
P2	SOL (min)	32.1†	14.3*	–17.8	17.5*
	SD (min)	402.9†*	419.3†*	+16.4	410†*
	SE (%)	74.6†	85.8*	+11.2	84.8*
P3	SOL (min)	115†	51.4†	–63.6	80†
	SD (min)	287†	334†	+47	330†
	SE (%)	57.1†	74.3†*	+17.2	70.7†
P4	SOL (min)	60†	35†*	–25	52†
	SD (min)	385†*	415†*	+30	450†*
	SE (%)	80.6†*	83.4†*	+10.4	91*
P5	SOL (min)	10*	13.6*	+3.6	11.9*
	SD (min)	445†*	462*	+17	462*
	SE (%)	89.1*	96.2*	+7.1	94.9*

Discussion

This study aimed to examine the feasibility and acceptability of a manualized CBTI+ that is specifically tailored to post-stroke insomnia. Overall, the results show that four out of the five participants reported significant improvements in sleep disturbance at post-treatment according to increased SD (all participants), reduced SOL (P1–4) and improved ISI score (all except P2). Three participants maintained improvements in at least two of these domains at follow-up. Three participants showed a significant decrease in unhelpful beliefs about sleep. This suggests that insomnia maintained by unhelpful cognitions and behaviours can be reversible in people who have had a stroke.

The group effect size calculations for pre- and post-differences showed stronger magnitudes >1 from the ISI and DBAS-16, which are at least comparable with RCTs (Manber et al., 2008). Sleep diary data showed medium effects for SOL and SE similar to RCTs (Currie et al., 2000; Edinger et al., 2005). SD showed a small effect whereas RCT results are at least medium. Similarly to the studies that have applied CBTI to TBI (De La Rue-Evans et al., 2013; Nguyen et al., 2017a; Ouellet and Morin, 2015), this study suggests that this protocol also has clinical benefits for post-stroke sleep disturbance.

The results did not show any improvements in fatigue reports. Whilst studies in insomnia populations show that improving sleep–wake cycles can also reduce fatigue (Morin et al., 2006), those with physical health problems are less likely to see improvements (Berger et al., 2009). A Cochrane review concluded that there is insufficient evidence to guide practice for post-stroke fatigue following a review of both pharmacological and non-pharmacological studies (McGeough et al., 2009). Given that stroke is associated with elevated levels of fatigue as a result of multiple neurological problems affecting mobility, cognition and general activity (Campos et al., 2005), it is likely that additional fatigue management and neurorehabilitation strategies are required to support improvement (White et al., 2011).

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Therefore, further research is necessary to develop 'holistic' approaches to manage both sleep and daytime difficulties in stoke. The work of Nguyen et al. (2017b) has shown promising results for applying CBT approaches to fatigue which would warrant further investigation.

Quality of life improved on one or more domain of the SIS, most notably social participation for three participants at follow-up. This is consistent with the studies that have shown quality of life benefits following CBTI in those with other long-term medical conditions (Espie et al., 2008).

Therapeutic reflections

Post-stroke considerations. All participants had post-stroke mobility problems and would have found it difficult to apply the 15 minute rule strategy as traditionally used in CBTI. The upheaval required to get up in the night and move to another room could have caused a greater level of arousal as well as concern about managing movement whilst drowsy or requiring carer assistance. Using relaxation or other meditation strategies did not appear to compromise benefit of CBTI+ according to the results. The sleep compression alternative to sleep restriction was taken on board by all participants and reduced potential risks from temporary sleep deprivation (Kyle et al., 2011). For any people with multiple physical health problems, sleep compression is a useful strategy for CBTI.

Two participants reported that pain symptoms interrupted their sleep at times. Pain management was not considered within the current CBTI+ model. The impact of post-stroke pain upon sleep has been documented as highly disruptive for sleep (Fictenberg et al., 2001; Widar et al., 2004). Furthermore, poor sleep can influence perception of pain (Lautenbacher, Kundermann and Krieg, 2006). For these patients, it may have been beneficial to consider additional self-management treatments for pain (Williams et al., 2012) which could be included in a CBTI+ manual. The literature strongly supports the use of CBT approaches for supporting self-management for fatigue (Malouff et al., 2008) and chronic pain (Eccleston et al., 2013) with promising results for stroke populations (Nguyen et al., 2017b; Harrison and Field, 2015).

Adherence. The literature on CBTI, as well as traditional CBT, highlights a strong correlation between level of adherence and outcome (Vincent, Lewycky and Finnegan, 2008). In the present study, most participants reported difficulty adhering to the treatment strategies at times. Low adherence in CBTI has been attributed to low self-efficacy regarding personal abilities to follow treatment advice (Bouchard, Bastien and Morin, 2003; Ouellet and Morin, 2007) and previous difficulty in managing insomnia (Janz and Becker, 1984). It is likely that participants in this study encountered similar problems. Participants from the current study fed back that barriers to adhering to strategies at times included social events, illness and difficulty in changing well-established habits surrounding sleep routines. This may further be influenced by the complications of stroke. It is therefore important to consider flexibility in manualized treatments where necessary, to address problems with adherence and facilitate open conversations about self-efficacy.

Medication. It is also important to consider medication when interpreting the results of this study. P2's use of sleep medication may have contributed to treatment outcome and

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may also contribute to lower adherence to the techniques. Medication use may represent a safety behaviour that allows temporary relief of symptoms and the effort in having to manage them as commonly observed in anxiety disorders (Wells et al., 1995). Sleep medication does not address the presumed mechanism underpinning insomnia; therefore, symptoms typically return once treatment is discontinued (Siebern and Manber, 2011). Although P2's therapeutic gains were further limited by lower engagement, his level of distress reduced, sleep improved and medication use lowered during the study from a maximum of six doses per week during treatment to no more than three per week from session four. A medication withdrawal programme (Morin et al., 2004) prior to CBTI+ may have been of further benefit for P2.

Methodological issues

Research suggests that those with sleep disorders are less accurate at reporting sleep behaviour (Kushida et al., 2001; Means et al., 2003). This has important implications for interpretation of data in the current study and findings in the literature, which relies predominantly on subjective reports of sleep for diagnosis and therapy outcomes (Morin et al., 2006). It has been noted that P5 fed back his average sleep length as 4 hours at assessment; however, through sleep diary collection, his average sleep duration was 7 hours at pre-treatment. Studies have shown that those with distress about sleep are likely to under-report sleep lengths (Dopke et al., 2004; Ouellet and Morin, 2007). It is also seen in CBTI studies that keeping a sleep diary, normalizing insomnia symptoms and recognizing behaviour change can help sleep, has implications for reducing distress about sleep and greater accuracy in reporting sleep parameters (Smith et al., 2004). For P5, attending assessment could have, in part, initiated these processes.

Future studies would benefit from utilizing objective measures such as actigraphy to supplement self-report measures, a favoured method when 'gold standard' sleep electroencephalography recordings are not possible (Ancoli-Israel et al., 2003). Actigraphy is recognized as a valid measure of long-term sleep behaviour in stroke populations (Hermann et al., 2008; Schuiling et al., 2005).

The ability to generalize small sample data to larger populations is limited in SCEDs due to lack of randomization for a truly representative sample of the population in question and likely recruitment bias (Wilson, 2000). Despite these limitations, the present study provides a useful therapeutic structure for adapted CBTI from which larger studies can be designed. Tate et al. (2016) encourage the use of SCEDs with stricter guidelines. Given the sample size in this study and the exclusion criteria, the results are suggestive of benefits of CBTI in post-stroke insomnia in participants without significant cognitive impairment. It will be important for future research to investigate the applicability of CBTI treatments for those with cognitive/communication difficulties given that between 30 and 34% of people will experience this following stroke (Arba et al., 2016; Flowers et al., 2016).

Previous studies have shown that up to two-thirds of participants maintain healthy sleep after CBTI (Morin et al., 2004) for several months after treatment. In order to fully assess the sustainability of sleep gains, longer follow-up data are required, which was not achieved with this study. This would be beneficial to determine the degree to which the learned techniques can be maintained in the absence of therapeutic sessions. It was observed in the current study that some improvements were not maintained at follow-up, which suggests that review sessions could be useful to support treatment efficacy.

Implications

While psychological options for treating insomnia are becoming more available in primary care (Edinger and Sampson, 2003; Espie, 2009), the lack of research in modified support for those with insomnia and long-term medical conditions is likely to reflect the lack of such treatment available in health services. Given that sleep is an important mediator for recovery from illness (Briones et al., 1996; Hyyppä and Kronholm, 1995; Worthington and Melia, 2006), it is surprising that sleep is poorly recognized as part of post-stroke care (Castriotta and Lai, 2001; Worthington and Melia, 2006). It is therefore important for health care professionals to have awareness of approaches available that can support the management of post-stroke symptoms without the use of pharmacotherapy. The findings of this study suggest the evidence-based protocols of CBTI, which is currently offered in many IAPT services, can be modified to be effective for people with stroke, and may therefore be an effective intervention offered in IAPT settings for this patient group. Furthermore, introducing these approaches early could be an important aspect for rehabilitation in building healthy sleep routines to support other aspects of recovery. It is also important to consider the importance of increasing accessibility of CBT treatments through adaptations and modifications of traditional protocols for those recovering from stroke (Kneebone, 2016).

Conclusion

People with post-stroke insomnia can benefit from a short-term CBTI+ intervention specifically designed to target post-stroke insomnia maintained by maladaptive behaviours and cognitions. The incorporation of CBTI+ into post-stroke care requires further investigation.

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Ethical statements: The Royal Holloway, University of London, Psychology Department Ethics Committee approved the protocol in May 2011. All procedures adhered to The Declaration of Helsinki (World Medical Association, 2000) and authors have abided by the Ethical Principles of Psychologists and Code of Conduct as set out by the American Psychological Association (2003).

Conflicts of interest: There are no conflicts of interest to declare.

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Supplementary material

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References

American Psychiatric Association (2013). *Diagnostic and Statistical Manual of Mental Disorders* (5th edn). American Psychiatric Publishing, Arlington, VA, USA.

Ancoli-Israel, S., Cole, R., Alessi, C., Chambers, M., Moorcroft, W. and Pollak, C. P. (2003). The role of actigraphy in the study of sleep and circadian rhythms. *Sleep*, *26*, 342–392.

Anderson, C. and Horne, J. A. (2008). Do we really want more sleep? A population-based study evaluating the strength of desire for more sleep. *Sleep Medicine*, *9*, 184–187.

Arba, F., Quinn, T., Hankey, G. J., Inzitari, D., Ali, M. and Lees, K. R. (2016). Determinants of post-stroke cognitive impairment: Analysis from VISTA. *Acta Neurologica Scandinavica*, *135*, 603–607.

Bassetti, C. and Valko, P. (2006). Post-stroke hypersomnia. *Sleep Medicine Clinics*, *1*, 139–155.

Bastien, C. H., Vallieres, A. and Morin, C. M. (2001). Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Medicine*, *2*, 297–307.

Beaulieu-Bonneau, S., LeBlanc, M., Merette, C., Dauvilliers, Y. and Morin, C. (2007). Family history of insomnia in a population based sample. *Sleep*, *30*, 1739–1745.

Berger, A. M., Kuhn, B. R., Farr, L. A., Lynch, J. C., Agrawal, S., Chamberlain, J. and Von Essen, S. G. (2009). Behavioral therapy intervention trial to improve sleep quality and cancer-related fatigue. *Psycho-Oncology*, *18*, 634–646.

Bouchard, S., Bastien, C. and Morin, C. M. (2003). Self-efficacy and adherence to cognitive-behavioral treatment of insomnia. *Behavioral Sleep Medicine*, *1*, 187–199.

Briones, B., Adams, N., Strauss, M., Rosenberg, C., Whalen, C., Carskadon, M., Roebuck, T., Winters, M. and Redline, S. (1996). Relationship between sleepiness and general health status. *Sleep*, *19*, 583–588.

Campos, T. F., Diogenes, F. P., Franca, F. R., Dantas, R. C. S., Araujo, J. F. and Menezes, A. A. L. (2005). The sleep-wake cycle in the last stage of cerebral vascular accident recovery. *Biological Rhythm Research*, *36*, 109–113.

Carney, C. E. and Edinger, E. D. (2006). Identifying critical dysfunctional beliefs about sleep in primary insomnia. *Sleep*, *29*, 440–453.

Carney, C. E. and Edinger, J. D. (2010). *Cognitive Strategies for Managing Anxiety and Insomnia*. Springer.

Castriotta, R. J. and Lai, J. M. (2001). Sleep disorders associated with traumatic brain injury. *Archives of Physical Medicine and Rehabilitation*, *82*, 1403–1406.

Chesson, A., Hartse, K., Anderson, W. M., Davila, D., Johnson, S., Littner, M., Wise, M. and Rafecas, J. (2000). Practice parameters for the evaluation of chronic insomnia. An American Academy of Sleep Medicine report. Standards of Practice Committee of the American Academy of Sleep Medicine. *Sleep*, *23*, 237–241.

Conley, S. and Redeker, N. S. (2015). Cognitive behavioural therapy for insomnia in the context of cardiovascular conditions. *Current Sleep Medicine Reports*, *1*, 157–165.

Currie, S. R., Wilson, K. G., Pontefract, A. J. and deLaplante, L. (2000). Cognitive-behavioral treatment of insomnia secondary to chronic pain. *Journal of Consulting Clinical Psychology*, *68*, 407–416.

De La Rue-Evans, L., Nesbit, K. and Oka, R. K. (2013). Sleep hygiene program implementation in patients with traumatic brain injury. *Rehabilitation Nursing*, *38*, 2–10.

Dopke, C. A., Lehner, R. K. and Wells, A. M. (2004). Cognitive-behavioral group therapy for insomnia in individuals with serious mental illnesses: a preliminary evaluation. *Psychiatriatric Rehabilitation Journal*, *27*, 235–242.

Duncan, P. W., Lai, S. M., Bode, R. K., Perera, S. and DeRosa, J. (2003). Stroke Impact Scale-16: a brief assessment of physical function. *Neurology*, *60*, 291–296.

Duss, S. B., Seiler, A., Schmidt, M. H., Pace, M., Adamantidis, A., Muri, R. M. and Bassetti, C. L. (2017). The role of sleep in recovery following ischemic stroke: a review of human and animal data. *Neurobiology of Sleep and Circadian Rhythms*, *2*, 94–105.

Eccleston, C., Morley, S. J. and Williams, A. C. de, C. (2013). Psychological approaches to chronic pain management: evidence and challenges. *British Journal of Anaesthesia*, *511*, 59–63.

Edinger, J. D. and Means, M. K. (2005). Cognitive-behavioral therapy for primary insomnia. *Clinical Psychology Review*, *25*, 539–538.

Edinger, J. D. and Sampson, W. S. (2003). A primary care 'friendly' cognitive behavioral insomnia therapy. *Sleep*, *26*, 177–182.

Edinger, J. D., Wohlgemuth, W. K., Krystal, A. D. and Rice, J. R. (2005). Behavioral insomnia therapy for fibromyalgia patients: a randomized clinical trial. *Archives of International Medicine*, *165*, 2527–2535.

Espie, C. A. (2009). 'Stepped Care': a health technology solution for delivering cognitive behavioral therapy as a first line insomnia treatment. *Sleep*, *32*, 1549–1558.

Espie, C. A., Brooks, D. N. and Lindsay, W. R. (1989). An evaluation of tailored psychological treatment of insomnia. *Journal of Behavioral Therapy and Experimental Psychiatry*, *20*, 143–153.

Espie, C. A., Fleming, L., Cassidy, J., Samuel, L., Taylor, L. M., White, C. A. et al. (2008). Randomized controlled clinical effectiveness trial of cognitive behavior therapy compared with treatment as usual for persistent insomnia in patients with cancer. *Journal of Clinical Oncology*, *26*, 4651–4658.

Evans, C. (1998). Reliable change criterion calculator. Available at: http://www.psyctc.org/stats/rcsc1.htm (accessed January 2012).

Folstein, M. and Folstein, S. (1975). Mini-mental state: a practical method for grading the cognitive state of patients for the clinician. *Journal of Psychiatric Research*, *12*, 189–198.

Fictenberg, N. L., Putnam, S. H., Mann, N. R., Zafonte, R. D. and Millard, A. E. (2001). Insomnia screening in post-acute traumatic brain injury: utility and validity of the Pittsburgh Sleep Quality Index. *American Journal of Physical Medicine and Rehabilitation/Association of Academic Physiatrists*, *80*, 339–345.

Flowers, H. L., Skoretz, S. A., Silver, F. L., Rochon, E., Fang, J., Flamand-Roze, C. and Martino, R. (2016). Post-stroke aphasia frequency, recovery, and outcomes: a systematic review and meta-analysis. *Archives of Physical and Medical Rehabilitation*, *97*, 2188–2201.

Glozier, N. Moullaali, T. J., Sivertsen, B., Kim, D., Mead, G., Jan, S., Li, Q. and Hackett, M. L. (2017). The course and impact of post-stroke insomnia in stroke survivors aged 18 to 65 years: results from the Psychosocial Outcomes In StrokE (POISE) Study. *Cerebrovascular Diseases Extra*, *7*, 9–20.

Harrison, R. A. and Field, T. S. (2015). Post-stroke pain: identification, assessment and therapy. *Cerebrovascular Diseases*, *39*, 190–201.

Harvey, A. G. (2002). A cognitive model of insomnia. *Behaviour Research and Therapy*, *40*, 869.

Hermann, D. M., Siccoli, M. M., Brugger, P., Watcher, K., Mathis, J., Achermann, P. and Bassetti, C. L. (2008). Evolution of neurological, neuropsychological and sleep-wake disturbances after paramedian thalamic stroke. *Stroke*, *39*, 62–68.

Hermann, D. M. and Bassetti, C. L. (2016). Role of sleep-disordered breathing and sleep-wake disturbances for stroke and stroke recovery. *Neurology*, *87*, 1407–1416.

Hohagen, F., Rink, K., Kappler, C., Schramm, E., Riemann, D., Weyerer, S. and Berger, M. (1993). Prevalence and treatment of insomnia in general practice. A longitudinal study. *European Archives of Psychiatry and Clinical Neuroscience*, *242*, 329–336.

Hossain, J. L., Ahmad, P., Reinish, L. W., Kayumov, L., Hossain, N. K. and Sharpiro, C. M. (2005). Subjective fatigue and subjective sleepiness: two independent consequences of sleep disorders? *Journal of Sleep Research*, *14*, 245–253.

Hyyppä, M. T. and Kronholm, E. (1995). Poor sleep among rehabilitation patients. *International Journal of Rehabilitation and Health*, *1*, 1573–1537.

Jacobson, N. S. W. C., Follette, W.C. and Revenstorf, D. (1984). Psychotherapy outcome research: methods for reporting variability and evaluating clinical significance. *Behavior Therapy*, *15*, 336–352.

Jacobson, N. S. and Truax, P. (1991). Clinical significance: a statistical approach to defining meaningful change in psychotherapy research. *Journal of Consulting Clininical Psychology*, *59*, 12–19.

Janz, N. K. and Becker, M. H. (1984). The Health Belief Model: a decade later. *Health Education Quarterly*, *11*, 1–47.

Joa, K. L., Kim, W. H., Park, C. H., Kim, E. S., Lee, S. J., Ko, S. H. and Jung, H. Y. (2017). The effect of sleep disturbance on the functional recovery of rehabilitation inpatients following mild and moderate stroke. *American Journal of Physical and Medical Rehabilitation*, *96*, 734–740.

Johns, M. W. (1991). A new method for measuring daytime sleepiness: the Epworth Sleepiness Scale. *Sleep*, *14*, 540–545.

Johnson, J. A., Rash, J. A., Campbell, T. S., Savard, J., Gehrman, P. R., Perlis, M., Carlson, L. E. and Garland, S. N. (2016). A systematic review and meta-analysis of randomised controlled trials of cognitive behaviour therapy for insomnia (CBT-I) in cancer survivors. *Sleep Medicine Reviews*, *27*, 20–28.

Jones, F. (2006). Strategies to enhance chronic disease self-management: how can we apply this to stroke? *Disability and Rehabilitation*, *28*, 841–847.

Kaplan, G., Vasterling, J. and Priyanka, C. (2010). Brain-derived neurotrophic factor in traumatic brain injury, post-traumatic stress disorder, and their comorbid conditions: role in pathogenesis and treatment. *Behavioural Pharmacology*, *21*, 427–437.

Kneebone, I. I. (2016). A framework to support cognitive behavior therapy for emotional disorder after stroke. *Cognitive and Behavioral Practice 23*, 99–109.

Krupp, L. B., LaRocca, N. G., Muir-Nash, J. and Steinberg, A. D. (1989). The fatigue severity scale. Application to patients with multiple sclerosis and systemic lupus erythematosus. *Archives of Neurology*, *46*, 1121–1123.

Kushida, C. A., Chang, A., Gadkary, C., Guilleminault, C., Carrillo, O. and Dement, W. C. (2001). Comparison of actigraphic, polysomnographic, and subjective assessment of sleep parameters in sleep-disordered patients. *Sleep Medicine*, *2*, 389–396.

Kyle, S. D., Morgan, K., Spiegelhalder, K. and Espie, C. A. (2011). No pain, no gain: an exploratory within-subjects mixed-methods evaluation of the patient experience of sleep restriction therapy (SRT) for insomnia. *Sleep Medicine*, *12*, 735–747.

Lacks, P. (1987). *Behavioral Treatment for Persistent Insomnia*. Pergamon Press, New York, USA.

Lautenbacher, S., Kundermann, B. and Krieg, J.-C. (2006). Sleep deprivation and pain perception. *Sleep Medicine Reviews*, *10*, 357.

Leppävuori, A., Pohjasvaara, T., Vataja, R., Kaste, M. and Erkinjuntti, T. (2002). Insomnia in ischemic stroke patients. *Cerebrovascular Diseases*, *14*, 90–97.

Lichstein, K. L., Means, M. K., Noe, S. L. and Aguillard, R. N. (1997). Fatigue and sleep disorders. *Behaviour and Research Therapy*, *35*, 733–740.

Lichstein, K. L., Stone, K. C., Donaldson, J., Nau, S. D., Soeffing, J. P., Murray, D. et al. (2006). Actigraphy validation with insomnia. *Sleep*, *29*, 232–239.

Lichstein, K. L., Taylor, D. J., Bush, A. J., Reidel, B. W. and Durrence, H. H. (2004). *Epidemiology of Sleep Age, Gender and Ethnicity*. Taylor and Francis.

Li Pi Shan, R. S. and Ashworth, N. L. (2004). Comparison of lorazepam and zopiclone for insomnia in patients with stroke and brain injury: a randomized, crossover, double-blinded trial. *American Journal of Physical Medical Rehabilitation*, *83*, 421–427.

Lu, W., Krellman, J. W. and Dijkers, M. P. (2016). Can Cognitive Behavioral Therapy for Insomnia also treat fatigue, pain, and mood symptoms in individuals with traumatic brain injury? – A multiple case report. *NeuroRehabilitation*, *38*, 59–69.

Malouff, J. M., Thorsteinsson, E. B., Rooke, S. E., Bhullar, N. and Schutte, N. S. (2008). Efficacy of cognitive behavioral therapy for chronic fatigue syndrome: a meta-analysis. *Clinical Psychology Review*, *28*, 736–745.

Manber, R., Carney, C., Edinger, J., Epstein, D., Friedman, L., Haynes, P.L., Karlin, B. E., Pigeon, W., Siebern, A. T. and Trockel, M. (2012). Dissemination of CBTI to the non-sleep specialist: protocol development and training issues. *Journal of Clinical Sleep Medicine*, *8*, 209–218.

Manber, R., Edinget, J. D., Gress, J. L., San Pedro-Salcedo, M. G., Kuo, Tracy, F. and Kalista, M. A. (2008). Cognitive behavioural therapy for insomnia enhances depression outcome in patients with comorbid major depressive disorder and insomnia. *Sleep*, *31*, 489–485.

Mathiowetz, V., Matuska, K. M. and Murphy, M. E. (2001). Efficacy of an energy conservation course for persons with multiple sclerosis. *Archives and Physical Medicine and Rehabilitation*, *82*, 449–456.

McGeough, E., Pollock, A., Smith, L. N., Dennis, M., Sharpe, M., Lewis, S. and Mead, G. E. (2009). Interventions for post-stroke fatigue. *Cochrane Database of Systematic Reviews*, *3*, CD007030.

Means, M. K., Edinger, J. D., Glenn, D. M. and Fins, A. I. (2003). Accuracy of sleep perceptions among insomnia sufferers and normal sleepers. *Sleep Medicine*, *4*, 285–296.

Mental Health Task Force (2016). Implementing the five year forward view for mental health: a report from the independent Mental Health Taskforce to the NHS in England Mental Health Network. NHS Confederation, issue 288.

Mitchell, M. D., Gehrman, P., Perlis, M. and Umscheid, C. A. (2012). Comparativve effectiveness of cognitive behavioral therapy for insomnia: a systematic review. *BMC Family Practice*, *25*, 13–40.

Morin, C. M., Colecchi, C., Stone, J., Sood, R. and Brink, D. (1999). Behavioral and pharmacological therapies for late-life insomnia: a randomized controlled trial. *Journal of the American Medical Association*, *281*, 991–999.

Morin, C. M. and Espie, C. A. (2003). *Insomnia: A Clinical Guide to Assessment and Treatment*. Kluwer Academic/Plenum Publishers, New York, USA.

Morin, C. M., Bastien, C., Guay, B., Radouco-Thomas, M., Leblanc, J. and Vallieres, A. (2004). Randomized clinical trial of supervised tapering and cognitive behavior therapy to facilitate benzodiazepine discontinuation in older adults with chronic insomnia. *American Journal of Psychiatry*, *161*, 332–342.

Morin, C. M., Bootzin, R. R., Buysse, D. J., Edinger, J. D., Espie, C. A. and Lichstein, K. L. (2006). Psychological and behavioral treatment of insomnia: update of the recent evidence (1998–2004). *Sleep*, *29*, 1398–1414.

Morin, C. M., Vallieres, A. and Ivers, H. (2007). Dysfunctional beliefs and attitudes about sleep (DBAS): validation of a brief version (DBAS-16). *Sleep*, *30*, 1547–1554.

Morin, C. M., Vallieres, A., Guay, B., Ivers, H., Savard, J., Merette, C., Bastien, C. and Baillargeon, L. (2009). Cognitive behavioral therapy, singly and combined with medication, for persistent insomnia: a randomized controlled trial. *Journal of American Medical Association*, *301*, 2005–2015.

Nair, W., Lehmann, J., Williams, J. M. and Weiss, B. (1993). Traumatic brain injury: an emerging therapeutic indication. *Drug News and Perspectives*, *6*, 600–603.

Neilson, W., Jensen, M. P., Karsdrop, P. A. and Vlaeyen, J. W. S. (2013). Activity pacing in chronic pain: concepts, evidence and future directions. *Clinical Journal of Pain*, *29*, 461–468.

Nguyen, S., McKay, A., Wong, D., Rajaratnam, S. M., Spitz, G., Williams, G. and Ponsford, J. L. (2017a). Cognitive behavior therapy to treat sleep disturbance and fatigue following traumatic brain injury: a pilot randomized controlled trial. *Archives of Physical and Medical Rehabilitation*, *98*, 1508–1517.

Nguyen, S., Wong, D., McKay, A., Rajaratnam, S. M., Spitz, G., Williams, G., Mansfield, D and Ponsford, J. L. (2017b). Cognitive behavioural therapy for post-stroke fatigue and sleep disturbance:

a pilot randomised controlled trial with blind assessment. *Neuropsychological Rehabilitation*, *19*, 1–16.

O'Neill, D. (2010). Reliable change and clinical significance, (2010). In Brown, J. M. and Cample, E. (eds), *The Cambridge Handbook of Forensic Psychology*. Cambridge University Press, New York, USA.

Ostwald, S. K., Godwin, K. M., Cron, S. G., Kelley, C. P., Hersch, G. and Davis, S. (2014). Home-based psychoeducational and mailed information programs for stroke-caregiving dyads post-discharge: a randomized trial. *Disability Rehabilitation*, *36*, 55–62.

Ouellet, M. C. and Morin, C. M. (2004). Cognitive behavioral therapy for insomnia associated with traumatic brain injury: a single-case study. *Archives of Physical Medicine and Rehabilitation*, *8*, 1298–1302.

Ouellet, M. C. and Morin, C. M. (2007). Efficacy of cognitive-behavioral therapy for insomnia associated with traumatic brain injury: a single-case experimental design. *Archives of Physical Medicine Rehabilitation*, *88*, 1581–1592.

Ouellet, M. C. and Morin, C. M. (2015). Sleep-walk disturbance after traumatic brain injury. *Lancet Neurology*, *14*, 746–757.

Perfect, M. M. and Elkins, G. R. (2010). Cognitive-behavioral therapy and hypnotic relaxation to treat sleep problems in an adolescent with diabetes. *Journal of Clinical Psychology*, *66*, 1205–1215.

Perlis, M. L., Giles, D. E., Mendelson, W. B., Bootzin, R. R. and Wyatt, J. K. (1997). Psychophysiological insomnia: the behavioural model and a neurocognitive perspective. *Journal of Sleep Research*, *6*, 179–188.

Perlis, M. L., Jungquist, C., Smith, M. T. and Posner, D. (2005). *Cognitive Behavioral Treatment of Insomnia: A Session-by-Session Guide*. Springer Science+Business Media, Inc., New York, USA.

Perlis, M. L. and Smith, M. T. (2008). How can we make CBT-I and other BSM services widely available? *Journal of Clinical Sleep Medicine*, *4*, 11–13.

Qaseem, A., Kansagara, D., Forciea, M. A., Cooke, M. and Denberg, T. D. (2016). Management of chronic insomnia disorder in adults: a clinical practice guidelines from the American College of Physicians. *Annals of Internal Medicine*, *165*, 125–133.

Rassafiani, M. and Sahaf, R. (2010). Single case experimental design: an overview. *International Journal of Therapy and Rehabilitation*, *17*, 285–289.

Reid, K. J., Martinovich, Z., Finkel, S., Statsinger, J., Golden, R., Harter, K. and Zee, P. C. (2006). Sleep: a marker of physical and mental health in the elderly. *American Journal of Geriatric Psychiatry*, *14*, 860–866.

Sanford, S. D., Lichstein, K. L., Durrence, H. H., Riedel, B. W., Taylor, D. J. and Bush, A. J. (2006). The influence of age, gender, ethnicity, and insomnia on Epworth sleepiness scores: a normative US population. *Sleep Medicine*, *7*, 319–326.

Schepers, V. P., Visser-Meily, A. M., Ketelaar, M. and Lindeman, E. (2006). Post-stroke fatigue: course and its relation to personal and stroke-related factors. *Archives of Physical Medicine and Rehabilitation*, *87*, 184–188.

Schuiling, W. J., Rinkel, G. J., Walchenbach, R. and de Weerd, A. W. (2005). Disorders of sleep and wake in patients after subarachnoid hemorrhage. *Stroke*, *36*, 578–582.

Siebern, A. T. and Manber, R. (2011). New developments in cognitive behavioral therapy as the first-line treatment of insomnia. *Psychology Research and Behavior Management*, *4*, 21–28.

Smith, M. T., Huang, M. I. and Manber, R. (2005). Cognitive behavior therapy for chronic insomnia occurring within the context of medical and psychiatric disorders. *Clinical Psychology Review*, *25*, 559–592.

Smith, S., Lang, C. P., Sullivan, K. A. and Warren, J. (2004). A preliminary investigation of the effectiveness of a sleep apnea education program. *Journal of Psychosomatic Research*, *56*, 245–249.

Stepanski, E. J. and Wyatt, J. K. (2003). Use of sleep hygiene in the treatment of insomnia. *Sleep Medical Reviews*, *7*, 215–225.

Sterr, A., Herron, K., Dijk, D.-J. and Ellis, J. (2008). Time to wake-up: sleep problems and daytime sleepiness in long-term stroke survivors. *Brain Injury*, *22*, 575–579.

Strine, T. W. and Chapman, D. P. (2005). Associations of frequent sleep insufficiency with health-related quality of life and health behaviours. *Sleep Medicine*, *6*, 23–27.

Tang, N. K. Y., Lereya, S. T., Boulton, H., Miller, M., Wolke, D. and Cappuccio, F. P. (2015). Systematic review and meta-analysis of patient-reported outcomes in randomnized controlled trials. *Sleep*, *38*, 1751–1764.

Tate, R. L. et al. (2016). The Single-Case Reporting Guideline In BEhavioural Interventions (SCRIBE) 2016 Statement. *Archives of Scientific Psychology*, *4*, 1–9.

Trauer, J. M., Qian, M. Y., Rajaratnam, S. M. and Cunnington, D. (2015). Cognitive behavioral therapy for chronic insomnia: a systematic review and meta-analysis. *Annals of Internal Medicine*, *163*, 191–204.

Troxel, W. M., Germain, A. and Buysse, D. J. (2012). Clinical management of insomnia with Brief Behavioral Treatment (BBTI). *Behavioural Sleep Medicine*, *10*, 266–279.

Trudel, T. M., Tryon, W. W. and Purdum, C. M. (1998). Awareness of disability and long-term outcome after traumatic brain injury. *Rehabilitation Psychology*, *43*, 267–281.

Valko, P. O., Bassetti, C. L., Bloch, K. E., Held, U. and Baumann, C. R. (2008). Validation of the fatigue severity scale in a Swiss cohort. *Sleep*, *31*, 1601–1607.

Vincent, N., Lewycky, S. and Finnegan, H. (2008). Barriers to engagement in sleep restriction and stimulus control in chronic insomnia. *Journal of Consulting Clinical Psychology*, *76*, 820–828.

Vitiello, M. V., McCurry, S. M. and Rybarczyk, B. D. (2013). The future of cognitive behavioural therapy for insomnia: what important research remains to be done? *Journal of Clinical Psychology*, *69*, 1013–1021.

Wade, A. G. (2011). The societal costs of insomnia. *Neuropsychiatry Disease and Treatment*, *7*, 1–18.

Wells, A., Clark, D. M., Salkovskis, P., Ludgate, J., Hackmann, A. and Gelder, M. (1995). The role of in-situation safety behaviors in maintaining anxiety and negative beliefs. *Behavior Therapy*, *26*, 153–161.

Wessendorf, T. E., Teschler, H., Wang, Y. M., Konietzko, N. and Thilmann, A. F. (2000). Sleep-disordered breathing among patients with first-ever stroke. *Journal of Neurology*, *247*, 41–47.

White, P. D. et al. (on behalf of the PACE trial management group) (2011). Comparison of adaptive pacing therapy, cognitive behaviour therapy, graded exercise therapy, and specialist medical care for chronic fatigue syndrome (PACE): a randomised trial. *The Lancet*, *377*, 823–836.

Widar, M., Ek, A.-C. and Ahlstrom, G. (2004). Coping with long-term pain after a stroke. *Journal of Pain and Symptom Management*, *27*, 215–225.

Williams, A. C., Eccleston, C. and Morley, S. (2012). Psychological therapies for the management of chronic pain (excluding headache) in adults. *Cochrane Database Systematic Reviews*, *14*, 11.

Wilson, S. L. (2000). *Single Case Experimental Design*. In Breakwell, G. M., Hammond, S. and Fife-Shaw, C. (eds). Sage Publications, London, UK.

Worthington, A. D. and Melia, Y. (2006). Rehabilitation is compromised by arousal and sleep disorders: results of a survey of rehabilitation centres. *Brain Injury*, *20*, 327–332.

Wroe, A. L., Rennie, E. W., Gibbons, S., Hassy, A. and Chapman, J. E. (2015). IAPT and long term medical conditions: what can we offer? *Behavioural and Cognitive Psychotherapy*, *43*, 412–425.